THE UNIVERSITY OF MINNESOTA

GRADUATE SCHOOL

Report

of

Committee on Examination

This is to certify that we the undersigned, as a committee of the Graduate School, have given Clifton Walter Ackerson final oral examination for the degree of

Master of Science.

We recommend that the degree of

Master of Science

be conferred upon the candidate.

L. A. Palmer
Chairman

J. F. C. Glendon

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Date May 25, 1922

THE UNIVERSITY OF MINNESOTA

GRADUATE SCHOOL

Report

of

Committee on Thesis

The undersigned, acting as a Committee of the Graduate School, have read the accompanying thesis submitted by Clifton Walter Ackerson for the degree of Master of Science. They approve it as a thesis meeting the requirements of the Graduate School of the University of Minnesota, and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science.

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THE ANTISCORBUTIC PROPERTY OF MARKET MILK

BY

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The Antiscorbutic Property of Market Milk.

Table of Contents.

Introduction.

Scurvy was probably first heard of as a sweeping plague in time of war infesting armies in the field, and working havoc with the population of beleaguered cities. Later it was heard of as a loathsome disease making heavy inroads in the ranks of the navy and the merchant marine. More recently it has manifested itself in the privations of the Hudson Bay trapper; in the gold rushes of Nome and Dawson on the Yukon; in the siege of Port Arthur by the Japanese, and even among the Allied and German troops during the World War.

Scurvy has also been looked upon as a nutritional disease endangering the lives of infants. In this connection, many types of food were studied with emphasis on their power to protect children from scurvy. Under the influence of this study it has taken on the aspect of a disorder caused by certain dietary factors.

The prevalance of scurvy under conditions where fresh food was not available, or else obtained with difficulty over long periods of time, very early tended to focus the attention of many investigators on that phase of the problem. Their work had for its object the discovery of the foods which possessed the power of preventing, or curing scurvy. As a result, foods are now fairly well classified in that respect. They also brought out the fact that the world is dependent on its annual crops for a supply of antiscorbutic food.

Naturally enough the antiscorbutic potency of milk has been

given considerable attention. This is due to the spread of infantile scurvy in the world among the infants which have been fed cows' milk as the sole diet. When mother's milk fails, artificial feeding must be resorted to, and cows' milk is most naturally chosen as a substitute. The feeding of cows' milk to infants presents the problem of modification to meet their peculiar dietary needs. Furthermore, the danger of infection thru the bacteria present in cows' milk has led to a complex system of processing which destroys harmful organisms without materially affecting the flavor of the product.

Unfortunately, pasteurization not only destroys the germs found in milk, but is believed to affect its antiscorbutic property as well. The ideal procedure would be one in which the destruction of the pathogenic bacteria is accomplished without loss of antiscorbutic value. The destruction of these bacteria, however, is a matter of temperature and time. The temperatures to be employed in pasteurization, therefore, are more or less fixed. Thus it follows that any variations in the antiscorbutic potency of milk which can be brought about thru changes in the methods of processing market milk must be accomplished by modifications in the mechanical operations of the commercial milk plant which have as their central idea the maintainence of a sufficiently high temperature over a period of time long enough to destroy pathogenic bacteria. The growth of the market milk business in large urban centers has resulted in certain modifications of the mechanical features of milk

pasteurizing. However, no attention has been paid to the possible effect any such changes might produce on the antiscorbutic potency of milk.

It is the purpose of the investigation herein reported to study the variations produced in the antiscorbutic content of market milks by diverse methods of pasteurization, and, in a measure, to attempt to account for the differences which apparently exist.

Historical.

It is rather difficult to get at the earliest description of scurvy. Hippocrates referred to the fact that a large number of men in the Greek army suffered from pains in the legs, and gangrene of the gums accompanied by loss of teeth; this disorder is most likely identified with true scurvy. There is no record to show that Greek, Roman, or Arabian historians were familiar with it. De Joinville in the thirteenth century described the disease as a livid and spongy condition of the gums, debility, tendency to faint, and black spots on the legs. Magnus (1555) stated that it occurred in army camps and prisons. Rosseus, Echtius, and Wierus wrote special treatises on scurvy and recommended many dietary measures which still hold true.

After the sixteenth century, descriptions of scurvy outbreaks on land began to be more plentiful. Accounts can be found to show that the French met with difficulty in the settlement of the northern part of America due to the insufficien-

cy of fresh food caused by the severe and long winters. The same trouble was encountered by the English in their settlement of Newfoundland, and prevented the early settlers of the Hudson Bay territory from colonizing it.

Bachstrom (1734) described an epidemic of scurvy which took place at the siege of Thorn, Prussia, by the Swedes in 1703. The striking feature of this report is that the epidemic took place in the summer and not in the cold season. In general, it may be stated that scurvy and war have, as history shows, gone hand in hand. This is true both for the armies in the field and the civilian population as well. Troops campaigning usually have a restricted ration where desiccated foods take the place of fresh food, with the result that cases of scurvy have been of frequent occurrence in all recorded wars. Deprivation of fresh food among the peoples of warring nations has been common and scurvy an almost certain result.

Hess (1920) mentions scurvy "in the Russian armies, in the war between the Austrians and Turks in 1720; in the English troops who had taken Quebec from the French in 1759; among the French soldiers in the army of the Alps in the spring of 1795." As examples in more recent times can be cited the outbreak during the Crimean War, and the fact that it was quite common in our Civil War; It was prevalent among garrisons in the Russo-Japanese War; recent reports show that scurvy was widely disseminated among the Russian, Serbian, German, Austrian, and Turkish soldiers during the World War. However, few cases were

described along the western front, although Korbsch (1915) described fifty one. Colonial Indian troops suffered considerable loss from scurvy, according to Colonel Hehir. French colonials also suffered, as did Italian troops in Macedonia. American troops, most probably on account of the large ration used, and their very brief period of activity in the field, escaped any serious trouble with the disease.

The civilian population of Russia must have suffered a good deal, although no data are available to indicate the extent. Brandt reported scurvy as occurring in a foundling asylum in Berlin; Great Britain had a few cases reported from institutions caring for the poor. Famine conditions in Vienna produced a scarcity of fresh vegetables and the development of a large number of cases of scurvy. Hess (1920) states that "a conception of the deficiency of the milk supply may be gained from the statement that there were but sixteen quarts of milk a day for about fifteen hundred people." One can but conclude that scurvy is only one of the inevitable results of war.

It was not until the appearance of Barlow's (1883) classical treatise that anatomical proof of the fact that adult and infantile scurvy were one and the same disease was furnished. This view was readily accepted in England and America, less readily in Europe. Many continental investigators maintained that Barlow's disease was a complication of rickets, but this view gave way to the acceptance of Barlow's report.

Etiology

Many theories as to the cause of scurvy have been advanced. It is not possible, however, to discuss separately the theories which have been held regarding the etiology of scurvy as it has manifested itself in infants and adults. In fact, the history of the two syndromes is more or less interwoven. Besides, as already stated, the diseases are now regarded as identical.

Among many others, the potassium deficiency theory suggested by Garrod (1848) gained rather widespread recognition. Experimental application of the theory disproved the proposition.

Netter (1899) supported the citric acid theory due to the known curative effects of citrous fruits, and the fact that the concentration of citric acid salts is higher in human milk than cows' milk and that some of these salts are lost on heating the milk. However, the feeding of these salts failed to cure or prevent scurvy, as shown by Hess and other writers.

Wright (1908) put forth the acidosis idea, maintaining that an excess of acid over alkaline food gave rise to scurvy. When tested out clinically this idea was quickly rejected.

The toxic theory was one of the theories regarding the etiology of scurvy that had many followers and opponents. This view held that poisons were ingested in the food or else produced in the intestines by the action of bacteria. The minority report of the American Pediatric Society (1898) concluded that scurvy appeared to be a chronic ptomaine poisoning due to the absorption of toxic substances. Among the most ardent supporters

of this belief were McCollum and Pitz (1917) who, after observations in their laboratory, stated that "it (scurvy) is the result of the absorption of toxic substances arising from putrefaction in the cecum due to undue retention of the feces." This was followed by the suggestion of Gerstenberger (1918) that a break in the carbohydrate metabolism produced a defunctioning substance which was possibly oxalic acid, a substance with a strong affinity for calcium.

It is evident that the frequency of constipation among thriving bottle fed infants excludes the retention of the feces as the sole cause of scurvy. Hess (1920) failed to note a characteristic distinction between scorbutic and non-scorbutic babies in this respect.

Still other writers considered scurvy to have a bacterial origin. Hess (1920) mentions Boerhave as having maintained such an idea, and that Villemin upheld it in the seventeenth century. Recent writers, notably European army surgeons, have advanced this theory. Clinical evidence disproves the hypothesis, however. One fact which is strongly against the belief that scurvy is an infectious disease is that whenever it (scurvy) appears among a body of troops, the officers are usually spared. This fact was noted in the Report of the War of the Rebellion, and by Hoerschelman and others in the World War. There has been no instance reported of the isolation of bacteria from the blood of a scurvy patient before death.

At present the most widely accepted theory is that of the

accessory factor, or vitamine theory. Eijkman (1397) demonstrated the nature of polyneuritis in fowls, and Hopkins (1906) showed the need of an unknown dietary factor in connection with the normal nutrition of the rat. Eijkman's postulation that polyneuritis in fowls and beri-beri in man were the same disease led Holst to attempt to find some experimental animal that would stand in the same relation to scurvy in man. This trial led to the publication of work by Holst and Frölich (1907) to show that they had produced in guinea pigs a disease identical in all its pathological and anatomical relations to human scurvy. Further than that, they demonstrated by means of exclusion that scurvy was due to the lack of an unidentified dietary factor. Since that time the evidence in favor of the vitamine theory has continued to pile up, until today there is little doubt of its correctness.

Infantile Scurvy.

One factor that has to be considered in the etiology of scurvy, is the part played by pasteurization of cows' milk. The term pasteurization, as defined by Hess (1920), is taken to mean the "entire commercial process - the heating, handling, subsequent cooling, aging and all other factors involved." Despite the many apparent advantages pasteurization offers in the control of infant mortality, its relation to the antiscorbutic value has been extensively studied.

Neumann (1901) reported an interesting case that showed the direct relation between the pasteurization of cows' milk and the

incidence of infantile scurvy. In Berlin that year a large dairy commenced to pasteurize their milk by heating to 140°F. After a few months infantile scurvy was reported from various sections of the city, resulting in a very great rise in the number of cases that year over the previous year. After an investigation had been held, pasteurization was discontinued, with the result that cases decreased as rapidly as they increased.

A similar situation was met with in Switzerland, where Bernheim-Karrer (1907) reported that nine cases of scurvy occurred on a type of homogenized milk. Le Cornu (1904) stated the same condition obtained in France following the use of homogenized milk in place of sterilized milk, and that scurvy was practically unknown before the introduction of "lait maternise."

There is a difference in opinion as to the effect boiling has on the antiscorbutic potency, although it is quite generally accepted that cows' milk which has been heated twice has suffered a greater loss of antiscorbutic value, than if it had been heated but once.

The general use of dried milk in infant feeding has been delayed somewhat by the fear that its use might induce scurvy. Papers by Hess and Unger claimed considerable antiscorbutic value for milk dried by the Just-Hatmaker process, where the milk remains in contact with a drum at 230°F. for two or three seconds. Barnes and Hume (1919) and Hart, Steenbock and Ellis (1921) showed that dried milk was inferior in antiscorbutic vitamine to raw milk, but that the amount of vitamine destroyed

depended on the manner in which the cows' milk was dried, confirming the belief that the loss suffered on the drum was less than in the spray process.

Hart, Steenbock, and Smith (1919) reported that commercial unsweetened condensed milk suffered a loss of the antiscorbutic factor as compared with the milk from which it was made.

The extensive use of proprietary foods has made infantile scurvy more prevalent in England and America than Europe. Several factors tend to bring about this condition. In the first place the small amount of milk used in their makeup; second the application of heat. Furthermore, Hess states that the majority of such infant foods are alkaline in reaction; according to Hess and Unger (1919) the antiscorbutic vitamine is sensitive to the slightest alkalinity.

Experimental Scurvy in Guinea Pigs.

It was not until very recently that investigators turned their attention to a study of scurvy from an experimental point of view. Prior to 1895, there had been no report of scurvy in animals. In that year Theobald Smith (1895) wrote "when guinea pigs are fed with cereal (it has been observed for several years in this laboratory) with bran and oats mixed, without any grass, clover, or succulent vegetable, a peculiar disease, chiefly reconizable by subcutaneous extravasations of blood, carries them off in from four to eight weeks." Bolle (1903) produced a disorder which he considered to be true scurvy by

feeding guinea pigs a diet of raw or sterilized milk. Bartenstein (1905) repeated this work and, in addition, described the bone changes in detail.

It was not until a few years later that a relation was discovered between human and experimental scurvy. Eijkman's postulation, that polyneuritis in fowl and beri-beri in man were identical diseases, as mentioned above, led Holst and Frŏhlich, two Norweigen workers, to attempt to find some laboratory animal that would stand in the same relation to scurvy as fowls did to human beri-beri. In 1907 these two workers published the preliminary report of their work, and stated that epidemiological facts favored the belief that the disorder produced by a strict cereal diet in guinea pigs was identical with human scurvy. Subsequent investigation by a host of other workers has since confirmed this belief. Viewed from the standpoint of mode of production, pathology and curative measures the malady is identical in man and the guinea pig.

Holst and Frŏhlich in the work mentioned above, used a diet of oats, hay and water; this ration has been used quite extensively in subsequent investigations. Chick, Hume and Skelton (1918) used a basal diet of oats, bran and water, or oats, bran and sixty cc. of milk autoclaved at 120°C. for one hour. These workers held that this ration lacked only the antiscorbutic vitamine. Delf (1920) used the same ration; Hart, Steenbock and Smith produced scurvy in from four to five weeks on any of the following diets:

Rolled oats, 84%; dried corn stover, 15%; NaCl, 1%

Dried alfalfa, 25%; rolled oats, 75%

Dried corn stover 49%; rolled oats 59%; NaCl, 1%

Corn meal, 35%; gluten feed, 14.3%; corn stover, 49%; NaCl, 1%. Sherman, La Mer and Campbell, (1922) report the use of the following:- Ground whole oats, 59%; Skimmed milk powder heated on open trays at 110°C. until all the antiscorbutic is destroyed, 30%; butter fat, freshly prepared, 10%; sodium chloride, 1%. Cohen and Mendel (1913) studied scurvy producing diets, such as cereals, oatmeal; oats and yeast; oats, calcium lactate, and sodium chloride; barley exclusively; a "soy bean cracker" composed of soy bean flour previously heated for thirty minutes at fifteen pounds pressure, three percent of sodium chloride and the same amount of calcium lactate and of dried brewers yeast, with enough raw milk to give five per cent fat.

It was probably Fröhlich (1912) who first suggested and used cows' milk to produce experimental scurvy in guinea pigs, basing his idea, no doubt, on Bolle's work. In 1912, he reported that the exclusive use of either raw, boiled or milk heated to a high temperature, eventually produced a disease in guinea pigs having all the characteristics of scurvy. Since that time a great amount of work has been done, both on cows' milk itself as an antiscorbutic food, or where some form of it has been used as a part of the basal diet.

It must not be taken for granted that the belief that the disease produced in guinea pigs was truly scorbutic in character,

was taken without question. One notable exception was the explanation offered by McCollum and Pitz (1917) who stated that "scurvy in guinea pigs is not the result of any deficiency of any specific protective substance, but is the result of the absorption of toxic substances arising from putrefaction in the cecum due to undue retention of the feces." This report served to stimulate research along that line, and soon Cohen and Mendel (1918) and Hess and Unger (1918) showed that there was no relation between the incidence of scurvy, and the impaction of the cecum.

At the same time they proposed the toxin theory, McCollum and Pitz (1917) reported a cure in guinea pigs suffering from scurvy with lubrication of the digestive tract with oil, and that laxatives could replace antiscorbutic food in the diet. Chick, Hume and Skelton (1918) and Hess and Unger (1918) failed to note any protective or curative effect of laxatives, while in 1919, the latter workers demonstrated that the antiscorbutic value of orange juice was not in its laxative properties. McCollum's view is no longer considered tenable, and since that time he has withdrawn from his belief that accessory food factors play no part in experimental scurvy.

Jackson and Moody, and Jackson and Moore (1916) advanced the theory that scurvy in experimental animals might have a bacteriological origin. This report followed the announcement that they had cultivated a dipplococcus from the tissues of scorbutic guinea pigs after death, which on innoculation into

the circulation of other animals produced hemorrhages, and could be recovered again from the tissues. Later workers have failed to confirm these observations.

Symptomatology of Guinea Pig Scurvy.

In considering the symptomatology of experimental scurvy in guinea pigs Hess (1920) stated that the striking feature which he observed in the examination of many hundreds of animals was the marked uniformity of the symptoms encountered. Most frequently, the first symptom to be noted is the loss of weight the animal may undergo when the disease first begins to manifest itself. However, Hess concludes from his data, that the weight curve is no criterion as to the diagnosis of scurvy, and that very often the animals may develop the disease, and yet maintain or even gain in weight.

Probably the first definite sign of scurvy to appear in the guinea pig is evidenced by an extreme tenderness of the joints, particularly the wrists. This tenderness is usually accompanied by an edema and a swelling which makes the clean-cut shape of the joint rounded and blunted. This condition is noted from twelve to fourteen days after the animal has been placed exclusively on a scorbutic diet. Shortly after these changes are observed the general appearance of the guinea pig denotes indisposition to move about. The coat becomes rough instead of remaining sleek or glossy. The tendons of the hind legs may also become palpably tender, and cause the animal to assume

the "scurvy position" described by Chick, Hume and Skelton (1913) indicative of hemorrhage in the joints or muscles. In this position the animal sits on three legs so that the tender leg may avoid subjection to pressure. The above writers noted that there is also a tendency for the animal to curl up and rest an apparently sensitive jaw on the floor, and termed this position the "face ache position". Hess (1920) states that in his experience the earliest diagnosis of scurvy was made on the eighth day, but that it usually took from twelve to fourteen days.

Holst and Fröhlich (1907) described the symptoms of scurvy in guinea pigs in detail. They said that pronounced hemorrhages occur in the muscles of the hind limbs, thighs, and limbs; in the intercostal muscles; in the deep muscular layers, and in the periosteal tissue at the inside of the lower jaws; in the muscles of the fore limbs, back and abdomen. Subcutaneous hemorrhages also occurred. Peritoneal hemorrhages are especially frequent in the duodenum. A subcutaneous edema also occurs. There is marked fragility of the bones. The molars become loose, and bleeding of the gums results. Frequently there is a loosening of the connection between the ribs and the cartilege; there occurs also a severing between the epiphysis and the shaft of the bones, especially in the tibia.

Hess (1920) states that "loosening of the teeth is another typical sign. It is, however, one which does not appear early and is somewhat difficult to elicit. The molar teeth are gener-

ally involved, especially those of the upper jaw, which may be so completely separated from their alveolar sockets that they can be readily removed by forceps." Satisfactory examination of the teeth during life is almost impossible, due to the construction of the mouth of the guinea pig. Very seldom does an incisor become loosened, but it is often found to have lost its polish and to have become dull.

One of the most interesting signs of scurvy, seen only upon necropsy, altho it may oftentimes be palpable in a guinea pig suffering from the disease, is that of the "beading"of the ribs. This feature has been noted by many workers, particularly by Jackson and Moore (1916). Hess (1920) stated that this symptom is not of diagnostic importance. Beading consists of a swelling of the costochondral junction of the ribs, more frequently occurring in the lower true ribs. Another symptom,visible at this time, is the so-called"white line" first described by Frankel (1904). This manifests itself at the end of the long bones near their epiphyses as a whitish or yellowish bar, when observed by means of the X-ray. A transverse white bar is also noticeable at the costochondral junctions of the ribs. This line may or may not be present, but if present is a positive indication of a scorbutic condition.

It is well to consider here the pathological changes occurring in scurvy which can be studied microscopically. Hess (1920) describes these changes in detail. He states "the bone marrow at the ends of the diaphyses in proximity to the epiphyseal

junction loses its normal lymphoid character and is replaced by a recticular or fibrillated substance, the so-called 'frame work' marrow containing a homogenous mucoid tissue and only a few osteoblasts and marrow cells." There occur also marked changes in the osseus tissue itself which correspond to the brittleness noticeable on gross examination. "The osseus trabeculae are fewer in number and those which remain are slender and irregular, and frequently appear as isolated islets." In some of the ribs there is a change in the intermediate cartileges, but in some cases only one or two of the ribs will show the characteristic microscopic variations. Normally the cells are arranged in orderly rows, but in scurvy, they are found to be irregularly placed, and at the same time are reduced in size and number. "The bone trabeculae on which they abut are not well formed or of equal length, and do not present an even or transverse plane, but are misshapen and small, so that the line of junction with the cartilage is zig-zag." In severe scurvy these changes may be noted macroscopically as the beading of the ribs. Delf and Tozer (1913) classified these changes according to their degree of severity as"incipient","definite", "acute", "chronic definite", and"chronic acute" scurvy. This microscopic examination of the rib junctions is undoubtedly the best aid in the definite diagnosis of scurvy, with the qualification, however, as voiced by Delf and Tozer (1918),"In other experiments, however, where a liberal supply of antiscorbutic was given and where the fat-soluble A growth factor was known

to be deficient, the resulting histological changes in the rib junctions of the animals examined were found to resemble closely those of 'Definite' or of 'Definite Chronic" scurvy."

Milk Processing and Its relation to Scurvy.

In using the term "pasteurization" in this paper it is taken to mean the heating, handling, cooling, aging and other factors involved in the transfer of cows' milk from the dairy to the market milk plant to the consumer. In a strict sense, the term means the heating of cows' milk to a temperature of 140° to 165°F. for a definite time, and the subsequent cooling.

Cows' milk, as it is produced in the dairy undergoes much the same treatment in various localities. Some of it is pasteurized on the farm, but usually it is merely strained and cooled and delivered to the creamery. There it is run thru a clarifier which removes a greater part of the dirt present, together with a considerable number of bacteria and leucocytes. This process serves to clean the milk but does not materially improve its keeping qualities. The milk may then again be filtered before pasteurization.

It is in the methods of pasteurization that the variations in the antiscorbutic property of market milk are believed to occur. Briefly stated, pasteurization, in the strict sense, means a process of heating cows' milk to about 145°F. and holding at that temperature for about thirty minutes. At the present time there are three ways of accomplishing pasteurization;

first, the flash or continuous process, where the milk is raised to the pasteurizing temperature, held there from thirty seconds to one minute only, and then cooled quickly; second, the holder or holding process, where the milk is quickly brought up to about 145°F. and held there for thirty minutes; third, pasteurization in bottles where the raw cows' milk is put in the bottles with water tight caps and immersed in hot water until heated to 145°F., and held there for from twenty to thirty minutes.

There are two main reasons for pasteurization. In the first place, milk dealers have found by experience that it improves the keeping qualities of the milk by decreasing the bacterial count, thus preventing, or at least delaying, spoiling. The other, and probably the most important reason is the destruction of pathogenic bacteria. Cows' milk undoubtedly is open to various sources of contamination in the process of handling, and it is to minimize the danger of infection when used as a food, particularly for infants' food, that this treatment is resorted to. Many cities have regulations regarding pasteurization, most of which are quite well enforced.

It is with the holding process of pasteurization that this paper is interested, especially as a study of variations produced on the antiscorbutic property of cows' milk by the various mechanical means of doing this work. It is assumed that there is but little variation in the effect on the antiscorbutic factor by differences in handling up to the time of pasteuri-

zation, for every effort is made to keep the milk cool to prevent bacterial growth. The methods employed in the holding process of pasteurization which were studied in this investigation will be described a little later.

The antiscorbutic property of cows' milk was under discussion before experimental animals came into general use for research work. Curran (1847) wrote that in the Irish epidemic of that year, eighty cases of scurvy were reported on a diet which included one pint of milk daily, but was deficient in vegetables. He concluded that milk was not especially rich in the factor which protected against scurvy. Parkes (1848) drew the conclusion that five hundred to seven hundred and fifty cubic centimeters of raw milk daily did not always protect against scurvy in adults. Similar conclusions were drawn from clinical experience with infants.

After the work of Barlow (1883) recognizing the identity of infantile and adult scurvy, work on the question of foods continued to increase. Milk, as a food for infants was taken up along with other foods. Barlow himself (1894) realized that milk is not a very potent antiscorbutic food. The work of Neumann (1902) brought out the causitive relationship existing between the heating of milk and the incidence of infantile scurvy. He observed the development of scurvy where cows' milk was first pasteurized at the dairy, and then, the next day before being fed, heated in a Soxhlet for from ten to fifteen minutes. This was confirmed by Heubner (1903) and Cheadle and Poynton

(1909) who had collected reports on eighty cases of scurvy due to the feeding of cooked milk. The American Pediatric Society (1393) reported a total of three hundred and fifty six cases on artificial feeding , with milk as the principal part of the diet.

The first experimental work to be done was that of Holst and Fröhlich (1912) who stated that the exclusive use of either raw or boiled cows' milk, or milk heated to a high temperature eventually produced a disease having the characteristics of scurvy, upon which lime juice had a beneficial effect. Further than that, raw cows' milk hindered the occurrence of scurvy. This work was not quantitative, as was the work of Funk (1916), who found that fifty cubic centimeters of fresh cows' milk to an oat diet would protect guinea pigs from scurvy.

From this point on in a consideration of the antiscorbutic property of milk, no attempt will be made in this paper to distinguish between human and experimental scurvy, for the history of the work done on cows' milk is quite interwoven in that respect.

Dennet (1912) in studying the effect of the use of boiled milk in infant feeding came to the conclusion that nutritional diseases were not necessarily caused by its use, if properly administered, and that scurvy was easily avoided if orange juice was fed. He stated that boiled cows' milk was not more difficult to digest than the unboiled milk.

Chick, Hume and Skelton (1918) were the first to undertake

a systematic investigation of the antiscorbutic property of milk. They showed that the estimate of Funk as to the amount of milk needed to protect against scurvy in the guinea pig was too low. They reported that if the daily consumption of fresh cows' milk was less than fifty cubic centimeters a guinea pig would die almost as quick as if no milk had been fed, but that some protection was secured with a consumption between fifty and one hundred cubic centimeters daily. When milk constituted practically the complete diet and when fed at a level of from one hundred to one hundred and fifty cubic centimeters daily, satisfactory growth and development resulted without any scurvy symptoms appearing.

Hess (1920) in a consideration of the needs of infants, stated that a minimum of sixteen ounces of fresh milk are required daily in order to protect an infant from scurvy, or to cure a scorbutic child. Twelve ounces of fresh raw cows' milk failed to prevent the onset of the disease. In this connection Hess (1920) states that there are two things to consider when dealing with pasteurized milk. In the first place it acts as an antiscorbutic if but little of its vitamine is destroyed, and secondly if a large amount of milk is consumed. Hess (1916) stated that the protection from infectious diseases afforded to babies by pasteurizing milk led to its use, and that an antiscorbutic must be supplied, for subacute scurvy developed in children when pasteurized milk was used.

Hess and Unger (1919) studied the factors involved in the

development of infantile scurvy on a diet of malt soup containing milk and found that the indications pointed to a destruction of the antiscorbutic vitamine in the milk by the alkali present in the malt soup extract. They showed that the antiscorbutic factor in milk was destroyed by alkalization associated with heating, as shown by the experiments with guinea pigs. They suggested that when the diet consisted of alkaline proprietary infant foods they be supplemented with an antiscorbutic such as tomato or orange juice.

On the other hand, Nobet (1921) reported seven children as being cured of Barlow's disease by feeding them exclusively on milk. In five of the cases the milk was heated for ten to thirty-five minutes, and in two cases the heating was prolonged for an hour. He concluded that the vitamine was not as thermolabile as it had been assumed to be, and that the water content of the food had something to do with the stability of the vitamine.

Moore and Jackson (1916A) found that an exclusive diet of raw cows' milk, pasteurized, boiled or skimmed milk produced scurvy in guinea pigs. Further, the addition of calcium lactate to raw whole milk did not prevent scurvy. A cream diet and a diet of olive oil added to cows' milk produced a fat constipation with early death.

Hart, Steenbock and Smith (1919) found that one hundred cubic centimeters of whole milk per guinea pig daily protected against scurvy for eighteen weeks. Unsweetened condensed milk,

milk powders, and milks sterilized for ten minutes at 120°C. suffer a loss or a destruction of the antiscorbutic. Scurvy was produced on a diet of forty cubic centimeters of fresh whole milk and rolled oats. In the presence of hay thirty cubic centimeters of fresh whole milk delayed the onset of scurvy, while eightyfour cubic centimeters of milk together with rolled oats and hay gave full protection. The use of mineral oil or phenolpthalein with either hay or hay and oats, without milk failed to protect guinea pigs from scurvy.

Hart, Steenbock and Ellis (1920) drew the following conclusions: "1. the concentration of the antiscorbutic factor in milk is dependent on the diet; 2. summer pasture milk is much richer in this nutritive factor than dry feed milk or winter produced milk, involving the use of corn silage or mangels: 3. fifteen cubic centimeters of summer pasture milk imposed upon the basal ration used afforded protection for twenty weeks to one guinea pig. In general it was insufficient." 4. At least seventy five cubic centimeters daily of dry feed milk per pig were needed for complete protection. This shows that the dry feeds used in producing the milk were not entirely devoid of C. 5. Silage or sugar mangel milk is on a par with dry feed milk, and not in the class with green pasture summer milk.

This work was followed by that of Dutcher and co-workers (1920) who concluded that the vitamine content of cows' milk was dependent upon the vitamine content of the ration ingested by the cow. "Twenty cubic centimeters of summer milk were su-

perior in nutritive value and in antiscorbutic potency to sixty cubic centimeters of winter milk. It was found that there is a tendency for the milk to become poor slowly, when the diet of the cow is low in vitamines, while the milk becomes of higher nutritive value almost immediately upon the ingestion of the vitamine rich-ration. In other words, there seems to be a tendency for milk to become poor slowly and rich rapidly as far as vitamines are concerned."

Supplee and Bellis (1921) noted a marked variation in the citric acid content of cows'milk from different animals due to individuality and thought that the ration had some effect upon the content of citric acid in the milk. They found no effect on this constituent due to the heating in the manufacture of evaporated, condensed and dried milks, and the parallelism between the citric acid content and the antiscorbutic properties of cows' milk did not hold true in the case of concentrated milk products.

Faber (1921) reported a case of scurvy developing in an infant fed from birth on modified raw certified milk to which a small amount of sodium citrate (a total of about eighty four hundredths of a gram daily) had been added. Definite proof that the antiscorbutic was destroyed by the use of sodium citrate was not obtained by the author, but he suggested that the use of the salt in infant feeding over long periods of time should be regarded as dangerous.

Hess (1921) stated that if the food was acid it was much

less apt to undergo destruction of the antiscorbutic. He demonstrated that oxidation played a large part in the destruction of C by adding four cubic centimeters of hydrogen peroxide to a liter of cows' milk and incubating at blood heat over night. Eighty cubic centimeters of milk treated in this manner failed to protect guinea pigs from scurvy. Likewise, orange juice subjected to a stream of oxygen lost its antiscorbutic power. Hess and Unger (1921) studied the effect of copper acting as a catalyst in the destruction of C. They heated cows' milk in a glass vessel to sixty degrees centigrade for forty minutes and in another case, in a copper vessel in like manner. Equivalents of one hundred cubic centimeters of fluid milk and oats in addition, were fed. Diluted dry milk was also used to concentrate the milk and to insure its consumption. Guinea pigs fed on the milk treated in the glass vessel failed to develop scurvy in four months. On the milk heated in the copper vessel all the animals developed scurvy and died in about four weeks. This milk contained about 1.4 parts of copper per million. Other guinea pigs given the milk heated in the copper vessel, and in addition, two cubic centimeters of orange juice made excellent gains. The authors concluded that the trace of copper present contributed to the destruction of the antiscorbutic vitamine, and account in some degree, for the varying amount of C in the heated milk. There might also, they believe, be an effect within the animal body.

Anderson, Dutcher, Eckles, and Wilbur (1921) studied the

effect of heat and oxidation on vitamine C. Boiled milk was found to be practically equal in nutritive properties to fresh, unheated, raw, cows' milk. Pasteurized milk heated to 63°C. produced scurvy deaths in a very short time. Since this milk was in contact with the air, and the same milk, when pasteurized in a closed vessel, had not lost its antiscorbutic, they concluded that the oxidation involved was the important factor. It was also found that bubbling carbon dioxide thru milk did not alter its content of vitamine C.

Experimental.

Anderson (1921) in a thesis presented to the Graduate School of this University, studied the effect of pasteurization with and without agitation and access to air, on the antiscorbutic vitamine in cow's milk. She concluuded that the largest factor involved in the loss of the antiscorbutic property was the oxidation undergon during pasteurization. This pasteurization was done in the laboratory and not on a commercial scale.

The investigation reported in this thesis undertook to determine the diminution of the antiscorbutic factor in commercially pasteurized milk. For the purpose of this investigation, milks from three different market milk plants were used in the experiments, each having a different mechanical method of pasteurizing their milk.

In the first plant, at the Quaker Creamery Company of Minneapolis, the vat system of pasteurizing is used. This con-

sists of placing the milk in large vats, and heating to 145°F. It is then held at that temperature for thirty minutes. In this process there is a minimum of agitation and exposure to the air.

In the second plant, the Sanitary Farms' Dairies, Inc., of St. Paul, the continuous system of pasteurization is used. This is called the Simplex system, and consists of a series of pipes thru which the milk is passed at a speed at which it takes about half an hour to pass thru the pasteurizer. The milk is first pumped thru pipes surrounded by a water jacket which raises the temperature of the milk to 145°F. as rapidly as possible. At the Sanitary Farms' plant this heater comprises 288 feet of piping. The milk then flows by gravity thru the pasteurizer, where it is maintained at a temperature of 145°F by means of insulation. In this process there is but little exposure to the air, if any, and but little opportunity for agitation.

At the third plant, the Franklin Cooperative Creamery Association of Minneapolis, a combination of the two above systems, known as the Davis system, is used, where the milk is passed thru a series of vats. The milk is poured into the first one, brought to 145°F., and agitated in the process. It then flows to the next vat and the process is repeated. This tends to speed up the process, as it is not necessary to wait for all the volume of milk, but to take it in smaller quantities regularly. There is probably more exposure to the air, and more agitation in this method than in either of the two previous ones.

General Methods.

The guinea pigs used in the feeding experiments were observed for nearly a week to be sure they were normal, and free from any disease. Then, animals weighing from 200 to 350 grams were selected and placed in groups of from three to five. Each group contained animals of varying weights to offset any variation which might be brought about by a difference in weight. Each guinea pig was kept in a separate cage made of wire screening with quarter inch meshes. The cages were about eleven inches in diameter, and set in shallow pans partly filled with shavings which served as bedding. The cages were cleaned and washed with hot water about every four days, and sprayed with a cresol preparation. The animals were kept free from parasites by the use of pine oil.

The scorbutic diet used in the first part of the experimental period was the one of Sherman, La Mer and Campbell (1922) and had the following composition:

Skim milk powder	30%	Heated in an autoclave for
Ground whole oats	59%	2 hours at 110°C.
Sodium Chloride	1%	" " "
Butter fat	10%	Added after heating.

Eddy (1921) stated that this diet will produce experimental scurvy in guinea pigs in from two weeks to a month, and is complete in every respect except the antiscorbutic factor.

The animals were fed this ration ad libitum, there being no attempt at first to keep a record of the amount consumed.

After the experiment had been under way for six weeks, such records were made. The dry food was placed in small tin cups fastened to the cage in a manner in which they could be readily cleaned and filled, which was done daily. The milk was fed daily in small clean porcelain dishes, and the amount the animal failed to clean up each morning noted. The milk was usually consumed before it became contaminated with material from the cage, and before it turned sour.

The animals were weighed every four days, as a rule. Their weight was taken in the morning before either milk or dry feed was fed, in order to avoid any discrepancy arising from variations in the time of weighing.

Four weeks after the beginning of the experiment the basal scorbutic ration was changed to one very similar to that used by Hart, Steenbock and Ellis (1921) because it was felt that not enough of the original ration was being consumed by the animals. The new basal ration had the following composition:

Alfalfa hay heated in an autoclave at 15 lbs. pressure for thirty minutes, and then ground	14 %
Casein	40 %
Ground rolled oats	40 %
Ground dried spinach	5 %
Sodium Chloride	1 %

Before feeding, the ration given each animal was weighed and then moistened with water to form a soft dough or cake. Guinea pigs take moistened food better than if fed in the dry state.

Furthermore, there is less waste when handled in this manner.

The guinea pigs were examined at frequent intervals in order that any symptoms of scurvy might be detected at once. The first diagnostic sign of scurvy was that of swollen, tender ankles. When the ankles were touched or squeezed, it was found that the sharp contour of the bones had been lost, and the edges of the bones rounded. The animal winced upon touching the ankles and often cried as if in pain.

Shortly after the appearance of this swelling, the animal would begin to lose its sleek appearance, the fur becoming rough and unkempt. Cessation of growth, or a definite loss of weight would then take place. Some animals, however, maintained their weight and some even gained, but the latter were the exception, and not the general rule. As the disease progressed the animal was often found in the "scurvy position" described by Chick, Hume and Skelton (1913) in which it sits on three legs in such a manner that a tender hind leg is drawn upward and outward so as to escape pressure. At this stage of the disease the guinea pig is inactive, and often eats with difficulty, or refuses food entirely. Oftentimes it lies curled up on the floor of the cage with one side of its face resting on the floor in order to rest a painful jaw - the "face ache position" described by the above authors. Sometimes fractures can be noted in life at the head of the tibia or the lower end of the femur; such fractures are frequently developed in the course of handling.

In the last stages of scurvy the guinea pigs often become

moribund, and when this condition was noted, they were usually chloroformed, as they refused to eat their food or take their milk, and death would have resulted in a very short time at any rate.

Upon death, an animal was autopsied as soon as practicable. The points noted in the operation were as follows: The finding of subcutaneous hemorrhages at the wrists, ankles, thighs, abdomen and thoracic region; deep hemorrhages in the muscles of the legs and forearm, and in the intercostal muscles. Upon opening the body cavity, the digestive tract was examined for hemorrhages, lesions, or a distorted condition. The degree of impaction of the cecum and intestine was usually noted. The adrenal glands were examined to note hypertrophy. The stomach and liver were examined for abnormalities. The thoracic cavity was entered and the condition of the lungs noted. The costochondral junctions were examined macroscopically for evidences of beading or of hemorrhage. Lastly, the condition of the teeth, bothe the incisors and the molars, was examined as to looseness in the sockets, discoloration and soundness. A diagnosis of scurvy was made upon the macroscopic findings; the severity of the disease was classed as being either mild or severe.

Experimental Details.

In the feeding experiment the problem naturally fell into the grouping of a suitable number of guinea pigs with respect to the amount and kind of milk fed. The problem was originally attacked from two sides: the preventative, and the curative side. In the former method, the groups were fed the various milks with a view of noting any variation in their antiscorbutic property. Raw whole milk from the Sanitary Farms plant was fed to the control groups, it being thought to be representative of the milk produced in the region of the Twin Cities. Control groups were established to act as a check on the amount of antiscorbutic destroyed.

The following groups were started on November first, 1921.

Group 1. Basal ration plus 30cc. of Sanitary Farms' raw whole milk.

Group 2. Basal ration plus 30cc. of Quaker pasteurized whole milk.

Group 3. Basal ration plus 30cc. of Sanitary Farms' pasteurized whole milk.

Group. 4. Basal ration plus 30cc. of Franklin Co-operative pasteurized whole milk.

Group 5. Basal ration before autoclaving to destroy possible antiscorbutic.

Group 6. Basal ration.

In attempting the curative method of estimating the amount of loss of the antiscorbutic property, the animals in group 6

were brought down with scurvy in from fifteen to eighteen days, the swelling of the ankles being taken as a sign of definite scurvy. They were then placed in groups as follows:

Group 7. Basal ration plus 30cc. of Sanitary Farms' raw whole milk.

Group 8. Basal ration plus 30cc. of Quaker pasteurized whole milk.

Group 9. Basal ration plus 30cc. Of Franklin Co-operative pasteurized whole milk.

With the choice of a new basal ration on November 26, 1921, additions were made to the groups on the thirty cc. levels to fill out the groups, and to determine the result of the new basal. The following new group was started:

Group 10. Basal ration plus water ad libitum.

As laboratory space and animals became available the following groups were added:

Dec. 10, Group 11. Basal ration + 40cc. Franklin Co-operative pasteurized whole milk.

Dec. 11, Group 12. Basal ration + 40 cc. Quaker pasteurized whole milk.

Dec. 17, Group 13. Basal ration + 40 cc. Sanitary Farms' pasteurized whole milk.

Jan. 18, Group 14. Basal ration + 40 cc. Sanitary Farms' raw whole milk.

Jan. 24. Group 15. Basal ration + 50 cc. Quaker pasteurized whole milk.

Feb.4, Group 16. Basal ration + 50 cc. Sanitary Farms' raw whole milk.

Feb.8, Group 17. Basal ration + 50 cc. Sanitary Farms pasteurized whole milk.

Feb.9, Group 18. Basal ration + 50 cc. Franklin Co-operative pasteurized whole milk.

Discussion and Interpretation of Results

In the first series of four groups, the various milks under consideration, Sanitary Farms' raw and pasteurized whole milk, Franklin Co-operative and Quaker Creamery pasteurized milk, were fed at a thirty cc. level. Examination of the data concerning this series (Chart I, Table I) failed to show any striking differences. The animals, excepting those which succumbed to disease, developed scurvy in uniform time, and died, as a rule, in from four to five weeks. In the raw milk group there were two survivals. One animal No. 442 lived 77 days, and then died with severe scurvy. Another, No. 450, survived the entire period of 91 days with no scurvy. On 30 cc. Franklin pasteurized milk, one animal, No. 425 survived 103 days showing symptoms of mild scurvy only.

In the second series where the preventative method of estimation of the antiscorbutic was used, the first group of check animals on the basal ration with no milk, succumbed to scurvy or disease in three weeks. Where 30 cc. of Sanitary Farms' raw whole milk, and Quaker and Franklin Co-operative pasteurized whole milks were fed after scurvy developed, no differences were noticeable. Scurvy developed uniformly in from two to three weeks, and scurvy deaths resulted in less than a month. The animals died as rapidly as if no milk had been fed. Neither the raw nor the pasteurized milk served to bring the animals out of their scorbutic condition.

In the third series fed, the attempt to measure the anti-

scorbutic property by the preventative method was abandoned as positive results seemed difficult to obtain. The basal ration was also adopted, which was consumed more readily by the animals. At this level 40 cc. of the various milks were fed daily to each guinea pig. Group 10, on this diet plus water all developed scurvy and died in a little over two weeks.

The striking feature noted in an examination of the data in the charts and tables for this series was the marked protection offered by the raw milk control group. None of the animals in this group (Nos. 459 - 463) showed scurvy symptoms during life. When taken off experiment after an average of 90 days, only mild scurvy signs were observed. When it is remembered that this milk was produced during the winter months, and was representative of the milk produced in this region, the results do not indicate as serious a deficiency in the antiscorbutic properties as one might be led to expect from the study of the literature. For example, Dutcher and co-workers (1920) stated that "20 cc. of summer produced milk were superior in nutritive value and antiscorbutic potency to 60 cc. of winter milk." The latter milk was produced, however, under experimental conditions. Group 11 showed that Franklin pasteurized milk had lost proportionately more of the antiscorbutic factor than the other two pasteurized milks, for scurvy symptoms developed in from three to four weeks, and death ensued in forty days. The Franklin Co-operative and Sanitary Farms' pasteurized milks showed nearly identical results, the scurvy symptoms being more marked in life in the case

of the former, but the survival period longer. Some of the animals on Franklin Co-operative pasteurized milk compared favorably with the controls on raw milk.

In the fourth series of guinea pigs, groups 15 to 18, the various milks were fed at a fifty cc. level. As in the 40 cc. series, the control animals on raw milk showed marked protection. The majority of animals in this group, Nos. 469, 470, 471, and 432 survived the experimental period of about seventy days with no, or only mild scurvy symptoms. In apparent contradiction to the results obtained at the forty cc. level, the animals in group 18, on Franklin Co-operative pasteurized whole milk showed milder symptoms, and a longer survival period than the other groups, for after concluding the sixty day experimental period, they showed mild scurvy symptoms only. In the case of the Quaker Creamery and Sanitary Farms' pasteurized whole milk the disease was more severe with a shorter survival period. One animal, No. 464, however, survived 33 days without developing scurvy symptoms on 50 cc. Quaker pasteurized milk. As a whole, the Quaker milk in this series appeared to suffer the greatest diminution of the antiscorbutic, while the animals on the Sanitary Farms' pasteurized milk reacted in much the same manner as those of the control raw milk group.

It was the purpose of this investigation to note any variations occurring in the diminution of the antiscorbutic potency of commercially pasteurized milks produced by the three distinct systems of pasteurization employed in the plants where the milk

was obtained. No such variations were present at the 30 cc. levels, as an examination of the data will show. It is apparent that at the 40 cc. level the Franklin Co-operative milk did not offer the same degree of protection the other groups did. At the 50 cc. level the Franklin was on a par with the Sanitary Farms' m milk, but, at the same time, the Quaker milk seemed to have lost much of its protective power against scurvy. These contradictions indicate that there is but little, if any, variation in the different milks, as regards their power to prevent or delay scurvy in guinea pigs.

As a possible explanation of these contradictions it should be remembered that the rations employed on the farms supplying the milk to the three different milk plants were probably not entirely uniform. It is known, for example, that the milk supply to the Sanitary Farm Dairies comes largely from a region lying southeast of the Twin Cities, while that supplied to the Franklin Co-operative Creamery comes largely from the region lying to the northwest of the Twin Cities.

A comparison of the results secured in the raw milk groups in each series shows that the group of animals on 50 cc. raw milk in Series 4, furnished slightly less protection than 40 cc. in Series 3. Series 3. was started Jan. 18, while the raw milk group in Series 4 on 50 cc. was started Feb. 4th. The indications are of a slight seasonal diminution in the antiscorbutic potency of the raw milk as the winter feeding season advanced.

Conclusions.

1. Milks pasteurized by three commercial plants each using distinct systems of pasteurization, namely, the Davis, Simplex, and Vat systems, when fed to guinea pigs show no marked variation in the diminution of the antiscorbutic potency.

2. The best grades of pasteurized market milk sold in the Twin Cities are somewhat less potent as an antiscorbutic than the same milk before pasteurization, but this difference largely disappears when the two types of milk are fed to guinea pigs at a level of 50 cc. a day. Since 40 cc. of raw milk protected guinea pigs from scurvy in these experiments, while 40 cc. of pasteurized milk did not do so, the indications are that pasteurization of milk as carried out commercially in the Twin Cities destroys about 20 to 25 percent of the antiscorbutic potency of the raw milk.

3. Pasteurized milk sold in the Twin Cities during the winter months undergoes no marked seasonal diminution in its antiscorbutic potency as the season advances.

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Appendix.

Protocols of each guinea pig in each group.

Group I.

Pig 419. No signs of scurvy during life. No hemorrhages present on autopsy. Intestine congested. Colon impacted. Condition pointed to acute constipation. Rib junctions normal.

Pig 420. No scurvy symptoms during life, save that the intestines were protruding in such a manner as to make chloroforming advisable. Autopsy showed hemorrhages present in the hind legs. Large intestine, small intestine, and lungs normal. Wrists swollen. One rib beaded at the costochondral junction.

Pig. 421. No signs of scurvy during life. On autopsy - no hemorrhages in legs. Large intestine congested. All lobes of lungs, particularly the lower ones congested. No evidence of scurvy. Pneumonia.

Pig 442. No scurvy symptoms during life. Autopsy showed old hemorrhages present in legs. Cecum and large intestine full of air locks. Small intestine empty, distorted, hemorrhagic. Kidneys normal - adrenals very much enlarged. Stomach empty. Last two true and first two floating ribs very badly beaded. Lower molars loose and brittle - other teeth solid.

Pig. 443. Scurvy symptoms in thirteen days. Moribund on 39th day, killed. Autopsy: wrists swollen. Hind legs slightly hemorrhagic. Cecum full of pasty,yellowish feces, distorted, hemorrhagic, and full of air locks. Large and small intestines practically empty, but air locks were noted. Stomach empty.

Lungs normal. Ribs slightly beaded. Teeth solid. Adrenals enlarged. Swelling present in tendon of side of jaw.

Pig 450. No symptoms during life, nor upon autopsy after 91 days on diet. Physical condition excellent.

Group 2.

Pig 428. Scurvy symptoms in 26 days, but survived for 69 days. Autopsy: no hemorrhages in hind legs. Cecum impacted and hemorrhagic. Small intestine hemorrhagic but empty. Large intestine distended and distorted. Air locks in intestine. Stomach bloated, with no solid food in it. Adrenal glands very much enlarged. One rib beaded. Lungs normal. Teeth solid. Wrists not hemorrhagic.

Pig. 429. No scurvy signs during life. Autopsy: Hemorrhages in hind legs. Lesions in large intestine. Small intestine hemorrhagic. Ribs beaded. Gall bladder noticeably enlarged. Lungs congested.

Pig. 430. Scurvy noted after 26 days. Survived for 33 days. Autopsy. Legs normal. Cecum hemorrhagic. Stomach and small intestine empty. Slight beading of the rib junctions present.

Pig 439. Scurvy symptoms noted in 17 days, but animal survived for 40 days. Autopsy. At death was bloated. Old hemorrhages present in hind legs. Bloody liquid found in abdominal cavity. Cecum distended, hemorrhagic, ulcerated, full of gas. Small intestine empty and hemorrhagic. Stomach distended with gas, and partially filled with milk. Air locks in large intestine. It was distorted and hemorrhagic. Liver spotted, and unnatural in

coloration. Ribs badly beaded. Adrenals enlarged. Teeth solid. Digestive disorders present.

Group 3.

Pig 422. No scurvy symptoms during life. Intestine found protruding on 23 day. Chloroformed. Large intestine and stomach very impacted. Lower portion of digestive tract very constipated, pointing to acute constipation. No scurvy signs apparent.

Pig 423. Severe diarrhea on the 8th day. Chloroformed. Lungs and intestines normal. Ribs not beaded. No scurvy.

Pig 424. Tender wrists on 16th day. At death acute constipation was only disorder found.

Pig. 431. Scurvy sign noted 5th day. Badly paralyzed prior to death. Wrists swollen. Legs normal. Ribs badly beaded. Adrenals enlarged. Stomach empty. Small intestine abnormal and filled with air locks. Head drawn back at death.

Pig 432. No symptoms during life. Hind legs slightly hemorrhagic. Intestines and stomach practically empty. Ribs slightly beaded. Lungs spotted.

Pig 433. Wrists quite swollen on 17th day. Legs and wrists very hemorrhagic. Stomach and intestines empty. Air locks present. Ribs beaded. Lungs normal. Adrenals enlarged. Molar teeth loose.

Pig 434. Wrists tender on 20th day. Autopsy. Right front wrist badly swollen - no sign on left one. Knees not hemorrhagic. Ribs badly beaded. Small intestines empty, with air pockets. Large intestine and colon impacted. No intestinal hemorrhage.

suprarenals greatly enlarged. Liver normal. One lobe of lung darkened. Scurvy present.

Pig 435. No symptoms during life. Autopsy showed hind legs normal. Cecum distended with pasty feces. Stomach and small intestines empty. Ribs normal. Upper lobes of lungs nearly destroyed. Suprarenals enlarged. Pneumonia.

Pig 440. Had tender wrists on the 12th day. Autopsy showed-hind legs hemorrhagic; ribs beaded; cecum hemorrhagic and impacted; large intestine hemorrhagic and empty; adrenals enlarged; left lobes of lungs very congested; air locks in intestine.

Group 4.

Pig. 425. No scurvy symptoms during life. Autopsy showed hemorrhagic thighs; small intestine hemorrhagic and empty; stomach empty; adrenals enlarged; line of Fraenkel evident; beading of ribs faint; upper lobes of lungs very congested; teeth misplaced but solid, as if they had once been loose, and had later set crooked; upper incisors broken; scurvy slight, but pneumonia was in evidence.

Pig 426. No scurvy symptoms during life. Autopsy showed normal hind legs; songested and hemorrhagic large intestine; small intestine and stomach empty; lungs normal, adrenals enlarged.

Pig 427. No scurvy symptoms during life. Autopsy showed normal hind legs; normal ribs; congested large intestine.

Pig 444. No scurvy symptoms during life. Autopsy showed legs, intestines and stomach normal; Lungs and heart region very congested; ribs slightly beaded. Pneumonia cause of death.

Group 5.

Pig 404. Swollen wrists on the 14th day. Autopsy showed old hemorrhage on legs; edema in viscera; large intestine normal; stomach and small intestine empty; ribs badly beaded.

Pig 405. No scurvy symptoms during life. Autopsy showed congestion in the lungs indicative of severe pneumonia.

Pig. 406. No scurvy symptoms during life. Autopsy showed internal hemorrhage in the abdominal cavity; adrenals enlarged; stomach and small intestines empty; large intestine congested; lower lobes of lungs congested.

Group 7.

Pig 411. Had tender wrists on the 13th day. Autopsy showed protruding intestine; hind legs hemorrhagic; digestive tract empty; ribs beaded; lungs congested; lung trouble, scurvy and starvation in evidence.

Pig 413. Had swollen wrist on the 22nd day. Autopsy showed hemorrhagic hind legs; small intestine and stomach empty; large intestine partially so; adrenals enlarged; lower lobes of lungs especially congested; ribs beaded; teeth solid. Scurvy with complications present.

Pig 416. Wrists swollen on the 14th day. Just prior to death had violent diarrhea. Badly paralyzed. Autopsy showed hemorrhagic hind legs; ribs badly beaded.

Group 8.

Pig 404. Given in group 5.

Pig 408. Had swollen wrists on 14th day. Autopsy showed hind

legs to be slightly hemorrhagic; large intestine congested and hemorrhagic; small intestine empty; stomach empty; ribs badly beaded; teeth solid. Scurvy present.

Pig 409. Wrists swollen on 13th day. Autopsy showed the hind legs and the large and small intestines to be hemorrhagic; ribs beaded; lungs congested; wrists swollen; death due to scurvy.

Pig 417. Swollen wrists on 14th day. Had abscessed jaw so was chloroformed. Autopsy showed badly beaded ribs; hind legs hemorrhagic; large ulcer with offensive odor on jaw.

Group 9.

Pig 407. Wrists swollen on 13th day. Severe diarrhea in evidence prior to death. Autopsy showed hemorrhages in fore legs; stomach and intestines empty; intestines hemorrhagic; ribs beaded; signs of starvation present.

Pig 414. Wrists swollen on 13th day. Autopsy showed severe scurvy present; hemorrhages in hind legs; ribs beaded.

Pig. 418. Wrists swollen on 14th day. Autopsy failed to show hemorrhagic condition of the hind legs; intestines hemorrhagic; small intestines and stomach empty; large intestines full of fecal matter; lungs spotted; ribs slightly beaded.

Group 10.

Pig 436. No signs of scurvy during life. Autopsy showed congested small intestine; air locks in large intestine; ribs normal; death due to unknown cause.

Pig. 437. No scurvy symptom during life. Autopsy showed scurvy present; very emaciated; acute constipation of lower bowel large

factor in death.

Pig 438. Scurvy symptom, swollen wrists noted on eleventh day. Autopsy showed slight hemorrhages of hind legs; intestines empty save for gas; stomach empty; ribs beaded.

Pig 446. No scurvy symptoms during life. Diarrhea in evidence prior to death. Autopsy showed impacted cecum; small intestine and stomach empty, legs not hemorrhagic; ribs slightly beaded. Complications present.

Group 11.

Pig 441. No scurvy symptom present during life. Left wrist swollen at death, bloated. Autopsy showed very hemorrhagic hind legs; subcutaneous hemorrhages present in flank; some edema in abdominal cavity; cecum hemorrhagic, and very impacted with greenish, hard feces, stomach empty of solid matter; bloated; the stoppage of the cecum had filled the large and small intestine and the stomach with gas; air locks present in large intestine; adrenals quite enlarged; slight beading of ribs on right side, with marked beading of three rib junction on left side; molars of right lower jaw were irregular, as if they had been loose, and then set crooked. Acute constipation resulting in bloating produced death.

Pig 447. Right wrist swollen on twenty sixth day. Diarrhea in evidence prior to death. Autopsy showed old hemorrhages present in hind legs; cecum filled with greenish pasty feces; air locks in large intestine; stomach and small intestine empty; adrenals very much enlarged; line of Fraenkel and beading of ribs very distinct; lungs congested; lpwer molars on right side loose; scurvy with com-

plications.

Pig 454. Wrists tender on twenty third day. Very bad diarrhea in evidence prior to death. Wrists swollen. Left femur fractured. Autopsy showed very hemorrhagic hind legs; air locks in large intestine; line of Fraenkel marked; ribs badly beaded; molar teeth in both jaws loose in their sockets. Severe scurvy present.

Group 12.

Pig 445. No scurvy symptoms during life. Autopsy showed the stomach filled with gas and badly ulcerated; adrenals twice normal size; small intestine empty; large intestine very impacted.

Pig 449. No scurvy symptoms during life. Suffered with chronic constipations through experiment. Chloroformed at end of experiment. Autopsy showed the colon, cecum and stomach abnormally large, probably three times their normal size; all were partially inflated with gas, but a large amount of pasty feces was retained; small intenstine full of air locks; rectum obstipated; adrenals slightly enlarged; some fatty degeneration in liver; ribs normal; teeth solid. No scurvy symptoms.

Pig 453. Wrists tender on twentieth day. Moribund, and so was chloroformed. Autopsy showed fractured wrists; hind legs very hemorrhagic; cecum distended, bloated and distorted; large and small intestines empty and very hemorrhagic; ribs quite badly beaded; stomach empty; adrenals very much enlarged; teeth solid; scurvy pronounced.

Pig 455. Assumed "face ache" position on thirty third day. Chloroformed at end of experiment. Autopsy showed old deep

hemorrhages present in the hind legs; intestines normal; adrenals slightly enlarged and somewhat hemorrhagic; fatty degeneration in the liver; ribs normal; bones of hind legs brittle. Slight scurvy.

Pig 457. Assumed 'face ache' position on the 17th day. Chloroformed at the end of the experiment. Post mortem showed subcutaneous and deep hemorrhages present in the hind legs, and in the left fore leg; physical condition good; adrenals slightly enlarged; intestines full of air locks; colon and cecum normal; lungs normal; bones of hind legs very brittle; one lower incisor and several molars loose; all molars discolored; mild scurvy.

Pig 458. Assumed 'face ache' position on 11th day. Autopsy showed left hind leg very hemorrhagic; femur fractured; hemorrhage in evidence at costochondral junction on right side; cecum filled with pasty yellow feces; small intestine filled with air locks; stomach empty; adrenals greatly hypertrophied; ribs badly beaded; line of Fraenkel very evident; lungs congested and spotted (possibly tuberculosis lesions); right adrenal had blood clot on anterior end; lower molars all loose; severe scurvy and pulmonary disease present.

Group 13.

Pig 451. No scurvy symptom during life. Autopsy showed slight hemorrhage in hind legs; adrenals very much enlarged and spotted; intestines nearly empty; cecum distorted; air locks in small intestine; ribs but slightly beaded. Mild scurvy?

Pig 452. No scurvy symptoms during life. Chloroformed at end of

experiment. Autopsy showed hemorrhagic left hind leg; colon and cecum normal; air locks present in the partially empty intestine; stomach normal; fatty degeneration in liver; left adrenal enlarged and hemorrhagic, right normal; marked beading in several ribs; lungs normal; teeth normal but quite discolored. Scurvy advanced.

Pig 455. Wrists swollen and very tender on 30th day. Became moribund so was chloroformed. Wrists swollen. Autopsy showed subcutaneous and deep hemorrhages on legs; also on intercostal muscles; likewise in the deep pectoral muscles; colon and cecum filled with greenish pasty feces; intestines empty; fatty degeneration in liver; ribs badly beaded; adrenals very much enlarged; lungs badly congested; stomach empty; teeth solid; severe scurvy plus pneumonia.

Pig 483. Wrists tender on 20th day. Became moribund and was chloroformed. Wrists swollen and fractured. Bones of leg brittle. Autopsy showed old and recent subcutaneous and deep hemorrhages in fore and hind limbs, and in intercostal muscles; intestines empty; ribs very badly beaded; fatty degeneration in liver; adrenals hemorrhagic, but not enlarged; lungs slightly congested; stomach normal; teeth somewhat loose and discolored brown. Severe scurvy.

Group 14.

Pig 460. No scurvy symptom during life. Chloroformed at end of experiment. Autopsy failed to reveal any subcutaneous or deep hemorrhages of legs; joints not swollen; cecum slightly impacted

and hemorrhagic; intestines empty; adrenals normal; stomach empty; fatty degeneration of liver present; teeth discolored and slightly loose; necrosis noted in tissue next to ribs; lungs normal. No definite scurvy.

Pig 461. No scurvy symptoms during life. Autopsy showed slight subcutaneous and deep hemorrhages in hind legs; colon quite hemorrhagic; intestinal tract filled with pasty brownish feces; air locks in cecum; stomach full of gas, ulcers present; ribs not beaded but one was hemorrhagic; adrenals very much enlarged; lungs not congested; teeth solid; slight scurvy.

Pig 462. No scurvy symptom during life. Chloroformed at end of experiment. Autopsy showed subcutaneous and deep hemorrhages in hind legs; digestive tract normal; liver normal; stomach normal; lungs slightly spotted; left adrenal slightly hemorrhagic; ribs normal; bones of hind legs brittle; lower incisor broken; one lower molar rotten; mild scurvy.

Pig 463. No scurvy symptom during life. Left hind leg broken in fall on 56th day. Chloroformed at end of experiment. Autopsy showed hemorrhage in region of break in femur; none in other limbs; intestines normal; fatty degeneration of liver very marked thoracic cavity filled with blood; lungs very badly congested; before killing difficulty in breathing was observed; teeth solid. No scurvy.

Pig 459. No scurvy symptoms present during life. Chloroformed at end of experiment. Autopsy showed hemorrhagic hind legs; normal digestive tract; liver normal; adrenals normal; ribs not

beaded; lungs normal; teeth slightly discolored; animal in good physical condition; mild scurvy.

Group 15.

Pig 464. No scurvy symptoms during life. Chloroformed at end of experiment. Autopsy showed none of the usual scurvy symptoms; internal organs normal; physical condition excellent.

Pig 465. Had swollen and tender wrists and suffered internal hemorrhage on 23rd day. Two days later was moribund, so was chloroformed. Autopsy showed very hemorrhagic fore and hind legs. Right femur broken; bones of leg very fragile; ribs badly beaded; several ribs broken and very hemorrhagic; fatty degeneration in the liver; adrenals slightly enlarged; stomach partially empty; small intestine empty; large intestine and cecum congested, air locks were present; heart normal; lungs spotted; teeth solid; wrists quite swollen.

Pig 466. No scurvy symptoms during life. Moribund - chloroformed. Autopsy showed complete fracture of the left tibia - other bones of body very brittle; colon and cecum very impacted, distorted, and hemorrhagic; stomach empty of solid material but full of gas; small intestine empty; large intestine impacted; digestive tract full of air locks; teeth solid; adrenals enlarged; lungs normal; line of Fraenkel present but costochondral junctions of ribs were not noticeably beaded; death due to obstipation.

Group 16.

Pig 463. No signs of scurvy during life. Autopsy showed no hemorrhages present; stomach and intestines empty; adrenals very

much enlarged; ribs normal; lungs very congested; cecum filled with black pasty feces. Bronchial disorder present.

Pig 469. Inanition and rough fur evident on 32d day. Chloroformed at end of experiment. Autopsy revealed emaciated condition; diarrhea in evidence prior to death; marked subcutaneous and deep hemorrhages of joints of hind legs, and intercostal muscles of right side; left adrenal very much enlarged and quite hemorrhagic digestive tract practically empty of any fluid or solid matter; stomach empty; air locks in intestine; ribs normal; some congestion in lungs; teeth not discolored; tibia and fibia brittle; advanced scurvy.

Pig 470. No symptoms of scurvy during life. Chloroformed at the conclusion of the experiment. On autopsy showed none of the usual scurvy symptoms; no hemorrhages; digestive tract normal; adrenals normal; ribs normal, also lungs.

Pig 471. No symptoms of scurvy during life. Was chloroformed at end of experiment. No symptoms of scurvy evident on autopsy; no hemorrhages present in fore or hind legs, or in intercostal muscles; left adrenal gland hemorrhagic; intestine normal; stomach pinched at pyloric end, and distended at cardiac end; ribs and liver normal; teeth solid; slightly discolored teeth; necrosis of tissue along ribs on left side; no scurvy.

Pig 472. No symptoms of scurvy during life. Was chloroformed at end of experiment, as nervous disorder spoiled animal for use. No sign of scurvy on autopsy.

Pig 482. No symptoms of scurvy during life. Was chloroformed at

end of experiment. Autopsy showed distinct hemorrhages in left leg; several hemorrhagic spots on solon; intestines normal; slight obstipation of lower bowel; left adrenal hemorrhagic; fatty degeneration of liver; ribs and lungs normal,; teeth sound; Milk scurvy.

Pig 435. Suffered internal hemorrhage on 30th day. Moribund five days later; so was chloroformed. Autopsy showed subcutaneous and deep hemorrhages on left hind leg; large and small intestine filled with air locks and hemorrhagic; colon filled with pasty brawn feces and air lock; left suprarenal enlarged; one rib slightly beaded; lungs totally hemorrhagic; fatty degeneration in liver; stomach empty of solids but distended with gas; teeth solid; bones of left hind leg brittle; slight scurvy plus lung trouble.

Group 17.

Pig 474. Wrists swollen on 27th day. Chloroformed at conclusion of experiment. Autopsy showed subcutaneous and deep hemorrhages in hind legs, and in pectoral and intercostal muscles; digestive tract normal; liver normal; left adrenal enlarged; stomach normal; three ribs badly beaded; lungs normal; teeth discolored; molars loose and rotted, being easily extracted; bones of hind legs very brittle; fur rough. Advanced scurvy.

Pig 475. Wrists swollen and tender on 9th day. Right hind leg broken, so animal was chloroformed. Autopsy showed the hind legs, especially the broken one, very hemorrhagic; intercostal muscles and junctions of ribs of right side hemorrhagic; left front foot

hemorrhagic; fatty degeneration in liver; stomach normal; large intestine distorted with air locks present and partially emptied; adrenals enlarged and hemorrhagic; ribs beaded and hemorrhagic, with lines of Fraenkel present; lungs congested; teeth solid.

Pig 476. Wrists swollen on 17th day. Chloroformed at end of experiment. Autopsy showed marked subcutaneous and deep hemorrhages present in front and hind lets, especially in the former; Intercostal muscles hemorrhagic; colon very hemorrhagic; cecum normal; intestines full of air locks; adrenals hemorrhagic but not enlarged; liver normal; several ribs markedly beaded; lungs congested; teeth solid. Severe scurvy.

Pig 477. Had internal hemorrhage on 17th day. Wrists swollen at death and left one fractured. Autopsy showed subcutaneous hemorrhages on both fore and hind legs; deep hemorrhages in hind leg muscles; colon filled with pasty yellow feces; badly hemorrhagic; intestines empty; cecum hemorrhagic; kidneys normal; adrenals slightly enlarged; stomach empty and hemorrhagic; fatty degeneration in the liver; ribs badly beaded and hemorrhagic; lungs and heart normal; teeth solid; typical scurvy signs present; bones of hind leg very brittle.

Group 13.

Pig 478. Wrists swollen on 22th day. chloroformed at conclusion of experiment. Autopsy showed subcutaneous and deep hemorrhages in hind legs; colon and cecum normal; air locks in distorted intestine; left adrenal enlarged and hemorrhagic; ribs normal; lungs spotted; liver normal; teeth discolored; stomach inflated;

mild scurvy.

Pig 479. Internal hemorrhage on 56th day. chloroformed at end of experiment. Autopsy showed slight hemorrhage in deep muscles of hind legs; digestive tract normal; ribs normal; some fatty degeneration in liver; lungs spotted; adrenals normal; teeth discolored but sound; mild scurvy.

Pig 480. No scurvy symptoms during life. Wrists not swollen at death. Autopsy showed subcutaneous and deep hemorrhages in hind legs; no impaction in cecum; cecum filled with semifluid brownish feces; large and small intestines empty; stomach empty; adrenals enlarged; ribs very badly beaded, and line of Fraenkel present; lungs normal; molars loose in sockets and rotten; severe scurvy.

Pig 481. No scurvy symptom during life. Autopsy showed hemorrhage in hind legs; cecum filled with pasty yellow feces; hemorrhages present in cecum, stomach, large and small intestines; small intestine empty; watery edema in abdominal viscera; ribs normal; adrenals normal; teeth solid; lungs congested to which death was probably due.

Pig 484. Right wrist swollen but not tender on 27th day. Chloroformed at end of experiment. Autopsy showed marked subcutaneous and deep hemorrhages in hind legs; digestive tract normal save for small hemorrhagic spots on colon; fatty degeneration in liver ribs and lungs normal; adrenals normal; teeth discolored but sound; mild scurvy.

No.	Started	First sympt.	Died	Length of exp.	Initial weight	Final weight	Max. wt.	% gain or loss	Cause of death
Group 1. Basal ration ad lib. + 30 cc. Sanitary Farms' raw whole milk									
419	Nov. 1	---	Nov. 28	28	250	255	286	+2	Acute constipation
420	Nov. 1	27	Nov. 27	27	250	230	251	-8	Scurvy
421	Nov. 1	---	Nov. 22	22	206	181	213	-12	Pneumonia
442	Nov. 26	---	Feb. 11	77	197	160	259	-18	Severe scurvy
443	Nov. 26	13	Jan. 4	39	277	210	277	-24	Killed; Mild scurvy
450	Dec. 30	---	Mar. 31	91	224	306	312	+32	Killed, no scurvy
Group 2. Basal ration ad lib. + 30 cc. Quaker pasteurized whole milk.									
428	Nov. 1	26	Jan 9	69	228	125	231	-45	Scurvy
429	Nov. 1	---	Nov. 26	26	282	193	282	-31	Scurvy + Constipation
430	Nov. 1	26	Dec. 4	33	256	160	256	-36	Mild scurvy
439	Nov. 26	17	Jan. 5	40	280	220	292	-21	Severe scurvy +complications
Group 3. Basal ration ad lib. + 30 cc. Sanitary Farms' Pasteurized whole milk									
422	Nov. 1	---	Nov. 23	23	285	237	285	-17	No scurvy, constipation
423	Nov. 1	---	Nov. 8	8	215	190	215	-11	Unknown
424	Nov. 1	16	Nov. 21	21	226	224	235	- 1	No S. Acute constipation
431	Nov. 9	5	Dec. 2	30	300	190	300	-36	Scurvy
432	Nov. 14	9	Nov. 24	10	293	180	293	-35	Scurvy + constipation
433	Nov. 26	17	Jan. 1	30	241	157	248	-35	Scurvy
434	Nov. 26	20	Dec. 24	28	220	160	227	-25	Scurvy + Complication
435	Nov. 26	---	Dec. 4	8	171	141	171	-17	Pneumonia
440	Dec. 4	12	Jan. 3	30	192	175	257	- 9	Scurvy + pneumonia

No.	Started	First sympt.	Died	Length of exp.	Initial weight	Final weight	Max. wt.	% gain or loss	Cause of death
Group 4. Basal ration ad lib. + 30 cc. Franklin Co-op. Past. whole milk.									
425	Nov. 1	--	Feb. 5	103	273	142	287	-48	Mild scurvy + pneumonia
426	Nov. 1	--	Nov. 21	21	232	164	192	-12	No S. Unknown
427	Nov. 1	--	Nov. 28	28	236	228	297	-20	No. S. Unknown
444	Nov. 26	--	Dec. 21	25	165	195	207	+18	Mild S. + pneumonia
Group 5. Basal ration ad libitum (unautoclaved									
404	Nov. 1	14	Nov. 26	26	272	187	237	-31	Scurvy
405	Nov. 1	--	Nov. 11	11	215	175	216	-13	Pneumonia
406	Nov. 1	--	Nov. 21	21	236	153	243	-33	Lungs congested
Group 6. Basal ration ad libitum.									
407	Nov. 1	18	Nov. 25	25	278	165	292	-39	Scurvy
408	Nov. 1	14	Nov. 28	28	215	196	243	- 7	Scurvy
409	Nov. 1	13	Nov. 27	27	284	184	284	-35	Scurvy
410	Nov. 1	14	Nov. 15	15	210	170	210	-19	Scurvy + complications
411	Nov. 1	18	Nov. 23	23	270	145	287	-45	Scurvy + complications
412	Nov. 1	--	Nov. 15	15	296	252	296	-15	Pneumonia
413	Nov. 1	22	Nov. 23	23	288	188	289	-34	Scurvy + complications
414	Nov. 1	18	Nov. 20	20	263	173	275	-34	Scurvy
415	Nov. 1	14	Nov. 18	18	395	195	311	-36	Scurvy
416	Nov. 1	14	Nov. 23	23	240	184	243	-23	Scurvy + complications
417	Nov. 1	14	Nov. 17	17	220	196	232	-10	Scurvy, killed due to abcess.
418	Nov. 1	14	Nov. 25	25	224	144	224	-35	Mild scurvy

Cure attempted on No. 411 on Nov. 13 with 30 cc. Sanitary Farms' raw whole milk.
" " " " 413 " " 15 " " " " " " "
" " " " 416 " " 15 " " " " " " "
" " " " 404 " " 15 with 30 cc. Quaker pasteurized whole milk.
" " " " 408 " " 15 " " " " " " "
" " " " 409 " " 13 " " " " " " "
" " " " 417 " " 15 " " " " " " "
" " " " 407 " " 18 with 30 cc. Franklin Co-op past. whole milk.
" " " " 413 " " 13 " " " " " " "
" " " " 414 " " 18 " " " " " " "

No.	Started	First sympt.	Died	Length of exp.	Initial weight	Final weight	Max. wt.	% gain or loss	Cause of death
Group 10. Basal ration ad lib. + water									
436	Nov. 26	---	Nov. 29	3	230	202	245	-12	Unknown
437	Nov. 26	---	Dec. 11	15	220	130	223	-31	Scurvy + constipation.
438	Nov. 26	11	Dec. 3	12	207	140	212	-32	Scurvy.
446	Nov. 30	---	Dec. 15	15	235	127	235	-46	mild scurvy + complication
Group 11. Basal ration ad lib. + 40 cc. Franklin Co-op. past. whole milk									
441	Dec. 10	---	Jan. 19	40	228	147	230	-35	Scurvy + constipation
447	Dec. 10	26	Jan. 18	39	237	147	237	-37	Scurvy + complication
454	Dec. 10	23	Jan. 19	40	281	180	361	-36	Scurvy + diarrhea

No.	Started	First sympt.	Died	Length of exp.	Initial weight	Final weight	Max. wt.	% gain or loss	Cause of death
Group 12. Basal ration ad lib. + 40 cc. Quaker pasteurized whole milk.									
445	Dec. 9	--	Dec. 19	10	201	150	201	-25	Unknown.
449	Dec. 22	--	Apr. 1	100	226	221	285	- 2	Killed; no scurvy
453	Dec. 11	20	Jan 12	33	260	139	230	-46	Scurvy
455	Dec. 11	35	Apr. 5	115	254	286	311	+12	Killed; mild scurwy
457	Jan. 4	17	Apr. 6	92	250	354	354	+41	Killed; mild scurvy
458	Jan. 6	11	Jan. 27	21	217	160	256	-26	Severescurvy
Group 13. Basal ration ad lib. + 40 cc. Sanitary Farms' past. whole milk.									
451	Dec. 17	--	Feb 12	57	216	132	216	-39	Mild scurvy
452	Dec. 17	--	Apr. 1	105	301	264	314	-12	Killed; severe S.
456	Dec. 17	30	Mar. 3	76	267	247	306	- 7	Killed; severe S.
433	Feb. 13	20	Mar. 27	33	304	260	333	-14	Killed; severe S.
Group 14. Basal ration ad lib. + 40 cc. Sanitary Farms' raw whole milk.									
460	Jan. 13	--	Apr. 7	94	247	209	259	-14	Killed; no scurvy
461	Jan. 13	59	Mar. 13	59	246	222	297	-9	Mild scurvy
462	Jan. 13	--	Apr. 11	93	203	359	392	+76	Killed; mild scurvy
463	Jan. 13	--	Apr. 3	90	195	280	281	+43	Killed; no scurvy
459	Jan. 13	--	Apr. 10	92	208	390	390	+87	Killed; mild scurvy
Group 15. Basal ration ad lib. + 50 cc. Quaker pasteurized whole milk.									
464	Jan. 24	--	Apr. 17	83	206	388	388	+88	Killed; no scurvy
465	Jan. 24	23	Feb. 13	25	242	256	283	+ 5	Scurvy
466	Jan. 24	--	Mar. 1	36	228	225	274	-1	Mild s.obstipation
473	Feb. 6	--	Mar. 2	22	333	196	333	-47	Scurvy

No.	Started	First sympt.	Died	Length of exp.	Initial weight	Final weight	Max. wt.	% gain or loss	Cause of death
Group 16. Basal ration ad lib. + 50 cc. Sanitary Farms' Raw Whole Milk									
468	Feb. 4	--	Feb. 19	15	298	222	311	-25	Pneumonia?
469	Feb. 4	32	Apr. 7	62	349	243	349	-27	Killed; advanced S.
470	Feb. 4	--	Apr. 17	72	377	412	413	+ 8	Killed; no scurvy
471	Feb. 4	--	Apr. 17	72	317	425	425	+34	Killed; no scurvy
472	Feb. 4	--	Feb. 16	12	302	282	308	- 7	Nervous trouble
482	Feb. 13	--	Apr. 17	53	345	484	484	+40	Mild scurvy; killed
485	Feb. 27	30	Apr. 5	37	360	237	404	-31	Killed; mild scurvy +respiratory dis.
Group 17. Basal ration ad lib. + 50 cc. Sanitary Farms' Past. whole milk									
474	Feb. 3	27	Apr. 14	65	319	234	319	-26	Killed; advanced S.
475	Feb. 3	9	Feb. 17	9	310	333	333	+ 7	Mild S. + Complication
476	Feb. 3	17	Apr. 14	65	290	224	290	-23	Killed; severe S.
477	Feb. 3	17	Mar. 2	22	340	227	340	-33	Scurvy
Group 13 Basal ration Ad lib. + 50 cc. Franklin Co-op. past. whole milk									
478	Feb. 9	22	Apr. 15	65	357	303	357	-15	Killed; mild scurvy
479	Feb. 9	56	Apr. 15	65	355	320	355	-10	Killed; mild scurvy
480	Feb. 9	--	Mar. 1	20	330	168	330	-49	Severe scurvy
481	Feb. 9	--	Feb. 21	12	342	218	342	-36	Unknown
484	Feb. 21	27	Apr. 17	55	385	444	444	+15	Killed; mild scurvy

Weekly Intake of Basal Ration for Individual Animals in Grams.

	Group 1.			Group 11.				Group 12.				
Week	442	443	450	441	447	454	445	449	453	455	457	458
1	37	33	36	37	33	50	16	30	40	24	42	26
2			31	30	31	71		29	32	70	63	41
3	27	42	27	24	39	50		22	20	51	67	16
4	44	56	35	2	25	0		27	3	36	55	
5	36	13	63	13	7	5		11	0	26	64	
6	33		61	3	4	4		23		19	60	
7	54		71	2 da.				27		33	33	
8	52		50					33		33	69	
9	43		44					21		30	60	
10	42		69					20		21	71	
11	28		63					20		37	67	
12			52					13		42	62	
13			31					13		17	43	
14								16		30		
15										30		
16										46		
										12 (2 da.)		

	Group 13				Group 14					Group 15			
	451	452	456	433	459	460	461	462	463	464	465	466	473
1	36	50	38	43	44	29	49	37	51	37	39	27	56
2	37	45	49	69	59	52	49	35	54	64	69	45	19
3	18	21	44	19	60	51	70	54	59	34	56	30	3
4	5	6	26	7	54	62	74	74	50	66		17	
5	20	22	8	2 da.	71	40	44	74	34	104			
6	30	40	40		67	30	40	78	59	126			
7	20	40	53		73	26	24	113	41	108			
8	28	29	50		33	14	8	95	58	45			
9		39	37		66	8		74	33	33			
10		27	27		70	21		40	31	33			
11		40	31		55	22		37	46				
12		31			62	4	2 da.	50	22	3 da.			
13		30			27	3da.		5 da.					
14		41											
15		25											

	Group 16.							Group 17.				Group 18.			
Week	463	469	470	471	472	482	485	474	475	476	477	478	479	480	484
1	70	67	93	41	56	50	62	29	75	40	63	36	72	29	44
2	29	23	41	43	41	75	9	38		55	20	29	45	26	70
3	4	25	31	30		63	12	36		15		39	23	0	55
4	1 da.	7	34	46		32	6	27		8		39	23		47
5		1 da.	34	39		62	4 da.	27		15		37	24		64
6			51	20		47		27		19		42	25		63
7			49	43				27		21			15		
8			34	50						44			31		
9			42	55											

Note

Records of intake of basal ration for groups 2, 3 and 4 on 30 cc. level with pasteurized milk only partially complete, and consequently are not presented here. The same holds true for groups where the curative method was attempted, Groups 7, 8 and 9.

Explanation of Charts One to Five.

Charts one to five show graphically the results of the feeding experiments by groups. Each square represents two weeks on the abscissa and 50 grams on the ordinate. The following symbols were used in the charts :-

S = Scurvy

S? = doubtful scurvy

NS = no scurvy

MS = mild scurvy

SS = severe scurvy

SC = scurvy plus complications

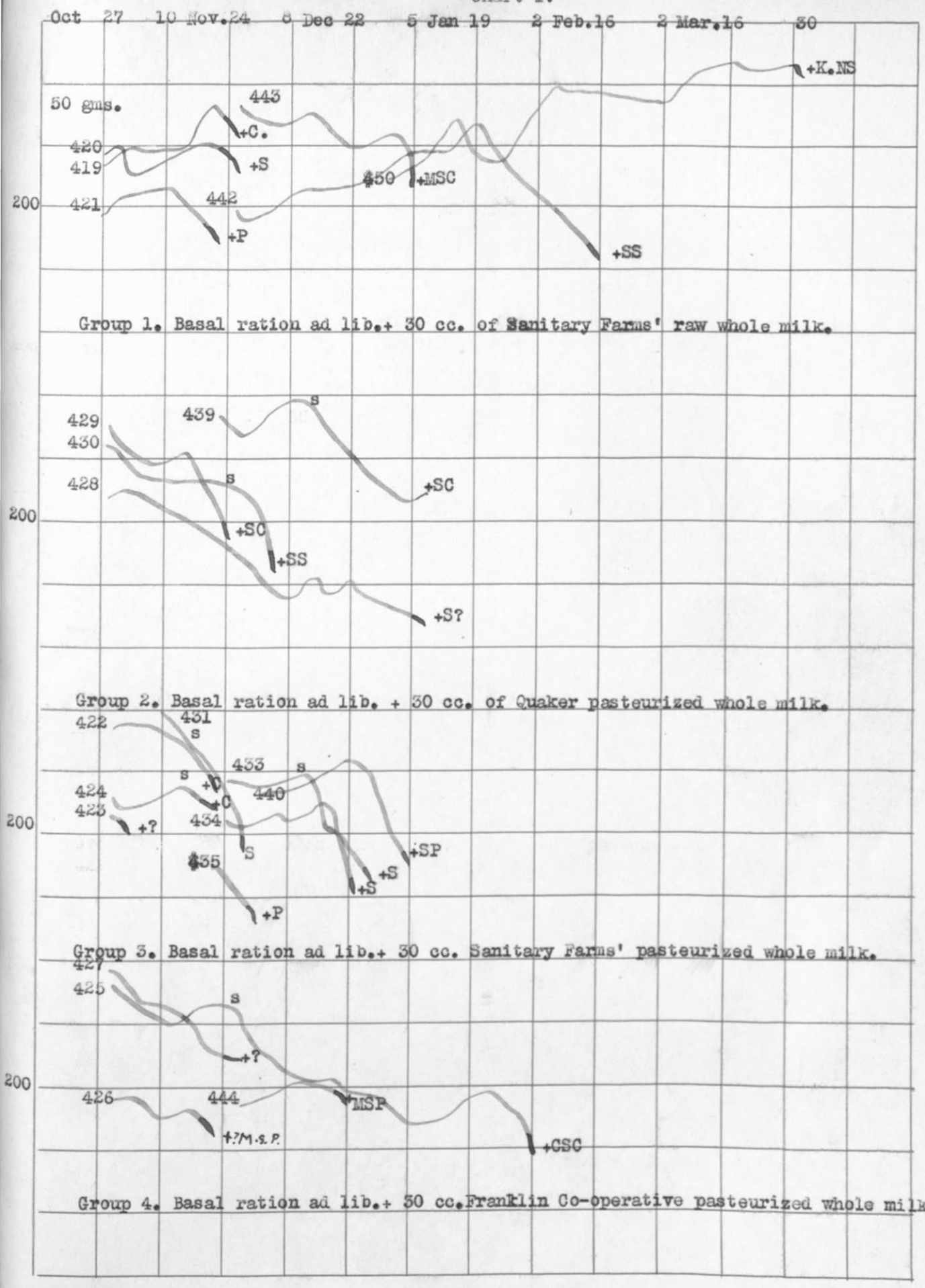
C = constipation

? = unknown

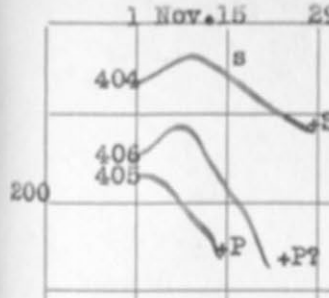
K = killed

P = pneumonia

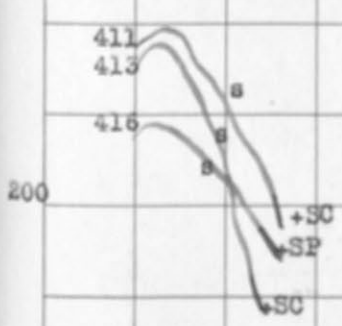
Chart 1.


Oct 27
10 Nov. 24
8 Dec 22
5 Jan 19
2 Feb. 16
2 Mar. 16
30
50 gms.
200
443
+C.
420
419
+S
421
442
+P
450
+MSC
+SS
+K.NS
Group 1. Basal ration ad lib.+ 30 cc. of Sanitary Farms' raw whole milk.
429
430
428
439
s
+SC
+SC
+SS
+S?
Group 2. Basal ration ad lib. + 30 cc. of Quaker pasteurized whole milk.
422
431
433
440
424
423
+?
434
435
+P
+SP
+S
Group 3. Basal ration ad lib.+ 30 cc. Sanitary Farms' pasteurized whole milk.
427
425
+?
426
444
+?M.S.P.
MSP
+CSC
Group 4. Basal ration ad lib.+ 30 cc. Franklin Co-operative pasteurized whole milk.

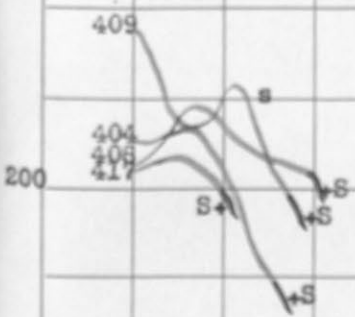
1 Nov. 15 29 Chart 2.



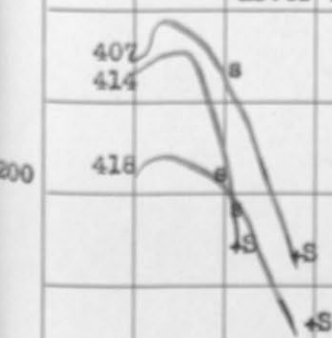
Group 5. basal ration and water ad lib. (Unautoclaved)



Group 7. Basal ration ad lib.+ 30 cc. Sanitary Farms' raw whole milk, after scurvydeveloped.



Group 8. Basal ration ad lib. + 30 cc. Quaker pasteurized whole milk, after scurvy developed.



Group 9. Basal ration ad lib.+ 30 cc. Franklin Co-operative pasteurized whole milk, after scurvy developed.

Chart 3.

New basal ration.

Casein.....	40 %
Ground rolled oats.....	40 %
Autoclaved alfalfa, ground....	14 %
Dried spinach, ground.....	5 %
Sodium chloride.....	1 %

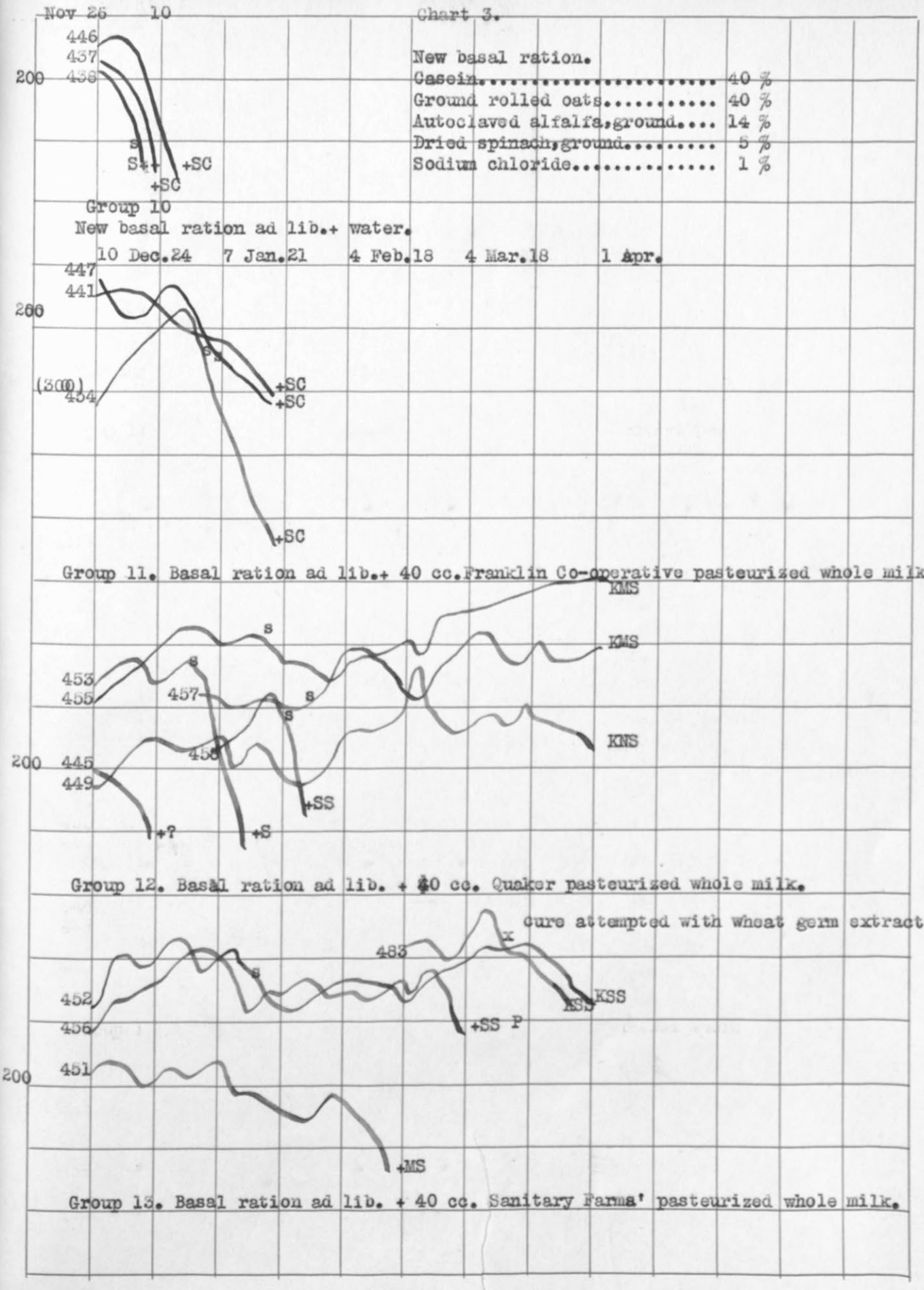
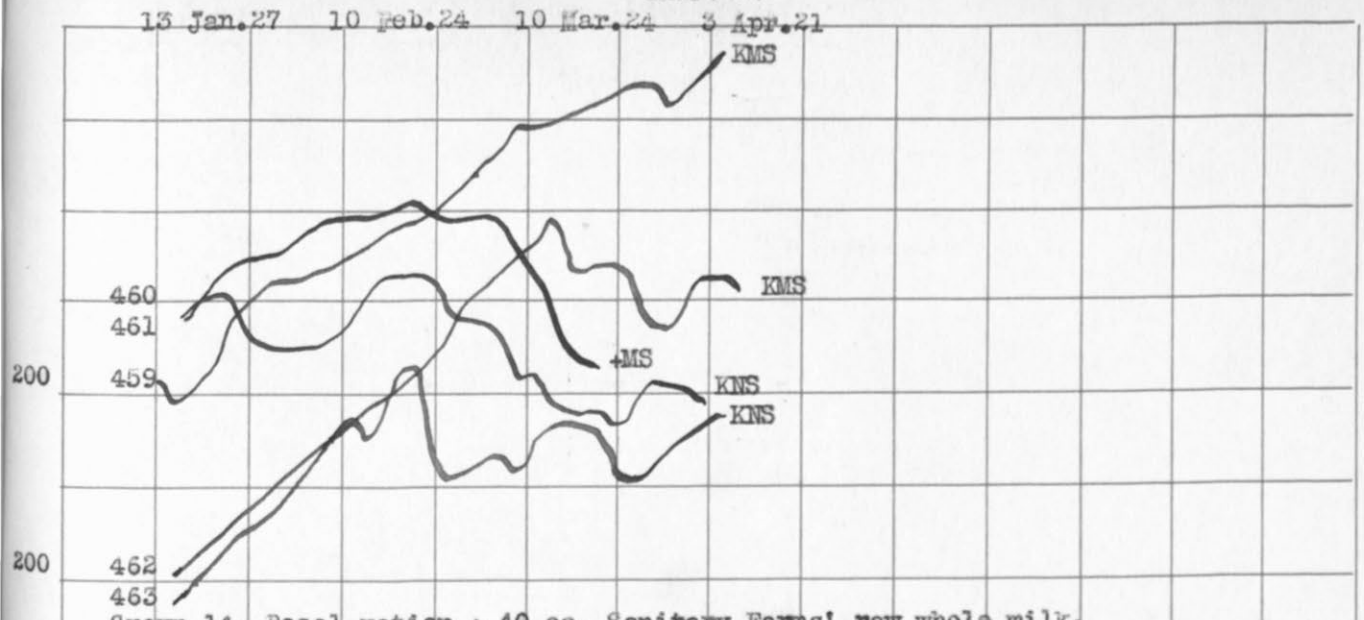
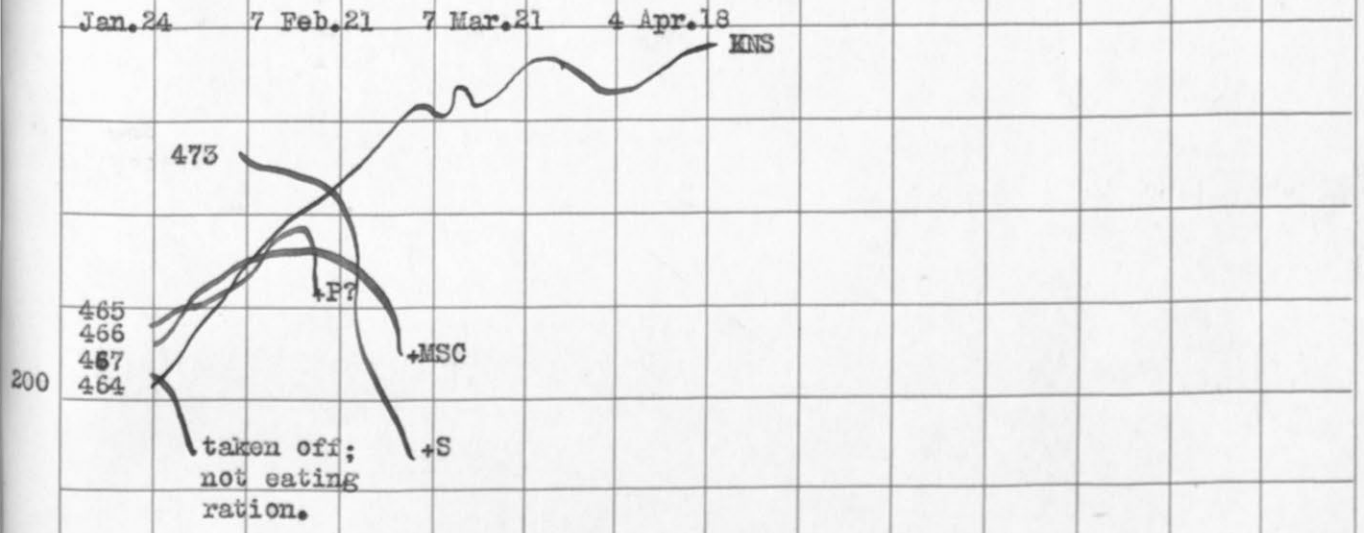


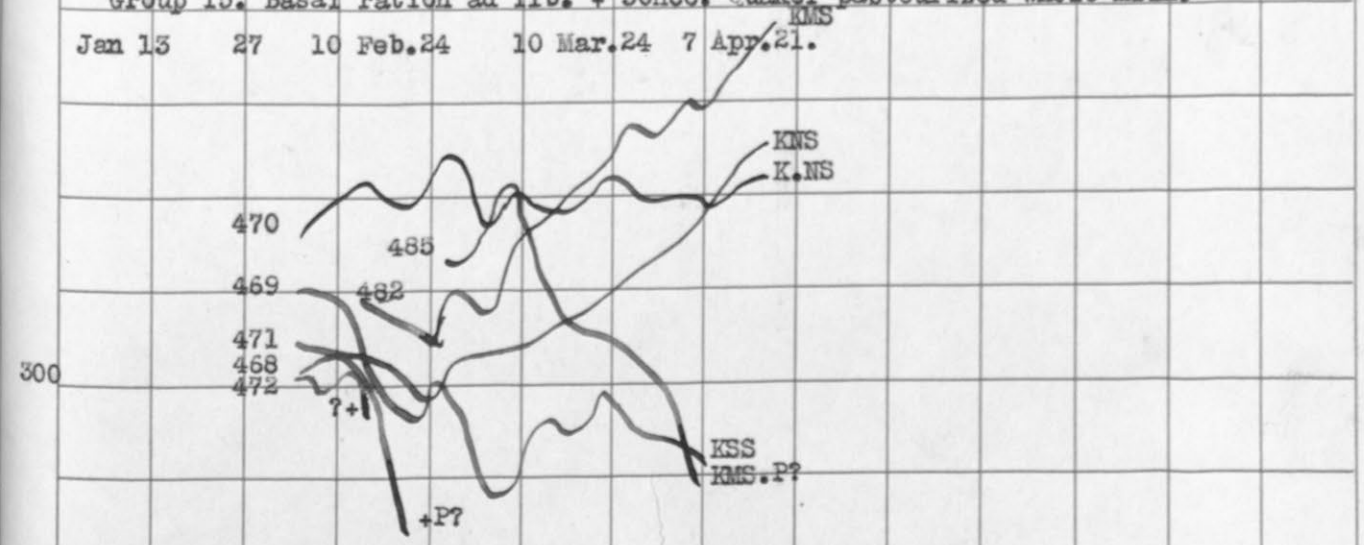
Chart 4.



Group 14. Basal ration + 40 cc. Sanitary Farms' raw whole milk.

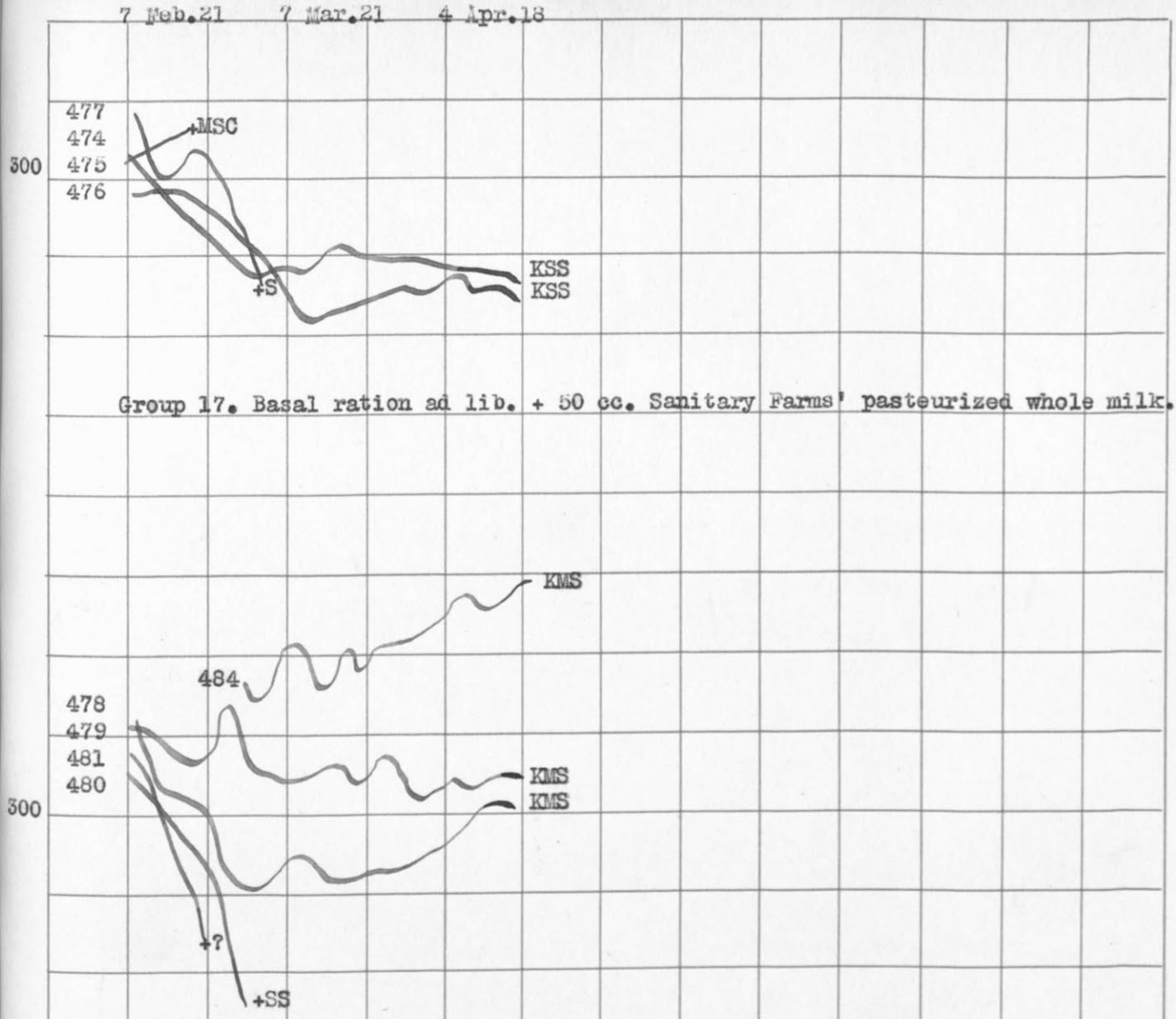


Group 15. Basal ration ad lib. + 50ncc. Quaker pasteurized whole milk.



Group 16. Basal ration ad lib. + 50 cc. Sanitary Farms' raw whole milk.

Chart 5.



Group 17. Basal ration ad lib. + 50 cc. Sanitary Farms' pasteurized whole milk.

Group 18. Basal ration ad lib.+ 50 cc. Franklin Co-operative pasteurized whole milk.

Explanation of Charts Six and Seven

The following charts represent the milk intake of the animals in the various groups which failed to completely consume their daily ration of milk, and include the following groups:- 3, 11, 12, 13, 15, and 16. The following groups had practically complete consumption of the milk offered daily, or else refused such small amounts as to have no effect on the experiment :- 1, 2, 4, 7, 8, 9, 14, 17, 18.

In these charts the abscissa represents time, each square being two weeks, The milk consumed is plotted on the ordinate against time, each square being 50 cc.

Chart 6.

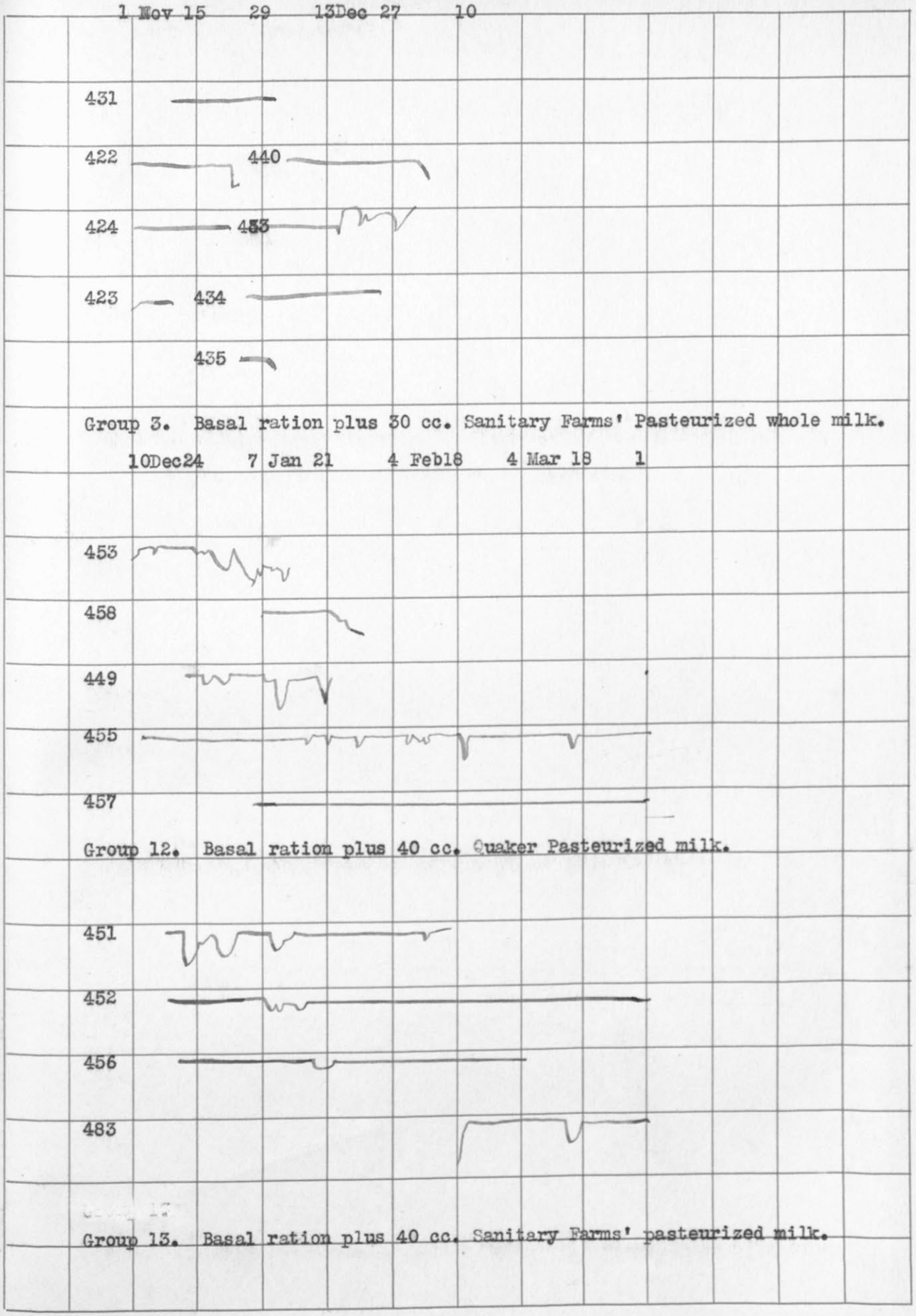
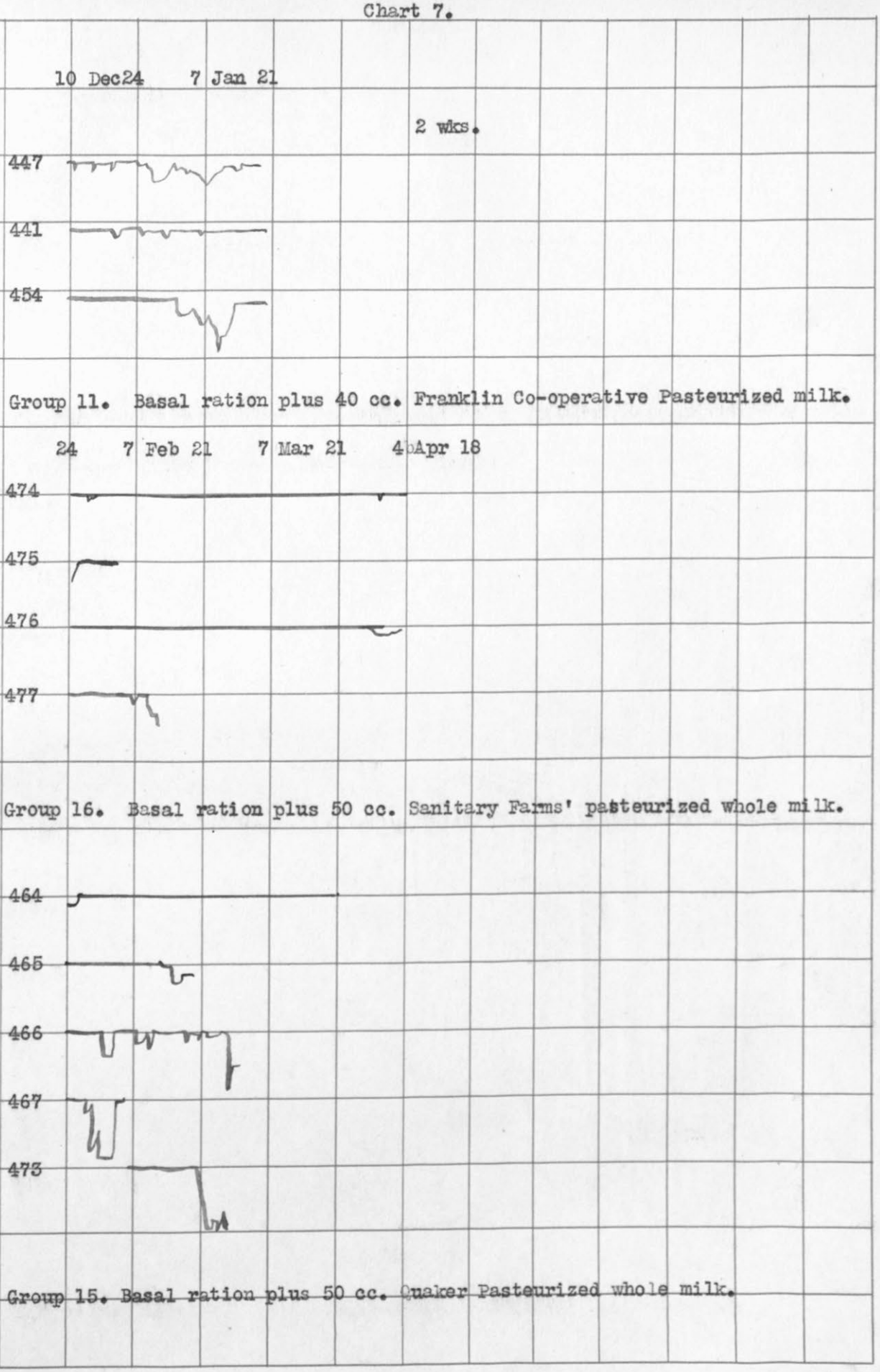

1 Nov 15 29 13Dec 27 10
431
422
440
424
453
423
434
435
Group 3. Basal ration plus 30 cc. Sanitary Farms' Pasteurized whole milk.
10Dec24 7 Jan 21 4 Feb18 4 Mar 18 1
453
458
449
455
457
Group 12. Basal ration plus 40 cc. Quaker Pasteurized milk.
451
452
456
483
Group 13. Basal ration plus 40 cc. Sanitary Farms' pasteurized milk.

Chart 7.


10 Dec24
7 Jan 21
2 wks.
447
441
454
Group 11. Basal ration plus 40 cc. Franklin Co-operative Pasteurized milk.
24
7 Feb 21
7 Mar 21
4 Apr 18
474
475
476
477
Group 16. Basal ration plus 50 cc. Sanitary Farms' pasteurized whole milk.
464
465
466
467
473
Group 15. Basal ration plus 50 cc. Quaker Pasteurized whole milk.

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