Infant nutrition : a textbook of infant feeding for students and practitioners of medicine / by Williams McKim Marriott.

Errata

Running heads, pages 233, 235, 237 and 239, should read "Vomiting in Infancy," instead of "Vomiting in Pregnancy."

On page 268, read "Buffered Lactic Acid Solution," instead of "Bacteria Acid Solution."

INFANT NUTRITION

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A TEXTBOOK OF INFANT FEEDING FOR STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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TO THE MEMORY
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PREFACE

There is perhaps no field in medical practice in which more difference of opinion has existed than in that of infant feeding. So numerous have been the theories advanced and so diverse the methods of feeding recommended that the practitioner is likely to become hopelessly bewildered. Much of this confusion, however, is avoidable, for the fundamental facts concerning the nutritional requirements of infants are now fairly well established, and the most that any type of feeding can accomplish is to fulfill these requirements.

It is the purpose of this book to summarize present-day knowledge concerning the nutritional requirements of infants under normal and pathological conditions and to indicate the effects of failure to meet any or all of these requirements.

In order that the results of feeding on the growth and development of infants may properly be appraised, a knowledge of normal growth and development is essential—a chapter on this subject has accordingly been included.

A food, although a complete one, cannot be expected to meet the nutritional requirements unless it is one which is capable of utilization by the infant; in other words, it must be digestible and absorbable and not capable of causing injury to the body. The selection of such a food must be based on an understanding of the characteristics of the chief food elements and the processes of digestion and absorption in infancy as well as alterations of these processes which are likely to occur under abnormal conditions. The chapters on metabolism and digestion outline the present-day viewpoint on these subjects.

With a thorough knowledge of the nutritional needs of the infant and of the means by which it is possible to meet those needs, the practitioner is in a position to appraise the value of any given type of feeding. If a formula or type of food is one which meets fully the requirements and at the same time is one which is capable of utilization without harm to the body, it will be successful. If it fails to meet any one of the essential requirements, it will fail as a whole. If these self-evident facts were more fuliy appreciated,

there would be fewer failures due to the feeding of well balanced digestible formulas in insufficient amounts to meet the minimal energy requirements or the feeding of mixtures meeting the caloric demands but deficient in such essential elements as protein or some of the vitamines.

There are numerous ways in which infants may be fed successfully, and there is no one method of feeding or type of food which is to be recommended to the exclusion of others; on the other hand, there is no reason to resort to the use of complicated formulas or expensive proprietary preparations when the particular requirements in the individual case may be fully met by simpler means. The simpler means are especially stressed in this book, as any complication of methods is not only unnecessary but introduces greater chances for error. Sufficient information is given, however, it is hoped, to enable the practitioner to use intelligently such special foods as are now available or may from time to time be introduced.

The chief gastrointestinal disturbances of infants—vomiting, diarrhea and constipation—receive especially full consideration, for the occurrence of these conditions may wreck any carefully laid plan of feeding.

So important is the relationship of infection to nutrition that a separate chapter is devoted to the subject. It is becoming recognized that many of the disturbances of infants which have previously been attributed to dietary faults are in reality the result of infections. The relationship, however, is a reciprocal one, for infections often occur because the nutrition has been inadequate. An infant suffering from an unrecognized otitis media cannot be made to thrive by merely changing the feeding formula, nor on the other hand can an infant with a chronic infection be expected to overcome the infection unless the diet is an adequate one.

Throughout the book an effort has been made to stress the importance of the chemical pathology of the body as related to feeding and nutrition, as an understanding of this enables one to apply much more intelligently therapeutic procedures. This phase of the subject receives especial consideration in the chapters on Anhydremia, Acidosis and Alkalosis, Rickets, and Tetany.

The author believes a thorough exposition of the fundamental principles of nutrition to be of greater value to the practitioner than a mere outline of methods of procedure, for, with a concep-

tion of the underlying principles, he can intelligently meet any condition as it arises and be independent of "rule-of-the-thumb" procedures. Nevertheless the practical application of the scientific principles laid down can perhaps best be appreciated by the use of specific examples. For that reason numerous clinical protocols have been introduced illustrating the more common nutritional problems; details of the management of cases described are given. An attempt has thus been made to give the *how* as well as the *why*.

As a further aid from the practical standpoint, separate chapters on therapeutic procedures and on medication in infancy have been included.

The author makes no claim of originality for the ideas expressed in this book. The successful methods of modern infant feeding are the outcome of the labors of many men in many countries, each contributing some useful or important observation. The last word has not yet been spoken. The establishment of a new fact may, at any time, change accepted methods of practice. An effort has been made in this book to evaluate present-day knowledge of nutrition on the basis of actual clinical trial, and conclusions have been arrived at not in the laboratory or easy chair, but in the clinic.

The author takes pleasure in expressing his indebtedness to the various members of the Department of Pediatrics of the Washington University School of Medicine who have contributed many helpful suggestions, and especially to Doctors Theodore C. Hempelmann and Alexis F. Hartmann, who have read the entire manuscript and have made many valuable suggestions.

W. McK. M.

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CHAPTER I

GROWTH AND DEVELOPMENT

Adequate nutrition of the infant must provide not only for the relatively high energy exchange, but also for normal growth and development. The best test of the adequacy of the nutrition is the character and the rate of growth. Even among normal and apparently healthy infants, there is considerable variation in growth and development, which is the result of both hereditary and nutritional factors. Infants belonging to certain races are larger than others and in some families there is a tendency for the children to be large. The maximum size which an infant may attain appears to be determined by heredity. How closely the infant may approach this maximum is determined by the character of the food and by the environment. Without question many infants fail to attain the maximum because of improper nutrition or poor hygiene. Average figures for the weights and measurements of infants at various ages should not be taken as an absolute guide in determining the development of an individual infant. Such average figures have been compiled from statistics gathered from infants of all types, both well nourished and poorly nourished and should be considered only for what they are, that is, *averages* and not *normals*. Babies of healthy stock given abundant food of suitable composition are generally distinctly heavier and larger in all measurements than the accepted averages.

Weight

The weight of infants born at full term averages 7¼ pounds (3350 gm.) boys being usually somewhat larger than girls. When adequately nourished, such average weight infants may be expected to double the birth weight by the end of five months and to triple it by the end of a year. Infants who are heavier than the average at birth remain heavier than the average throughout the first year, but the proportionate rate of gain is not so great. Very large infants may not double the birth weight before six months of age,

and may not triple it before fourteen months. Very small infants, on the other hand, gain at a somewhat more rapid rate, those weighing five or six pounds doubling the weight during the fourth month and tripling it by ten months. Infants weighing less than 4½ pounds at birth usually double the weight by three months and triple it by six months. The average weights of healthy, breast-fed infants at various ages as compiled from the figures of Camerer appear in Table I.

TABLE I

AVERAGE WEIGHTS AND BODY LENGTHS OF FULL-TERM AND PREMATURE INFANTS (AFTER CAMERER)

	FULL-TERM INFANTS				PREMATURE INFANTS			
	BIRTH WEIGHT OVER 6.5 LB.				BIRTH WEIGHT 4.5 TO 6.5 LB.		BIRTH WEIGHT LESS THAN 4.5 LB.	
	WEIGHT		BODY LENGTH		WEIGHT		WEIGHT	
	POUNDS	GRAMS	INCHES	CM.	POUNDS	GRAMS	POUNDS	GRAMS
Birth	7.6	3433	19.3	49	5.4	2440	3.7	1700
1 week	7.5	3408	19.7	50	5.5	2500	3.8	1720
2 weeks	7.9	3567	20.0	51	5.7	2570	4.1	1850
4 weeks	8.8	3995	20.5	52	6.4	2890	4.8	2180
8 weeks	10.6	4818	22.0	56	8.1	3660	6.4	2910
12 weeks	12.2	5546	22.8	58	9.5	4320	7.8	3560
16 weeks	13.7	6225	23.6	60	11.0	5000	9.2	4160
20 weeks	15.0	6788	24.4	62	12.2	5550	10.5	4750
24 weeks	16.1	7320	25.2	64	13.1	5940	11.7	5300
28 weeks	17.1	7767	25.6	65	13.8	6270	12.5	5700
32 weeks	18.0	8147	26.0	66	14.6	6650	13.1	5940
36 weeks	18.9	8585	26.4	67	15.2	6910	13.3	6040
40 weeks	19.5	8859	27.2	69	15.7	7130	14.0	6360
44 weeks	20.3	9209	28.0	71	16.2	7370	14.2	6430
48 weeks	21.0	9526	28.7	73	17.1	7760	14.3	6500
1 year	21.7	9862	29.5	75	18.1	8220	14.5	6570
18 months	25.5	11590	31.5	80	21.1	9590		
2 years	28.0	12740	33.5	85				

These weights are plotted graphically in Fig. 1.

It has generally been stated that the weights of artificially fed infants throughout the first year average less than those of breast-fed infants. This statement is true if one considers the entire group of artificially fed infants, a large proportion of whom are undoubtedly underfed. The statement, however, does not apply to infants who are *properly* fed artificially. The infant given an adequate artificial feeding should weigh no less than the infant entirely breast fed.

It will be noted from the weight curves that the rate of weight increase is not the same throughout the first year, but is greater during the first few months than during the later months of the

year. This difference in the rate of growth during the first year is especially marked in the case of smaller infants. The weight curve of an individual infant should not be expected to parallel exactly the average curves given throughout the first year. Even

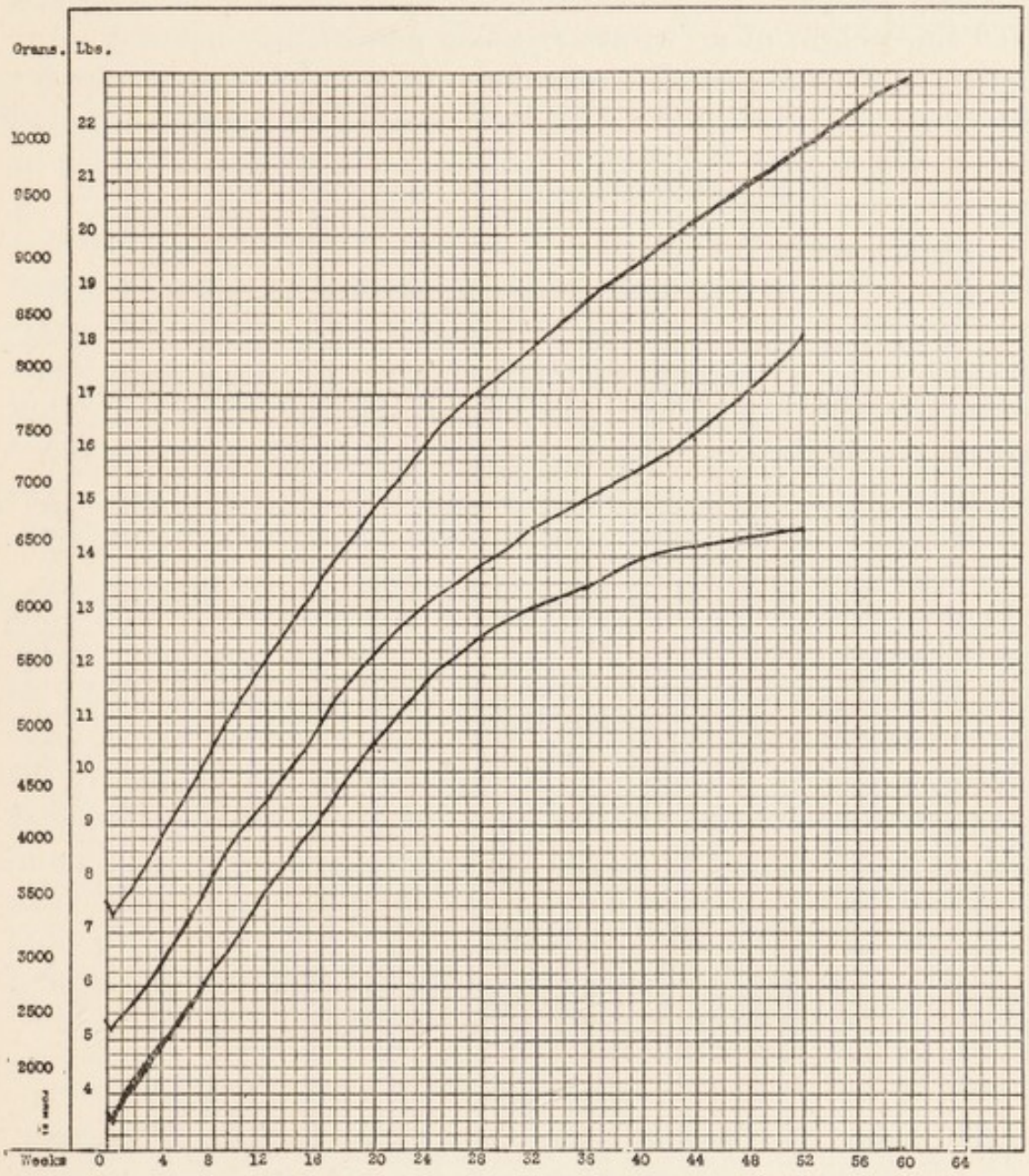


Fig. 1.—Average weight curves of infants during the first year of life. (After Camerer.)

normal and apparently healthy infants show periodical variations in the rate of growth. The weight curves given take no account of constitutional or hereditary factors. Two full-term infants, weighing the same at birth, may differ largely in constitution; one may be of the long, slim, "linear" type and the other of the short,

stocky, "lateral" type. One may be the offspring of a family, the majority of the members of which are of small stature and slim; the other of a family of large proportions. The two infants will not gain in length and in weight at the same rate, even though both may be given the same adequate diet. In general, however, when the weight of an infant remains persistently below the expected, as shown on the curve of average weights, or when the rate of weight increase is regularly below the average, one should suspect either that the diet is inadequate in some particular or that the infant is suffering from some disease.

A weight above the average may be considered an indication that the infant is constitutionally of a large type and has been adequately fed. Many normal infants exceed in weight the average figures by as much as 15 or 20 per cent. This should be no cause for alarm and should certainly not be taken as an indication to decrease the feeding, provided the food has been a well-balanced one, not containing an excessive proportion of carbohydrate and provided the infant's growth in length and in muscular development has been satisfactory.

In each of the weight curves it will be noted that a loss of weight occurs during the first few days of life. This loss of weight averages 5 to 10 per cent of the initial weight, and is due chiefly to passage of urine and meconium, rather than to actual loss of body tissues or fluids. In the case of infants who are nursed at the breast, and who receive no additional food or fluid, the intake of food may be insufficient to meet the needs of the body for a number of days, and this is an added factor in causing the weight loss. If, on the other hand, infants are given considerable food and water from the time of birth, the loss in weight is but slight, and in some instances no loss of weight at all occurs.

Body Length

The total length of the body is dependent more upon hereditary and constitutional factors than upon the nutrition. An infant who is fed on a diet deficient in energy value may continue to grow in length at almost a normal rate, although the weight lags far behind. Ultimately, if the insufficient diet is continued, the rate of increase in the length of the body also slows down. When an adequate diet is subsequently given, increase in weight and circumference of the body occurs and may even exceed the growth

in length so that finally the infant's weight becomes proportionately greater than his length. The body length is, of course, profoundly influenced as the result of such constitutional anomalies as achondroplasia, of endocrine disturbances, and of rickets. The average relationship between weight and body length in normal infants is given in Table I (Camerer). In estimating the adequacy of the nutrition, it is more important to determine whether the weight corresponds to the body length than to the age.

Skin and Subcutaneous Tissues

The normal, well-nourished infant has a moist elastic skin, which is pink at birth and which gradually becomes somewhat paler throughout the first year. An icteroid tint is commonly observed during the first week or ten days of life (icterus neonatorum), but after this time any yellowness of the skin is abnormal. Pallor of the skin is seen in the presence of anemia, when the diet has been deficient in protein, green vegetables or iron, and especially in the case of very fat infants fed on diets containing excessive proportions of carbohydrate. A gray color of the skin is not seen in normal infants. It is seen in conditions of dehydration and extreme undernutrition where the blood volume has been decreased and the circulation impaired. One of the first evidences of poor nutrition is a loss in the degree of turgor of the skin. The skin and subcutaneous tissues become loose, flabby, and inelastic.

The normal infant has, throughout the first year, a relatively thick layer of subcutaneous fat. This can best be appreciated by pinching up a fold of the skin of the abdomen. Under normal conditions, such a fold will have a thickness of one-half to one inch, whereas in cases of undernutrition, the thickness will be much less, the combined skin and subcutaneous tissues being at times no thicker than blotting paper. The thickness of this adipose layer, even in the absence of information as to age, weight or height, serves as a good indication of the state of nutrition.

Head

The head grows more rapidly during the first year than at any other time, the increase in circumference being as much during the first year as during the remainder of life. This growth of the head continues, even in conditions of extreme underfeeding, so

that the head may appear disproportionately large as compared with the remainder of the body. The circumference of the head at birth in the case of full-term infants averages 13.5 to 14 inches (34.3 to 35.5 cm.). By six months the circumference averages 17 inches (43.2 cm.), at one year 18 inches (45.7 cm.), and at two years 18.75 inches (47.5 cm.). The heads of premature infants are smaller than those of full-term infants at the time of birth, but are large in proportion to the remainder of the body. By one year of age, however, the circumference of the heads of prematurely born infants is approximately normal.

The posterior fontanelle is usually closed before the end of the second month. The anterior fontanelle increases slightly in size during the first six or nine months of life and then slowly becomes smaller as ossification of the surrounding bones proceeds. This fontanelle normally closes between the ages of fifteen and twenty months. There is, however, variation in well-fed and apparently normal infants. Nutritional disturbances and especially rickets often result in delayed closure of the anterior fontanelle.

Chest

The circumference of the chest at birth is slightly less than that of the head, the difference usually being no greater than one-half inch (1.25 cm.). A greater disproportion is seen in the case of premature infants. By the age of one year the head and chest normally have the same circumference and thereafter the chest circumference increases somewhat more rapidly than that of the head, exceeding the latter by about one inch (2.5 cm.) at the age of five years. In the case of undernourished infants the circumference of the chest fails to increase at the same rate as that of the head. The smaller circumference of the chest of undernourished infants is due not only to a thinner layer of adipose tissue, but also to a smaller bony framework.

Teeth and Jaws

The development of the teeth begins early in intrauterine life with deposition of lime salts in the crowns of the temporary teeth as early as the sixteenth or seventeenth week of fetal life. At the time of birth, all of the temporary teeth are calcified and the permanent first molars are partially calcified.

There is considerable variation among normal infants in the time of eruption of the temporary teeth, although the order of eruption is quite uniform. The lower central incisors usually erupt between the fourth and eighth months, followed within a few weeks by the upper central incisors. After a period of quiescence, the eruption of the anterior molars and remaining incisors occurs. These appear between the tenth and sixteenth months. The four canines (eye and stomach teeth) appear between the eighteenth and twenty-fourth months, and the posterior molars between the twenty-fourth and thirtieth months. The teeth erupt in groups at fairly long intervals, rather than continuously.

Eruption of the temporary teeth is retarded as the result of rickets. It is also retarded in hypothyroidism. An additional cause for delayed eruption of the temporary teeth is deficiency in the diet of the mother during the period of pregnancy. Under such conditions the teeth may not only be erupted late but may be definitely hypoplastic and subject to enamel defects. Since the development of the jaws is dependent upon the growth of the teeth, the jaws of undernourished infants and those who are rachitic are likely to be poorly formed, narrow and pointed.

Stomach

The size of the stomach is subject to wide variations even in the case of infants of the same weight and age. Measurements of the capacity of the stomach made postmortem do not indicate the physiologic capacity, which is always considerably greater. An infant is capable of taking much more fluid food than the anatomical capacity of the stomach. This is due to the fact that food leaves the stomach while being taken. Furthermore, the stomach is subject to considerable distention.

The average amounts of food taken by normal breast-fed infants are indicated in Table II.

TABLE II

AMOUNTS OF MILK TAKEN AT A FEEDING BY AVERAGE-SIZED NORMAL INFANTS

AGE	QUANTITY		AGE	QUANTITY	
	OZ.	C.C.		OZ.	C.C.
1 week	2.5	75	4 months	6	180
2 weeks	3.0	90	6 months	7	210
1 month	4.0	120	8 months	8	240
2 months	4.5	135	1 year	8	240

The volume of food taken at a feeding by artificially fed infants depends upon the feeding intervals and the degree of dilution of the formula. Infants fed on dilute formulas, having low caloric values, must of necessity take larger amounts if the nutrition is to be maintained. Infants fed on these dilute mixtures are usually hungry and take large volumes at a feeding. Furthermore, the more dilute feedings leave the stomach somewhat more rapidly while being taken. On the other hand, with more concentrated feedings the infant's appetite is more completely satisfied and smaller volumes of food are taken. Infants who are habitually fed large volumes develop enlarged and distended stomachs. Infants who have been underfed for a period often show a remarkable capacity. Thus we have observed infants during the second month of life who have taken as much as ten ounces (300 c.c.) at a single feeding, and we have seen infants of four months take and retain fifteen ounces (450 c.c.) of milk at a time. In the case of infants properly fed there is no need for the giving of such enormous volumes at a feeding.

The position of the stomach during early infancy is almost transverse. As the infant grows older and assumes an upright position for a portion of the day, the pyloric end of the stomach moves downward and in time the stomach as a whole assumes a more vertical position.

The Intestinal Tract

The length of the intestinal tract at birth averages 13 feet (4 meters) and increases in length by about one-third during the first year of life (Scammon). The large intestine at birth averages 26 inches (66 cm.) in length and increases to 35 inches (89 cm.) by the end of the first year. The ascending and descending portions of the colon in the young infant are short as compared with the transverse colon. The sigmoid at birth is greatly distended with meconium and throughout infancy extends higher into the abdomen than during later life. In infants who are habitually constipated, the distention of the sigmoid persists and later the remainder of the colon also becomes distended. In the case of infants who are underfed and especially in those cases in which the diet is deficient in protein and "B" and "D" vitamins, the intestinal musculature becomes atrophic and atonic. The entire gastrointestinal tract is distended and incapable of normal peristaltic and peristolic movement.

The Blood

The total volume of the blood, as well as the red cell and hemoglobin content, is higher at the time of birth than at any other period during life. Considerable destruction of the excess blood occurs during the first week or two of life, the conversion of blood pigment into bile pigment being in part at least responsible for the icterus of the newly born infant.

The volume of the blood at birth is stated by Lucas and Dearing to average 14.7 per cent of the body weight. After the neonatal period this volume falls to 9 or 10 per cent of the body weight. In the case of infants who have been underfed or who have become dehydrated, the absolute volume of the blood is decreased, but not necessarily to the same extent as the decrease in weight. A decrease in the blood volume is regularly associated with a marked impairment of the circulation. We have observed malnourished and dehydrated infants in whom the volume flow of the blood in the extremities was less than one-tenth of the normal. The volume flow in the internal organs, although not susceptible to accurate measurement, appears also to be decreased, and results in a diminished degree of absorption from the gastrointestinal tract as well as functional incapacity of the heart and of some of the secreting organs.

The hemoglobin content of the blood at birth varies from 100 to 120 per cent (based on a scale of 100 per cent for a normal adult). By the third week the hemoglobin has usually fallen to about 90 per cent. A slow decrease then occurs for the next four or five months, so that by the end of the fifth or sixth month the hemoglobin has fallen to 75 or 80 per cent. From this time on the hemoglobin content of the blood is largely dependent upon the character of the feeding. If an infant receives only milk, whether it be human or cow's milk, a progressive decrease in hemoglobin occurs. If, however, the diet contains green vegetables, meat and eggs, any further marked reduction in the amount of hemoglobin does not usually occur. The fluctuations in hemoglobin are to be explained by the fact that as the infant grows the blood volume increases proportionately, the total volume of new blood formed during the first year of life amounting to from 20 to 25 ounces (600 to 750 c.c.). If a normal hemoglobin content of this newly

generated blood is to be maintained, considerable amounts of hemoglobin must be manufactured in the body, and for this purpose iron, as well as pigment, and certain protein constituents are required. Relatively little iron is contained in either human milk or cow's milk and when either of these forms the sole diet of the infant, insufficient iron is supplied to provide for the formation of the required amount of hemoglobin. A fair amount of available iron, however, is present in the liver of the normal full-term infant, and this supply of iron may be drawn upon for the manufacture of hemoglobin. This reserve supply of iron is usually pretty well exhausted by the end of the first four or five months, and unless iron is supplied in the diet, in addition to the small quantities present in milk, hemoglobin production does not keep pace with the increase in blood volume. Green vegetables, eggs and meat supply not only iron but also certain organic radicles which are constituents of the hemoglobin molecule. Prematurely born infants have very small iron reserves, since mobilization of iron in the fetal liver does not occur until the later months of pregnancy. Premature infants therefore tend to become especially anemic during the early months of life and may require the feeding of additional iron or even transfusions.

The red blood corpuscles in the full-term newly born infant usually number well over 5,000,000 per c.mm. and are occasionally as high as 7,000,000. Nucleated red blood cells may be present during the first week of life, but they are not found under normal conditions after this time. The number of red blood corpuscles diminishes rapidly during the first week or two to 4,500,000 or 5,000,000 per c.mm. The count remains about this figure throughout the first year of life, even though the hemoglobin may be considerably reduced, the color index thus becoming low. One frequently observes pale, poorly nourished infants, who have normal red blood cells counts. In the condition of anhydremia the red blood cell count is usually distinctly above the normal, although the infants are actually suffering from greatly decreased total blood volumes. It is essential that one should distinguish between anemia and oligemia. An infant with a normal red cell count or a normal hemoglobin may still have a greatly decreased blood volume and be in need of transfusion.

The white blood corpuscles number from 15,000 to 20,000 per

c.mm. at the time of birth, but drop to between 11,000 and 14,000 by the end of the second week. The average white blood cell count remains in the neighborhood of 12,000 during the next two years. The percentage of polymorphonuclear cells during the first few days of life varies from 70 to 75 per cent of the total white cell count. By the end of the second week this percentage falls to 30 or 40 and at the same time the percentage of lymphocytes increases to 50 or 65 per cent. It is not until the fourth or fifth year of life that the percentages of polymorphonuclear neutrophiles and mononuclear cells become equal. Thereafter there is a gradual increase in the polymorphonuclear elements until the adult proportion of 60 to 75 per cent is attained about the twelfth year. The percentages of eosinophiles and basophiles in the blood of the infant do not differ materially from those in adult blood. In the case of malnourished infants there is usually a moderate increase in the total white blood count.

The blood may from the time of birth show distinct differentiation into the usual blood types and groups. It is, therefore, as necessary to match the blood for transfusion of the infant as in the case of older individuals. Strange to say, the mother's blood is not always compatible with that of the infant.

Mental and Muscular Development

During the third month of life the normal infant is usually able to hold the head erect when the body is supported. About the same time he begins to grasp objects, but coordination is very defective. By the age of six months he can reach for objects and has an appreciation of distance. By the seventh month he can sit alone, but not very steadily until the eighth or ninth month. By eight or nine months the normal infant usually is able to creep a short distance. Walking with support is accomplished about the end of the first year. The infant recognizes his mother some time during the third month, begins to understand certain words by the end of the ninth or tenth month, and by the end of the first year can say a few words. By two years short sentences may be spoken. There is very considerable difference, even in normal infants, as to the time of talking.

Defects in nutrition lead to delayed muscular development and consequent delay in sitting alone, crawling, and walking. In addi-

tion, severe nutritional disturbance not infrequently leads to delay in mental development. Some children three or four years old, suffering from undernutrition or rickets, are unable to talk, walk, or sit alone. The retardation may be such that actual mental insufficiency is suspected, but with the administration of an adequate diet, the mental development of such children may become entirely normal.

CHAPTER II

ENERGY METABOLISM

The metabolic processes of the infant are of essentially the same character as those of the adult, but are relatively much more active. A large intake of food per unit of body weight is necessary in the case of infants, not only to supply material for growth, but also to allow for a very active energy exchange.

The living body, like a machine, requires fuel and materials for repairs and replacements. In the growing individual there is the additional requirement for growth. These needs are met by food, which must have a sufficient fuel value, that is, must supply enough calories. It must supply certain minimum amounts of protein, carbohydrate, mineral salts, water and vitamins, and for practical purposes should supply a certain amount of fat, although this latter is not an absolutely indispensable constituent.

The Significance of Calories

No matter what the type of food, it must provide sufficient energy or fuel value to supply body heat and the energy necessary for the activities of the heart, lungs, skeletal muscles and digestive organs. When food is utilized in the body as fuel, the energy derived from it is ultimately given off in the form of heat. For this reason the amount of heat liberated by the body is a measure of the amount of food "burned" in the metabolic processes. This value may most conveniently be expressed in terms of heat units or *calories*.

The calorie is merely a measure of energy expressed in terms of heat. To make the meaning of this clear, let us take a simple food, such as sugar. If this is burned, heat is liberated; at the same time oxygen is taken up and carbon dioxide and water are produced. The exact amount of heat given off by this combustion and the amounts of oxygen consumed and carbon dioxide produced may be measured in an apparatus known as a calorimeter. In such an apparatus a known amount of sugar, say one gram, is placed in a small, water tight, metal "bomb," and oxygen under pressure is introduced so as to insure complete combustion. The bomb is immersed in a known amount of water, and the sugar is

ignited by an electric spark. The increase in the temperature of the surrounding water following the burning of the sugar is noted and subsequently the oxygen and carbon dioxide contents of the gas in the bomb are analyzed. If one gram of sugar is burned, and the volume of water surrounding the bomb is one liter, it is found that the temperature of the water is raised approximately 4° C. Since a large calorie (the unit used in metabolic work) is defined as the amount of heat necessary to raise the temperature of one liter of water 1° C., the gram of sugar in burning liberates four calories.

If we could imagine a small steam engine placed inside the bomb and the sugar burned under the boiler of this engine, so as to make it run, the heat of the burning sugar would be converted into mechanical energy which would, in turn, be converted back into heat, so that when the whole system once more had come to rest, it would be found that the temperature of the outside water would have been raised to exactly the same extent, as when the sugar burned directly, without first producing mechanical energy. A calorie, in terms of mechanical energy, is equivalent to approximately 3000 foot pounds, that is to say, the energy of one calorie is sufficient to raise 3000 pounds or a ton and a half to a height of one foot, or to raise one pound to a height of 3000 feet.

When sugar is "burned" in the body, it produces exactly the same amount of heat as when burned in the air, and exactly the same amounts of oxygen are utilized and carbon dioxide given off. The energy produced may be converted into such mechanical work as muscular contraction, but ultimately appears as heat. If an individual is put into a large enough calorimeter chamber surrounded by water and the air entering and leaving the chamber is analyzed, the exact amount of sugar or other foods utilized in the body may be determined and from this the amount of heat which should be produced can be calculated. It has been found by actual experiment that the amount of heat so calculated is the same as that actually produced and measured by the rise in temperature of the surrounding water.

All foods do not have the same fuel value. One gram of fat has a fuel value of a little over 9 calories in the calorimeter, and also in the body. Protein does not form the same end-products when burned in the air as in the body, but when due allowance is made for the fact that a portion of the protein used in the body

is converted only to the stage of urea, instead of being completely oxidized as in the calorimeter, it is found that the fuel value is the same; namely, about 4 calories per gram. Individual sugars, fats and proteins have slightly different fuel values. The figures given above are rough averages.

During life, the human body continuously gives off heat, the source of which is the combustion of proteins, carbohydrates, and fats, together with small amounts of other foodstuffs such as alcohol and organic acids. Even when no food is taken, the body continues to liberate heat, which comes from the utilization of glycogen of the liver and muscles, fat of the subcutaneous tissues and other fat depots and to a lesser extent of protein of the muscles and other organs.

The amount of fuel consumed as measured by the oxygen intake and carbon dioxide output, or by actual heat production, bears a definite relationship to the age, weight, and size of the body.

Basal Metabolism

The term "basal metabolism" is used to designate the heat output of an individual at complete rest and in the "postabsorptive" state, as regards food. The basal metabolism of normal infants during the first year of life has been found to average 55 calories per kilo of body weight per day (25 calories per pound). That is to say, a normal infant weighing 5 kilograms (or 11 pounds) would be expected to liberate approximately 275 calories in 24 hours. Unless the food taken in has a caloric value at least this great, the body itself is consumed as fuel. If food having a caloric value just equal to the basal metabolism were given, it would be found insufficient because the utilization of food in itself leads to a certain increase in the heat output. Carbohydrate utilization raises the rate of metabolism very little, fat somewhat more and protein very considerably. The average increase in heat production due to the taking of a milk diet is in the neighborhood of 10 or 15 per cent. Human milk with its low protein content increases the heat output less than does cow's milk with its higher protein content. This effect of food in increasing the heat output or rate of metabolism is termed the "specific dynamic action." In order, therefore, to give enough food to meet the minimum energy requirements of the body, a diet having an energy value 10 or 15 per cent above that corresponding to the basal metabolism is necessary.

Allowance for Activity

These calculations are based on the assumption that the infant remains at complete rest; but no infant remains at complete rest throughout the twenty-four hours and any activity necessarily calls for an increased expenditure of energy and a correspondingly greater heat output. Vigorous crying, while it lasts, may increase the heat output over 100 per cent. On the basis of very complete metabolic experiments carried out on infants over long periods of time, it has been determined that a fair extra allowance for activity in the case of average infants is from 20 to 25 calories per kilogram of body weight per day (10 calories per pound). Very active infants may use up more than 80 calories per kilo by extra activity, and very placid ones as little as 10 or 15 calories per kilo.

Allowance for Growth

These figures make no allowance for growth. The amounts of food required for the building up of new tissue cannot be estimated by the heat output since the food used for this purpose is not burned as fuel but is stored. An estimate may, however, be made on the basis of the known average daily increase in weights of infants and the proportions of this increase which are due to protein and fat deposition. These estimates have been checked by observations on the actual amounts of food needed to maintain stationary weight and the additional amounts necessary to bring about a normal gain. It has been found that 15 or 20 calories per kilogram of body weight per day (8 calories per pound) is an adequate growth allowance for an infant gaining at an average rate. Infants who are undernourished as the result of long-continued inadequate feeding may gain very rapidly in weight and deposit two or three times as much protein and fat as infants in average nutritional condition. These undernourished infants therefore require a growth allowance of food much in excess of the average.

Allowance for Unutilized Food

Food taken in by mouth is never completely digested and absorbed, but a certain amount is lost by way of the bowel. The quantity so lost under normal conditions is equivalent to 10 or 15 per cent of the intake, which in the case of the average infant corresponds to 10 or 15 calories per kilo of body weight daily (5

to 7 calories per pound). In the presence of diarrhea or digestive disturbance the loss of food by way of the bowel may be as great as 30 or 40 per cent of the intake.

The Total Energy Requirements

To sum up, the total caloric requirements of the average normal infant during the first year of life are as follows:

Basal resting metabolism	55 calories per kilo	(25 per pound)
Allowance for specific dynamic action	10 calories per kilo	(4 per pound)
Allowance for activity	25 calories per kilo	(10 per pound)
Allowance for growth	15 calories per kilo	(7 per pound)
Allowance for unutilized food	10 calories per kilo	(6 per pound)
Total	115 calories per kilo	(52 per pound)

Observations on normal infants have confirmed the above calculations. The actual intake of food by young healthy breast-fed and artificially fed infants, gaining in weight at normal rates, has been measured and the caloric value has been found to average from 110 to 115 calories per kilogram per day or 50 to 55 calories per pound.

The above figures are averages for the entire first year. Actually there is considerable variation, even in the case of the same infant at different periods during the first year. For the first week or two of life the metabolism is relatively low; but by the end of the second week the food requirement is as much as 120 or 130 calories per kilogram (55 to 60 per pound). Thereafter the requirement slowly diminishes to about 110 calories per kilogram (50 calories per pound) by the sixth month and 90 to 100 calories per kilogram (40 to 45 calories per pound) by the end of the first year.

The total energy requirements of the average infant are expressed graphically in Fig. 2 (Talbot).

The figures given must not be taken as literally applying to every infant. They serve merely to indicate the approximate total food requirements. Strong active infants have considerably greater energy requirements than weaker, apathetic ones of the same age and weight. The total food requirements during cold weather are greater than during warm weather. Infants who are markedly underweight or small for the age have greater food requirements than normal infants.

For underweight infants the *total* food requirement per day may

be as great as that of normal infants of the same age, but weighing much more. For example, a normal infant six months of age, weighing 15 pounds, would require in the neighborhood of 800 calories per day. A malnourished infant, 6 months of age, weighing only 7½ pounds would also require very close to 800 calories per day, which would be the equivalent of 100 calories per pound,

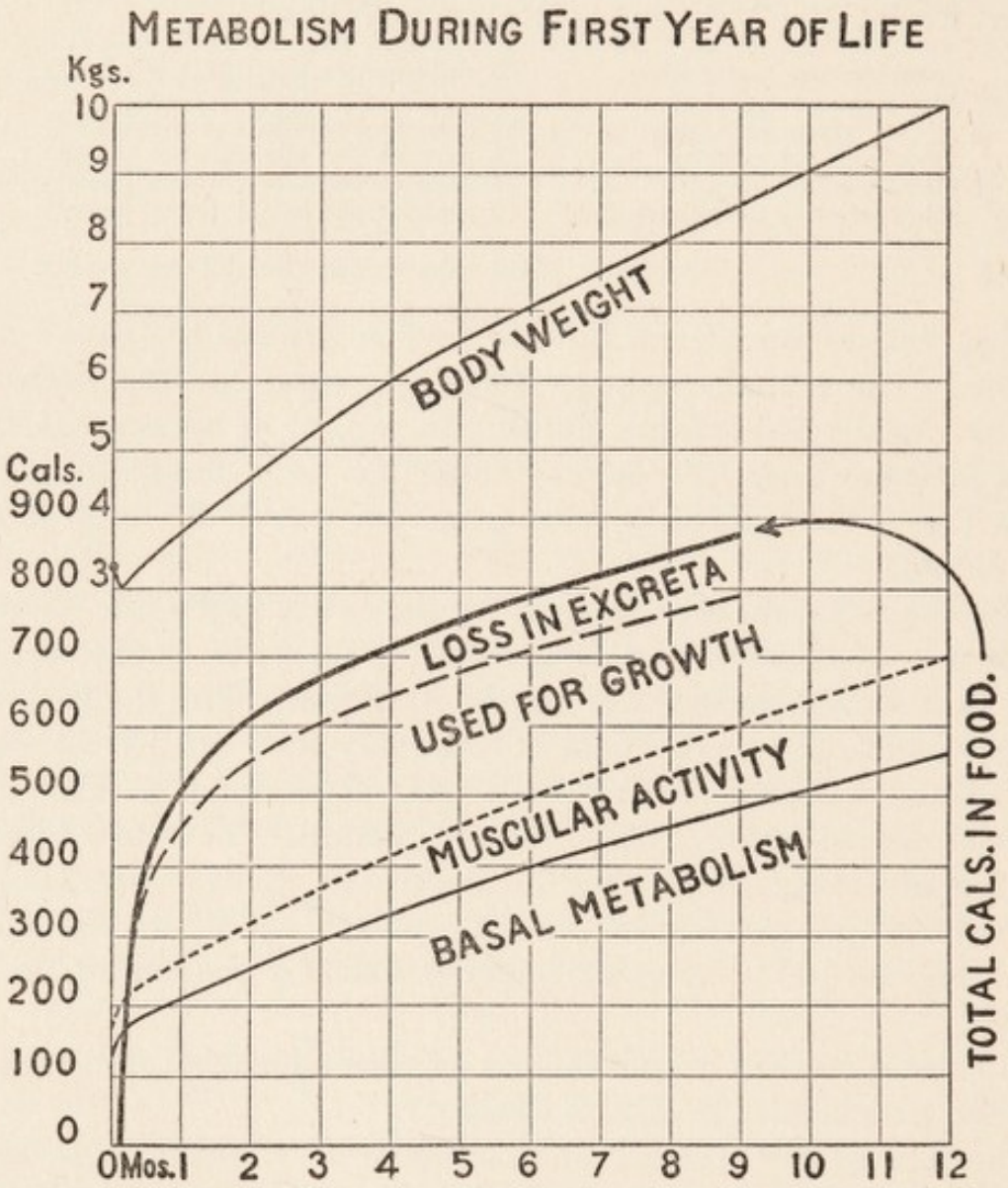


Fig. 2.—The metabolic requirements of infants. (Talbot, F. B.: Am. J. Dis. Child. XVIII, 229, 1919.)

instead of 55 as in the case of the normal infant. The reason for this is that the thin infant and the well-nourished one might have approximately the same amount of active muscular tissue and the same size heart and lungs. The chief difference would be in the layer of adipose tissue, a relatively inert substance, requiring but little food for its maintenance, the fat acting very much as an

overcoat and preventing loss of heat. It is the active protoplasmic tissue of the body which determines the food requirement rather than the actual weight. Accordingly, in attempting to arrive at an estimate as to the proper food requirement of an undernourished infant, the first step is to calculate what would be the approximate weight were the infant normal. In order to do this, reference should be made to the normal weight curves, taking into consideration the birth weight. (See Fig. 1, p. 19.)

In the case of excessively fat infants, reverse conditions hold true. The fat infant does not require much more food than a normal infant of the same age. His caloric requirement per pound of body weight, however, is less than the normal.

In the case of an individual infant, it is impossible to state exactly how many calories will be needed in order to bring about a normal gain in weight. One can, however, obtain a general idea on the basis of the principles just laid down. It is important that the caloric value of the food should be calculated in every instance in order to avoid gross errors of underfeeding. The average total caloric need of normal infants at various ages is approximately as follows:

1 month	500 calories	9 months	900 calories
2 months	600 calories	One year	1000 calories
4 months	700 calories	Two years	1200 calories
6 months	800 calories		

The final test of the adequacy of the diet is the response on the part of the infant. It is, at times, necessary to give food of a considerably higher caloric value than would be anticipated. *The giving of food of too low a caloric value to meet the infant's needs is by all odds the chief cause of failure in infant feeding.* Unless the energy requirements are met, the infant will fail to do well, even though the food may be readily digestible and otherwise admirable in composition.

It is essential that food having a sufficient caloric value be given, but the form in which the calories are supplied is also of importance. If an infant were to receive the total caloric requirement in the form of pure carbohydrate, the maintenance of life and health over a prolonged period would not be possible. Certain essential elements are necessary to supply the wear and tear of body tissues, to build up new tissues and to assure the proper utilization of the energy provided. The diet must be correct in quality as well as quantity. In the succeeding chapters the significance of the individual food constituents is considered.

CHAPTER III

PROTEIN METABOLISM

Protein is essential for the maintenance of life and for the building up of body tissue. The protein need of the infant is proportionately greater than that of the adult because of the rapid growth.

All proteins do not have the same composition or the same value in nutrition, although they are all composed of combinations of "amino-acids" united in complex chains. There are eighteen or twenty amino-acids which exist in varying proportions in the proteins. Certain amino-acids are entirely lacking in such proteins as gelatin and many of the vegetable proteins. Inasmuch as the human body is incapable of synthesizing certain of the amino-acids; incomplete proteins or those deficient in these amino-acids cannot be converted into body protein unless the essential amino-acids are supplied from some other source. For example, if a protein such as gelatin, which is deficient in the amino-acids, tyrosin, cystin and tryptophan is fed as the sole source of protein, no new body protein at all can be built up, even though the other amino-acids are present in adequate amounts. Proteins containing small amounts of essential amino-acids can build up only proportionately small amounts of body protein. The amount of protein needed by the individual, therefore, depends upon the character of the protein given. Proteins which have a composition closely simulating that of the body have a greater value in nutrition than those in which the composition differs widely from that of body tissues.

When protein of high nutritional value is fed, the minimum amount upon which the average infant can thrive is approximately 1.5 grams of protein per kilogram of body weight per day (0.025 oz. per pound). A moderate excess of protein above this minimal requirement is distinctly desirable. The average breast-fed baby receives from 2.0 to 2.5 grams of protein per kilogram (0.03 to 0.04 oz. per pound) of body weight per day, and this may be considered an optimum amount for normal infants receiving the protein of human milk. The protein of cow's milk, though well

digested and absorbed, has a lesser nutritional value, so it is essential that the artificially fed infant should receive more protein. An adequate amount is from 3 to 4 grams per kilogram of body weight per day (0.05 to 0.06 oz. per pound). This amount of protein is obtained when the infant receives a quantity of cow's milk equivalent to one-tenth of his body weight per day (1.5 ounces of cow's milk per pound of body weight). In the case of undernourished infants, the *expected* rather than the actual body weight should be taken as the basis of the calculation.

In the case of young infants, a very large proportion of the protein taken in with the food is stored in the body and does not appear as nitrogenous end-products in the urine. In young breast-fed infants, receiving relatively small amounts of protein, as much as 80 per cent may be stored in this way. The growing infant is normally in a state of positive nitrogen balance, whereas the fully grown individual excretes in the urine as much nitrogen in the form of end-products as is contained in the ingested protein.

The young infant, whether breast fed or artificially fed, receives its protein chiefly from milk. In both human milk and in cow's milk the proteins may be divided into two main groups—whey protein, or *lactalbumin,* and curd protein, or *casein.* Lactalbumin differs materially from casein in its composition, being the more valuable of the two proteins, because it has more nearly the composition of body protein. Casein is relatively deficient in certain amino-acids, and hence larger amounts must be fed than in the case of lactalbumin in order to provide for the building up of the same amount of body protein. Human milk contains less protein than cow's milk, but of the total protein present, almost 60 per cent is lactalbumin as compared with about 15 per cent in the case of cow's milk. It is for this reason that artificially fed infants require more protein than those receiving mother's milk.

When the protein content of the diet is insufficient, growth is slow, resistance to infection decreased, the musculature becomes flabby, and secondary anemia develops. In addition, in severe or prolonged protein starvation, edema not referable to cardiac or renal disease may develop (nutritional edema). This may be extreme. A moderate excess of protein in the diet above the figures mentioned apparently does no harm. It is well digested and absorbed and causes no general disturbances. A diet containing an enormous excess of protein with the simultaneous exclusion of

other dietary factors leads to a condition of dehydration with concentrated blood, fever and symptoms of "intoxication." It would be impossible to feed such excessive amounts of protein except by the addition of extra protein in dried form to whole cow's milk. Hence, the danger of excess of protein in the diet may be considered practically nonexistent.

One effect of high protein in the diet is to lead to an increased secretion of alkaline intestinal juices. At the same time little if any acid is produced by bacterial decomposition of protein, so that the stools become alkaline in reaction and constipated in type. Casein is more effective than lactalbumin in bringing about this phenomenon, as it appears to be less readily attacked by intestinal bacteria, and furthermore leaves an alkaline residue with a high lime content. This effect is especially desirable in cases of fermentative diarrhea.

Anaphylaxis

Protein is, under normal conditions, completely broken down into amino-acids before absorption from the intestinal tract. (See Chapter IX.) In the case of very young infants, however, or those suffering from gastrointestinal disturbances some incompletely digested protein may be absorbed directly into the blood stream. In rare instances, the amount absorbed is sufficient to sensitize the individual against the particular protein so that subsequent feeding of the same protein may result in anaphylactic phenomena. Definite anaphylaxis to the proteins of cow's milk is a rare but serious condition, calling for the temporary withdrawal of all cow's milk protein and subsequent gradual desensitization. The feeding of raw egg white to very young infants is generally to be avoided because of the possibility of sensitization. The feeding of egg yolk or of cooked meat is, however, not likely to lead to sensitization.

CHAPTER IV

CARBOHYDRATE METABOLISM

A certain amount of carbohydrate is necessary for the maintenance of life. The minimum amount upon which a young infant will survive for any length of time is about 3 grams per kilogram of body weight (0.05 ounce per pound). The optimum amount of carbohydrate during infancy is from 8 to 14 grams per kilogram of body weight per day (0.13 to 0.22 ounces per pound) or approximately 1/100 of the body weight. The infant fed on human milk receives about 12 grams of carbohydrate per kilo of body weight (0.2 ounces per pound) per day. The infant not only needs more carbohydrate per unit of weight than the adult, but is also capable of taking much more carbohydrate in proportion to his weight without the development of glycosuria.

Carbohydrates are chemically much simpler substances than proteins. They are composed only of carbon, hydrogen and oxygen, the latter two elements being in approximately the same proportions as in water. The simplest carbohydrates are the monosaccharides, such as dextrose, levulose, and galactose. Two molecules of any of these joined together form the disaccharides, examples of which are cane sugar, milk sugar, and malt sugar. These latter are the forms of carbohydrate usually fed to infants. In addition to these simple sugars, there are carbohydrates containing a larger number of molecules of the monosaccharides, as, for example, starch, which is composed of a very large number of molecules of dextrose. Intermediate between starch and the disaccharides are the dextrins, which represent partially broken-down starch molecules.

All carbohydrates are ultimately utilized by the body in the form of monosaccharides or simple sugars, usually dextrose. The conversion to monosaccharides occurs in the intestinal tract. (For details, see Chapter IX.) Only when the intestinal mucosa is injured does any carbohydrate, except in the form of the monosaccharides, pass into the circulation. Neither cane sugar nor milk sugar can be utilized when injected into the blood stream or absorbed from the intestinal tract. Each of these is excreted unchanged. Maltose, on the other hand, may be partially utilized

and the monosaccharides, dextrose, levulose or galactose are completely utilized when introduced into the circulation.

During the absorption of carbohydrate from the intestinal tract, there occurs a moderate rise in the dextrose content of the blood. During fasting the dextrose content of the blood averages about 0.1 per cent. At the height of digestion of a meal containing carbohydrate, the blood-sugar level does not usually increase to more than 0.15 per cent. With an increase as high as 0.17 per cent, which rarely occurs in the normal individual except when huge amounts of carbohydrate are given, some dextrose passes out into the urine.*

The level of the blood sugar is kept within these narrow limits by an efficient regulatory mechanism. Dextrose which is needed for fuel is promptly burned in the tissues, the remainder is stored in the liver and muscles in the form of glycogen or animal dextrin or else converted into fat and stored as such in the subcutaneous tissues and the other fat depots of the body. During starvation the stored glycogen is reconverted into dextrose, which is poured into the blood and utilized as food. In long-continued starvation the glycogen reserves of the body may be depleted so that the level of the blood sugar falls. Some additional dextrose becomes available through the metabolism of protein, approximately 60 per cent of the latter being convertible into dextrose. This tends to prevent the blood sugar from falling very far below the normal level. When the blood sugar falls lower than 0.04 per cent, which is unusual except after the administration of insulin, definite symptoms of "hypoglycemic shock" may occur. A moderate fall in the blood sugar results in hunger contractions of the stomach and the individual experiences a sense of hunger. The administration of quickly absorbable carbohydrate, by raising the blood-sugar level, tends promptly to satisfy hunger.

The rate of utilization of carbohydrate and its transformation into fat is markedly accelerated by the administration of insulin. Advantage is taken of this fact in the treatment of undernourished infants (see Chapter XIX).

Carbohydrate is the most readily available supply of energy for the body and may replace either protein or fat for this purpose. The administration of adequate amounts of carbohydrate

*The figures for blood dextrose are based upon the usual blood-sugar determination methods. These methods give values somewhat in excess of the "true" sugar of the blood, which is about 0.02 per cent lower.

spares body protein from destruction during periods of underfeeding. Carbohydrate, however, cannot entirely replace protein in the diet as it cannot build up the essential protoplasmic structures of the body. In the case of infants who are much underweight, it is often desirable to feed large amounts of carbohydrate, and this is without danger, provided the carbohydrate is not of the readily fermentable type and there is fed at the same time an adequate amount of protein and fat. The administration of a diet consisting mainly of carbohydrates and deficient in protein, fat, and minerals, may result in a rapid increase in weight, which is due largely to water retention. Infants so fed are pale, flabby, waterlogged, rachitic and lack resistance to infections. The effects of such a diet are due not so much to an excess of carbohydrate as to deficiencies in other constituents. Sweetened condensed milk and malted milk are examples of foods of the type just mentioned.

The carbohydrates commonly used in infant feeding are milk sugar (lactose), cane sugar (sucrose), maltose, dextrin, dextrose and starch. Any of these forms of carbohydrate ultimately serve the same functions in nutrition. In the choice of a carbohydrate, however, certain characteristics should be taken into consideration which are different for the various sugars. One has to consider the ease of digestion and absorption, the degree to which the carbohydrate is fermented in the intestinal tract, and the degree to which it irritates the intestine.

Milk Sugar (Lactose)

Lactose is the sugar occurring naturally in the milk of all mammals. There would seem to be a theoretical advantage in using this natural sugar as an addition to milk formulas, but, as a matter of fact, milk sugar has very few advantages over other sugars for this purpose and has certain distinct disadvantages. If there is any special need for lactose by the body (and it has never been proved that there is), such a need is met by the lactose present in the milk which invariably forms the basis of the infant's diet. Experience has shown that the remainder of the carbohydrate may be supplied by some other sugar with no deleterious effects on the nutrition. Milk sugar is not so sweet as cane sugar, and this is a certain advantage in that it does not accustom the infant to excessive sweetness of the food. Lactose is readily fermented by most of the normal intestinal bacteria of

infants with the production of various acids, including lactic acid. Lactose, more than other sugars, tends to make the stools strongly acid in reaction and sour in odor. Lactose, when fed, is not digested and absorbed so rapidly as some other sugars and reaches lower portions of the intestinal tract. In the case of most babies, it exerts a more laxative effect than the other sugars commonly used in infant feeding. Consequently, it cannot be used with as great safety or fed in as large amounts as other carbohydrates. Claimed advantages of lactose are that its presence encourages the growth of the type of intestinal bacteria that predominate in breast-fed infants and further that the acid produced favors the absorption of calcium salts.

Cane Sugar (Sucrose)

Cane sugar (sucrose or beet sugar) has the same nutritive value as milk sugar. It is somewhat more quickly digested and absorbed from the intestinal tract and does not ferment as readily with most intestinal bacteria; it is, however, fermented by yeasts. Cane sugar is not so laxative as milk sugar and may with safety be fed in somewhat larger amounts, especially to infants suffering from digestive disturbances. In concentrated solution, it is irritating to mucous membranes and as a result is likely to cause vomiting. This does not apply, however, to the more dilute solutions. Cane sugar is the sweetest of the commonly used sugars. In general, cane sugar is a suitable form of carbohydrate for the feeding of the average infant.

Malt Sugars

When starch is subjected to the action of malt diastase, or heated, especially if in the presence of acid, it is converted first into intermediate products known collectively as the dextrins. These, by the further action of diastase, or heat, are converted into maltose and ultimately into dextrose.

Starch
↓
Dextrin
↓
Maltose
↓
Dextrose

These are the same stages through which starch passes during digestion in the body. The composition of the final mixture will depend upon the length of the time that the processes of conversion are allowed to proceed. If only for a short time, there will be relatively large amounts of dextrin present. If for a longer time, there will be more maltose and some dextrose. The properties of the various malt preparations depend, to a considerable extent, upon the relative proportions of the different constituents present.

Dextrin

Dextrin is really a mixture of a number of closely related substances. It is a white, or as usually obtained, slightly yellowish, somewhat sticky powder. It does not have a sweet taste, but is, if anything, slightly bitter. It is not very irritating, even in highly concentrated solutions, because of its low osmotic pressure. Dextrin is not fermented by most intestinal bacteria; it is, however, readily converted into maltose through the action of the enzymes normally present in the intestinal tract. This conversion does not appear to occur much more rapidly than the maltose produced can be digested and absorbed so that when dextrin is fed there is not, at any one time, a great deal of readily fermentable carbohydrate present in the intestinal tract. Dextrin, therefore, is not likely to lead to intestinal fermentation, and this is an especial advantage in the presence of diarrhea. Dextrin may be given safely in larger amounts than other carbohydrates and its value in nutrition is as great. Dextrin alone has not been widely used in infant feeding; mixtures of dextrin and maltose being the customary form. Fairly pure commercial dextrin is obtainable at a reasonable price, and is especially valuable as a carbohydrate addition to milk formulas in the treatment of diarrheal conditions.

Maltose

Pure maltose is not used in infant feeding, but mixtures of maltose and dextrin are extensively used. The proportion of maltose in these preparations ranges from 30 to 60 per cent. Maltose itself is a white powder, slightly sweet. It is quite readily fermented by most intestinal organisms, but at the same time is quickly converted into dextrose and absorbed, so that unless excessively large amounts are fed, serious fermentation is not likely

to result. The products of fermentation of maltose do not seem to be as irritating as those of cane sugar or milk sugar.

Dextrose (d-glucose)

Dextrose requires no digestion, being immediately absorbable. Dextrose is very easily fermented, but is the most quickly absorbed of all the sugars, disappearing from the intestinal tract with astonishing rapidity. Fairly large amounts of dextrose may be added to the food of an infant without the danger of any great degree of intestinal fermentation. Dextrose in concentrated solution is irritating but apparently not so much so as either cane sugar or milk sugar. Pure dextrose has been used in infant feeding, but mixtures of dextrose with maltose and dextrin are, for most purposes, preferable.

Mixed Sugars

A mixture of several carbohydrates possesses certain advantages over a single sugar, as larger amounts of such mixtures may usually be fed without the danger of producing diarrhea. At times the intestinal tract contains organisms which ferment certain carbohydrates more readily than others. When a mixture of carbohydrates is fed only certain of the components may be fermented so that excessive fermentation at any one time is not likely to occur. This explains the especial advantages of mixtures of dextrin, maltose and dextrose as additions to milk which already contains lactose.

There are a large number of such mixed sugars on the market. The composition of these is given in detail by the manufacturers. Of the dry preparations, prepared by the action of malt diastase, the most widely used are Mead's Dextrimaltose and Mellin's Food. Mead's Dextrimaltose, having a somewhat higher proportion of dextrin and less maltose, is somewhat less laxative than Mellin's Food. Either of these, however, may be given in larger amounts than cane sugar or milk sugar without the danger of producing gastrointestinal disturbances.

Differing from the other dextrin-maltose mixtures are Squibb's Vitavose, which is prepared from the germ of wheat and Mead's Dextrimaltose with vitamin B which contains extracts of wheat germ and of yeast. Both of these contain large amounts of vi-

tamin "B"; the properties are otherwise similar to the other maltose dextrin preparations.

Starch, when toasted or heated dry, is partially converted into dextrin. A number of such dextrinized-flours are on the market (Imperial Granum, Allenberry's Food, etc.). The properties of these dextrinized-starches are intermediate between starch and dextrin.

Liquid malt preparations (malt extract) have a distinctly greater laxative effect than the dried sugars. This laxative effect is due to the presence of extractives and possibly to the active diastase still present. The liquid malt extracts are not used for the routine feeding of normal infants, but are used when there is a tendency to constipation, a portion of the sugar being then substituted by the malt extract. Examples of such malt extracts are Borcherdt's Malt Extract, Keller's Malt Soup Stock, Loeflund's Malt Soup Extract.

Karo Syrup (corn syrup) is a mixture of dextrin, maltose and dextrose, with a small amount of added cane sugar, and is prepared by the acid hydrolysis of cornstarch. Karo Syrup contains a large proportion of dextrin with relatively small amounts of maltose, dextrose and cane sugar. The dextrin is nonirritating and nonfermentable, but is readily converted into maltose and dextrose and absorbed. There is not sufficient maltose, dextrose or cane sugar present to cause irritation or any considerable degree of fermentation. Karo Syrup may, therefore, be fed in large amounts without danger and is, at the same time, readily utilized. In our experience, it has been the most satisfactory form of carbohydrate for the feeding of normal and most sick infants.

Starch

Starch occurs as the chief constituent of all cereal grains and the tuberous vegetables. It has approximately the same composition, no matter what its source, but varies considerably in physical properties. Starch occurs in nature in small granules surrounded by envelopes of cellulose. On heating the grains, the cellulose envelopes rupture and the starch is liberated. Raw starch is digested only with great difficulty because of the indigestibility of the cellulose envelope. Starch used for the feeding of infants is prepared by milling the grain followed by thorough cooking. Even when properly prepared and well cooked, starch is incom-

pletely digested by the very young infant. If, however, starch is fed regularly from an early age, the infant gradually develops the power of digesting it fairly well, although at first much starch may be detected in the stools by the iodine test.

Starch does not ferment readily and acts, to some extent, as a demulcent in the intestinal tract. It is probable that the passage of undigested starch through the infant's intestinal tract does little or no harm in most instances, though occasionally it may give rise to slight fermentation and gas production.

Decoctions of starch, being colloidal, possess certain advantages as additions to infant diets. When starch gruels are added to cow's milk, the curds precipitated by the action of acid and rennin are more finely divided than would be the case otherwise. Another valuable use of starch in infant feeding is in rendering milk mixtures so thick as not to be vomited readily. Such thick gruel feedings are used in the treatment of vomiting of various types, such as that due to pyloric stenosis or rumination. (See Chapters XXIII and XXIV.) The starches chiefly used in infant feeding are barley, wheat, oat, corn and rice flours. Of these, barley flour is the most widely used as it makes the most demulcent gruel.

During the process of digestion of starch, it is converted through the stages of dextrin and maltose to dextrose, in which form it is finally absorbed. It is not possible to meet the carbohydrate needs of the young infant by starch alone, but after the age of six months, starch may gradually replace the simple sugars in the diet.

The Carbohydrates of Fruits

All fruits contain carbohydrates, largely dextrose and levulose, and these are utilized readily by the infant. Of especial interest is the carbohydrate of bananas, which comprises about 22 per cent of the total weight of the ripe banana. This consists of a mixture of sugars, chiefly sucrose, dextrose and levulose, which, for some unexplained reason, is very well digested and absorbed even by infants and young children with gastrointestinal disturbances. Ripe bananas have been used largely in the treatment of chronic intestinal indigestion (celiac disease). (See Chapter XXII.) Dried banana meal has found some use in infant feeding.

Honey consists of a mixture of sugars, chiefly levulose and dextrose with some cane sugar. It is rather readily fermentable

and possesses no especial advantages over other forms of sugar. It has been claimed that it contains vitamins, but the amount of these present is negligible.

Sugars as a Cause of Diarrhea

Any sugar, when fed in excessive amounts, may cause gastro-intestinal fermentation and diarrhea. The sugars which have smaller molecules, as the monosaccharides, dextrose and levulose, and the disaccharides, lactose and sucrose, when in solution, have greater osmotic pressures than the higher carbohydrates, such as dextrin and starch. Concentrated solutions, therefore, act somewhat as hydragogue cathartics. Solutions of dextrose stronger than 5 per cent, or of milk sugar and cane sugar stronger than 8 per cent are hypertonic and likely to exert some laxative effect for that reason alone, aside from fermentation. Dextrin and starch, on the other hand, may be given in solutions of a concentration of 20 per cent or more without being hypertonic. This is an important practical point in the construction of infant feeding formulas. It was, at one time, very generally stated that the total sugar in the final formula fed should not exceed 7 to 8 per cent. We now know that when sugars containing a considerable amount of dextrin are used, sugar concentrations up to 20 per cent may be given without harm and may at times even exert a constipating effect, because of the absorption by the intestine of large amounts of water along with the sugar.

Parenteral Administration of Carbohydrate

Of the three chief food substances, protein, carbohydrate and fat, carbohydrate is the only one which may be administered other than by way of the intestinal tract. Cane sugar, milk sugar, dextrin and starch are not utilized when given parenterally, but dextrose is utilized when given intravenously, subcutaneously or intraperitoneally. In the case of infants with lowered intestinal tolerance to carbohydrates, the parenteral administration of dextrose may serve to maintain the nutrition over critical periods. Dextrose solutions, when injected parenterally, also serve other important functions. (For further discussion, see Chapters XIX and XXXIV.)

CHAPTER V

FAT METABOLISM

The fats, like carbohydrates, are composed of carbon, hydrogen and oxygen. The proportions, however, are quite different from those in the case of carbohydrates, there being considerably less oxygen present in proportion to the carbon and hydrogen. All fats consist of a combination of fatty acids with glycerin.

Fats as such cannot be absorbed from the intestinal tract, but must first be *saponified.* Saponification consists in a separation of the fatty acid portion of the fat from the glycerin and is accomplished by the action of the pancreatic juice. (See Chapter IX.) The alkaline salts of the fatty acids are known as soaps. Fat, after saponification, is absorbed as glycerin and fatty acid (or as soap). These are reunited after passage through the intestinal wall to form neutral fat. There is, at the same time, a considerable rearrangement so that the recombined fat resembles more nearly the natural fat of the individual than the food fat. Fat is not absorbed directly into the blood, but is taken up by the lymphatics and carried to the blood stream in a fine emulsion by way of the lacteals and the thoracic duct.

The fat which enters the blood is in part carried to the subcutaneous tissues and deposited, and in part to the liver, where it undergoes a chemical transformation, rendering it capable of being metabolized as fuel. When there is relatively abundant carbohydrate and protein in the diet, but little fat is burned, the major part being stored. During starvation, however, a large portion of the energy requirement of the body is met by the utilization of fat, which is removed from the fat depots, carried to the liver by way of the blood and subsequently metabolized. The amount of fat used by the body, therefore, is dependent upon the state of nutrition and the relative amounts of other foods fed and not merely upon the intake of fat. An infant who is starving may actually use larger amounts of fat than one who is fed on a diet containing much fat, but at the same time adequate carbohydrate and protein.

Fat cannot be completely metabolized by the human body for energy unless a certain amount of carbohydrate is simultaneously used, or, as it has been aptly stated, "fats burn only in the fire of

carbohydrate." One molecule of dextrose is necessary for the complete combustion of two molecules of fatty acid, and unless this amount of dextrose is present and being used at the same time, fats are broken down only to the stage of the acetone bodies, and this may lead to acidosis. (See Chapter XXXII.) Overfeeding with fats does not produce acidosis unless there is, at the same time, a diminished intake of carbohydrate and protein, the latter being in part convertible into carbohydrate in the body.

Fat is not absolutely essential in the diet, as it may be replaced by carbohydrate or protein as sources of energy. Fat, however, has a caloric value more than twice that of either carbohydrate or protein and serves very well to make up the necessary energy or caloric requirements. When fat is omitted from the diet, it becomes necessary to give excessively large amounts of carbohydrate and protein. In both human and cow's milk, the fat provides approximately one-half of the total calories. Two of the important vitamins, "A" and "D" (see Chapter VII) are associated with the fat of milk, and when the diet is low in milk fat, these vitamins must be supplied in some other form.

Fat occurs in the milk of all mammals, but differs considerably in composition in the various species. The total amount of fat in average cow's milk, as supplied by dairies, is approximately the same as that in average human milk (3.5 to 4 per cent), but the fat of cow's milk is more irritating to the intestinal tract and less digestible than that of human milk. Cow's milk fat contains more of the esters of the lower fatty acids (butyric, caprylic, caproic, etc.). When liberated by saponification, these lower fatty acids have an irritating action upon the intestinal mucosa, which may result in vomiting or diarrhea. The irritating effect of these lower fatty acids is more marked when an excess of fermentable carbohydrate is present at the same time in the intestinal tract. Cow's milk fat differs further from the fat of human milk in that it contains a relatively larger proportion of palmitic and stearic acid fats and less oleic acid fat. Palmitic and stearic acids and their soaps are not so soluble in the intestinal juices or so readily absorbed as the oleic acid derivatives. These insoluble soaps, particularly the lime soaps formed when cow's milk is fed, tend to make the stools firm, bulky and alkaline, especially if considerable protein with its high content of casein and lime is fed. The effect of overfeeding with fat is to produce constipation when relatively

little carbohydrate and considerable protein is given at the same time. On the other hand, high fat feeding results in diarrhea when much fermentable carbohydrate is contained in the diet and at the same time relatively little casein.

In general, an excess of fat in the diet of the artificially fed infant should be avoided. Ordinary grades of cow's milk containing not more than 3.5 to 4 per cent of butter-fat should be used in preference to the richer milk of Jersey or Guernsey cows. Top milk or cream mixtures are best dispensed with entirely, although the deleterious effects of these may be partially neutralized by the simultaneous administration of lime water. The relationship between fat and casein in ordinary grades of cow's milk is such that it is only in the presence of severe gastrointestinal irritation that it is necessary to reduce the fat content by partially skimming the milk.

The fat of milk exists in the form of a fine emulsion, the individual globules of which are somewhat smaller in human milk than in cow's milk. It is not certain how much of a factor this is in rendering the fat more digestible. Cow's milk which has been homogenized, that is, atomized by passage through a fine hole under pressure, has the fat in the form of extremely fine globules. Evaporated milk, some forms of dried milk, and certain proprietary infant foods such as "S.M.A." are homogenized.

Because of the relative indigestibility of the fat of cow's milk, efforts have been made to substitute a more digestible fat for the feeding of infants. In early attempts, the cream of milk was removed and an equivalent amount of oil in the form of olive oil was substituted and the whole homogenized. Later, refinements have consisted in the substitution of a mixture of fats containing among others cod liver oil with its high vitamin content, the mixture of fats being such as to resemble in physical and chemical properties the fat of human milk. The best known example of such a reconstructed milk is "S.M.A." (Synthetic Milk Adapted) of Gerstenberger. (See Chapter XVII.) Another method of altering the fat of cow's milk has been that used in the preparation of the "butter flour mixture" of Czerny and Kleinschmidt. In the preparation of this (see Chapter XVII) butter is boiled in an open saucepan to remove volatile fatty acids. It is then heated with flour and water in order to effect emulsification and added to milk from which a portion of the cream has been removed.

CHAPTER VI

MINERAL AND WATER METABOLISM

Protein, fat and carbohydrate furnish the necessary sources of energy for the body. Protein, in addition, supplies materials for the building up of new tissue, but these three foodstuffs together fail to supply all of the materials necessary for the maintenance of life. Mineral matter, in addition to the three foods mentioned, is essential. All cells, tissues and fluids of the body contain mineral salts which serve to maintain a constant osmotic pressure, to supply the necessary balance of ions for the normal functioning of cells, and to aid in the digestion of food. Even on a diet free from mineral matter, there occurs a constant excretion of mineral salts; therefore, unless an equivalent amount of these salts is supplied in the diet, wasting of the body occurs. Without mineral matter, no new body tissue can be formed. For each gram of protein laid down in the body, about 0.3 grams of mineral matter is also stored. There exists a definite relationship between the mineral salts, especially sodium chloride, and water contents of the body. When loss of mineral matter occurs as the result of a salt-deficient diet, of starvation, or of prolonged diarrhea, the body becomes incapable of maintaining a normal water content.

When the diet is lacking in any one of the essential mineral salts, normal growth and function become impossible. When mineral salts in excess of the requirements are ingested, the amounts necessary are retained and the remainder excreted. The mineral composition of the body is usually but little altered from the normal by the administration of a fairly large excess of any of the inorganic salts, although there is some evidence that a large intake of potassium salts results in an increased excretion of sodium salts, and the ingestion of an excess of magnesium salts leads to some calcium loss.

The bony framework of the body contains chiefly calcium, magnesium and phosphates. The body cells are relatively rich in potassium salts and phosphates and the body fluids in sodium salts, chlorides and bicarbonates. Iron is an essential constituent

of hemoglobin and iodine of the thyroid gland. A disturbance of the balance between the various inorganic salts in the circulating fluids occurs in certain pathologic conditions. In tetany, there is a relative deficit of calcium salts in the blood plasma and in acidosis a decrease in the sodium bicarbonate of the plasma and tissue fluids. Such alterations in the composition of the body fluids are usually due to disturbances of the metabolism rather than to a poor balance of salt in the diet.

Fortunately many common foods contain fair amounts of mineral salts. Milk, the chief food of the infant, is a rich source of mineral matter, containing all of the inorganic salts which are essential for life and especially large amounts of calcium. Green vegetables contain sodium and potassium, small amounts of calcium and iron and traces of iodine; fruits and fruit juices, a good deal of potassium, and the whole cereal grains a variety of salts, including calcium, phosphates and iron. Eggs contain considerable calcium and iron.

Sodium and Potassium

Sodium salts occur almost entirely in the fluids of the body—the blood plasma, lymph, cerebrospinal fluid, gastric and intestinal secretions. Potassium salts, on the other hand, are the chief inorganic constituents of the fixed tissues and red blood cells and are present in but small amounts in body fluids. The urine usually contains a fair amount of potassium, which represents the excess not needed by the body and also the residue from cell destruction.

Sodium of the body fluids is in combination chiefly with bicarbonate and chloride. The chloride is the chief factor in maintaining a normal osmotic pressure. Sodium bicarbonate, and to a lesser extent, sodium phosphate serve to maintain a normal hydrogen-ion concentration and acid-base equilibrium. (See Chapter XXXII.)

A constant supply of sodium salts in the diet is essential as there is always a loss of some sodium by way of the urine and intestinal secretions. A moderate excess of sodium salts, either as chloride or in other forms, is readily excreted and apparently does no harm. The hydrochloric acid of the gastric juice is derived from the sodium chloride of the blood. The alkaline carbonate of the bile, pancreatic juice and intestinal secretions is

derived from the sodium bicarbonate of the blood. Some sodium chloride is also secreted with the gastric juice. The sodium salts poured out into the intestinal tract are largely reabsorbed but not entirely. Appreciable amounts of sodium are lost in the form of sodium soaps when much fat is fed. Sodium is also lost in the form of salts of organic acids when acid fermentation occurs in the intestinal tract. In the presence of severe diarrhea, very large amounts of sodium salts, as well as of other inorganic constituents of the body, are lost by way of the bowel. Acidosis results in an increased excretion of sodium as well as other fixed bases by way of the urine.

An infant, receiving sufficient human milk to meet his energy requirements, receives, at the same time, adequate amounts of sodium salts for normal nutrition. Cow's milk contains something more than three times the quantity of sodium salts as human milk, so that the infant receiving artificial feeding, even though dilute, usually obtains an adequate supply of sodium. It is only in such exceptional conditions as acidosis or after severe diarrhea or prolonged starvation that the administration of additional quantities of sodium salts is necessary. (See Chapters XX and XXXII.)

The potassium needs of the infant are relatively much greater than those of the adult because of the necessity for building up of new body tissues, all of which contain potassium. Both human milk and cow's milk contain more potassium than sodium salts, the excess of potassium over sodium being somewhat greater in the case of human milk. Cow's milk, however, contains in absolute amounts almost twice as much potassium as human milk. There is sufficient potassium in either form of milk to meet all needs of the normal infant, and additions of potassium salts to the diet are not usually necessary. Potassium salts in excess of the needs of the body are very rapidly excreted by way of the urine except when there is impairment of renal function, in which case some retention of potassium salts in body fluids may occur. Since an excess of potassium salts in the circulating fluids exerts a distinctly deleterious effect, especially on the heart muscle, it is dangerous to administer potassium salts in the presence of possible renal insufficiency. In this connection, the practice of administering potassium citrate in the treatment of pyelitis should be condemned.

Calcium and Magnesium

The growing infant requires relatively large amounts of calcium to provide for bone growth. The more rapidly the infant grows, the greater the calcium need. Furthermore, the maintenance of the calcium content of the blood plasma above a certain minimum level is essential, or symptoms of tetany may occur. The deposition of calcium salts in the bones and the maintenance of the calcium content of the body fluids is, however, dependent chiefly upon factors other than the calcium intake. Even with a large intake of calcium salts in the food, absorption may be very poor. An excess of phosphate or of fat in the diet leads to the formation in the intestinal tract of relatively insoluble calcium phosphate or calcium soaps which are absorbed with difficulty, especially if the intestinal contents are alkaline.

The amounts of calcium absorbed and retained in the blood or deposited in the bones is influenced largely by the amount of the antirachitic or "D" vitamin in the diet, or the amount of this vitamin developed in the body as the result of exposure to ultraviolet light. (See Chapters VII and XXIX.) When the calcium intake is extremely low, a condition somewhat similar to rickets may be observed, but at the same time the concentration of calcium in the blood plasma does not necessarily fall to a very low level, the blood calcium being maintained by withdrawal of calcium salts from the bones. When the antirachitic vitamin is deficient, rickets may occur, even despite an adequate calcium intake. When the parathyroid hormone is deficient, the calcium content of the blood may fall from the normal 10 or 11 mg. per 100 c.c. to 5 mg. or less, even when there is a fairly adequate calcium intake. The administration of vitamin D, exposure of the body to sunlight or the administration of the parathyroid hormone (parathormone) leads to an increase in the blood calcium content. When the blood calcium content is low, it may be increased temporarily by the feeding of large amounts of calcium salts. When calcium salts are fed by mouth or injected parenterally, the calcium is excreted chiefly by way of the bowel, and the anion or acid component by way of the urine. The administration of calcium chloride is, therefore, to a certain extent, equivalent to the administration of hydrochloric acid, since the acid component must be neutralized and eliminated. The organic calcium salts, the lactate, acetate and

gluconate act only as bases, the acid components of these salts being completely metabolized to carbon dioxide and water.

Cow's milk contains from three to four times as much calcium as human milk, but when cow's milk is fed to infants, a smaller proportion of the calcium present is absorbed. This poor absorption of calcium from cow's milk is probably due to the greater relative excess of phosphate present, which tends to hold the calcium in insoluble form, and to a lesser extent to the character of the fat and the fact that the intestinal contents tend to be more alkaline following the feeding of cow's milk. In any event, there appears to be sufficient utilizable calcium in either human milk or cow's milk to meet the needs of the average normal infant provided reasonable amounts of either form of milk are given. An infant taking as much as 2.5 ounces of human milk or 1.5 ounces of cow's milk per pound of expected body weight receives sufficient calcium salts for normal requirements. It is only in the treatment of such a pathologic condition as tetany that the addition of calcium salts to the diet is indicated. The methods of the administration of calcium salts are discussed in Chapter XXX.

Magnesium metabolism is very similar to that of calcium. Calcium and magnesium are, to a certain extent, antagonistic in their actions, but so far as tetany is concerned, the administration of magnesium relieves the symptoms as does calcium. There is a small amount of magnesium present in the bones, muscles and blood serum. Just how much magnesium is needed by the young infant it not certainly known, but it may be assumed that human milk contains sufficient to meet the requirements. Cow's milk contains more than twice as much magnesium as human milk. Vegetables and meats contain fair amounts of magnesium.

Iron

Iron is a necessary constituent of hemoglobin and of muscle cells. Inasmuch as a large amount of hemoglobin is formed during the first year of life, an adequate supply of iron is essential. (For further discussion, see Chapter I.) The amount of iron in human milk is very small and in cow's milk considerably smaller. There is barely enough iron in human milk to meet the needs of the developing infant and an insufficient amount in cow's milk. Therefore, some breast-fed infants, and most artificially fed infants, during the latter half of the first year, have a low hemoglobin content

of the blood unless additional iron-containing foods are given. The administration of a small additional amount of iron after the sixth month of life, either in the form of iron salts or as green vegetables, meats, egg yolk or cereals is desirable. Green vegetables, especially spinach, are of further advantage in supplying the chlorophyl pigment, which may be utilized in the synthesis of hemoglobin. The feeding of small amounts of whole liver may likewise be expected to have a good effect in supplying iron and blood pigment. Liver extracts, on the other hand, are not of specific value in preventing the alimentary anemia of infants.

Iodine

A very small amount of iodine is a necessary constituent of the thyroid gland. Sufficient amounts of iodine are present in human milk or in cow's milk to meet the needs of the infant under ordinary circumstances, and symptoms of colloid goiter are extremely rare during infancy. In goitrous regions, however, it is conceivable that both human and cow's milk may be deficient in iodine. The administration of a little sodium iodide to the mother or the addition of a few milligrams of iodide to the formula used for the feeding of the infant might be resorted to under such circumstances.

Chlorides

Both human milk and cow's milk contain adequate amounts of chlorides for the normal requirements of the infant. The addition of chlorides in the form of sodium chloride to the diet is, therefore, usually unnecessary. When vomiting occurs, chlorides are lost by way of the gastric juice and this loss may be sufficient to deplete largely the chlorides of the blood. Under such circumstances, the administration of sodium chloride is indicated. (See Chapters XXIII and XXXII.)

Phosphates

Phosphates are necessary in the diet to provide for the structure of bone and of protoplasm and for the maintenance of the acid-base equilibrium of the body. Human milk contains all of the phosphate required by the infant and cow's milk contains seven or eight times as much phosphate as human milk. This excess of phosphate in cow's milk tends to render calcium absorption

poorer, but otherwise seems to produce no serious effects. There are, so far as is known, no conditions during infancy which call for the administration of an extra amount of phosphate above that contained in the usual infant dietary.

Elementary phosphorus has been used in the treatment of rickets and certain bone diseases. The phosphorus has usually been administered with cod liver oil, the dosage of phosphorus being very minute, generally not more than 1/200 grain (0.3 mg.) to 1/100 grain (0.6 mg.) per day. The extra amounts of phosphorus given in this way are negligible compared with the total phosphorus intake as phosphate. Any effects observed are to be attributed to a specific stimulative action on bone formation. It is now generally believed that elementary phosphorus is valueless in the treatment of rickets, but there is evidence that it is useful in the treatment of certain congenital disturbances of bone growth. (Phemister.)

Sulphur

Small amounts of sulphate are present in the blood and tissue fluids and fairly large amounts are excreted daily in the urine. The sulphate of the blood appears to serve no special function and represents merely a waste product of the metabolism of protein. All complete proteins contain sulphur, the average amount present being one per cent. The chief source of sulphur in the food is protein. There is no indication for the addition of extra sulphates or organically combined sulphur to the diet.

Acid-Base Balance

In the ash, or mineral residue, of both human milk and cow's milk basic constituents are slightly in excess of acid. In most fruits and vegetables the excess of base is more marked than in milk, whereas in cereals, eggs and meats, acid constituents predominate. In the infant's ordinary diet, however, there is not a sufficient excess of either acid or base to be of any especial significance. When a condition of acidosis is present or when it is desired to render the urine alkaline as in the treatment of pyelitis, the administration of fruit juices, especially orange juice, is effective in providing extra base. (For further discussion of acid-base balance, see Chapter XXXII.)

Water Metabolism

The water requirement of the infant in proportion to his body weight is high, being approximately three times that of the adult. The high water requirement of the infant is to be explained on the basis of the more active metabolism. The infant's heat output is greater in proportion to his weight than that of the grown individual, and this heat is removed from the body chiefly by water evaporation. The relatively large food intake requires constant circulation of water from the blood to the intestinal tract and back into the blood during the processes of digestion and absorption. Additional large amounts of water are required for the urinary excretion of waste products, a minimum daily amount of from ¼ to ½ ounce of water per pound of body weight (15 to 30 gm. per kilogram) being necessary for this latter purpose.

Of the total water intake under normal conditions, approximately 50 to 60 per cent is eliminated by the kidneys, 30 to 35 per cent by evaporation from the skin and lungs, and 5 to 10 per cent by way of the bowel. From 1 to 2 per cent of the water taken by the young infant is retained in the body. The amount of water eliminated by various channels is greatly influenced by extraneous conditions. In the presence of high external temperatures, an enormously increased elimination of water from the skin is necessary in order to maintain the normal body temperature. Vigorous exercise also increases markedly the water loss from the skin and lungs. In the presence of severe diarrhea, the water loss from the bowel may equal or exceed the fluid intake. In severe vomiting much of the water taken by mouth may be lost. In any condition associated with an increase in the rate or volume of the respirations, as, for example, pneumonia or acidosis, the water elimination from the respiratory tract is increased.

In the light of the above considerations, it is difficult to state specifically the water requirements of the individual infant since this is subject to very considerable variation. The average normal breast-fed infant under one year of age receives throughout the first year approximately two and one-half ounces of water per pound of body weight per day (150 c.c. per kilogram) from the milk and this amount suffices to meet the needs of most infants and to allow a reasonable excess, except during extremely hot weather or in the presence of diarrhea or vomiting. In the case

of artificial feeding, the protein and salt contents of the diet are usually higher than with breast feeding, and there is consequently an increased elimination of salts and nitrogenous waste products in the urine. This requires a somewhat greater fluid output by the kidney. The extra amount required is, however, but slight, and for practical purposes the artificially fed infant, under average conditions, need be given no more fluid than the one fed at the breast. It is not necessary that the total fluid given be contained in the milk or milk mixture fed. Extra amounts of water may be given in between the feedings. Under special conditions, fluid may be given in the form of dextrose or saline solutions subcutaneously, intraperitoneally, or intravenously. (See Chapter XXXIV.)

An insufficient water intake or excessive loss of water from the body is incompatible with a normal gain in weight. In extreme cases where the water output actually exceeds the intake, there occurs a rapid loss in weight and the development of serious symptoms which may eventuate in death. (See Anhydremia, Chapter XXXIV.)

A reasonable excess of water intake over the actual requirements produces no serious disturbance, but great dilution of the food is undesirable as this necessitates the intake of a very large volume in order that the infant may receive sufficient of the food constituents. Not infrequently the infant's stomach capacity is insufficient to permit the large intake required when dilute formulas are used. In general, it is preferable to prepare formulas which are fairly concentrated and to rely on the administration of water between feedings to supply the necessary fluid. One of the all too common errors in infant feeding is the giving of too dilute formulas, based upon the idea that the individual food elements cannot be digested and absorbed when present in the formula above certain concentrations. For example, there is a prevalent, but erroneous, belief that the percentage of sugar in a formula should not exceed 7 or 8 and that young infants cannot well digest a food containing over 1.5 or 2 per cent of protein. Although many infants may be fed without exceeding these concentrations, there are often conditions in which it is advisable to use more concentrated formulas. There are, of course, limits to which the concentration of a formula may

be carried, but most infants can take and thrive on considerably more concentrated formulas than was at one time supposed.

In general, it is not necessary that the volume of the formula should exceed 10 per cent of the body weight—one and one-half ounces per pound (100 gm. per kilogram). Occasionally, however, in the case of undernourished infants, it is necessary to give 50 per cent more than this amount in order to supply the necessary food constituents even when fairly concentrated formulas are used.

Pigment

Certain complex organic radicles containing the pyrrole group

are necessary to life in both the plant and animal kingdoms. These radicles appear to be intimately associated with processes of oxidation and reduction. The chlorophyl of green plants and the hemoglobin of the blood are the best known examples of compounds containing these organic radicles. These substances are highly pigmented and give the green color to plants and the red color to blood. Closely related substances occur in the yolk of eggs and in vegetable pigments other than chlorophyl. The composition of chlorophyl and of hemoglobin is remarkably similar, each containing in the formula the same organic radicles—the pyrrole rings. In the case of chlorophyl no iron is present. In hemoglobin iron is combined with a pigment group, hematoporphyrin, closely resembling chlorophyl in its chemical structure. In order that hemoglobin may be synthesized in the body, the pyrrole radicles must be supplied. Sources of these are the amino-acids, proline and tryptophane, constituents of most proteins. The amounts present in milk proteins, however, are relatively small, and unless the protein intake of the infant is large, there may not be sufficient amounts of these particular amino-acids present to provide adequately for the building up of hemoglobin during periods of rapid growth. The feeding of chlorophyl in the form of green vegetables, such as spinach, provides a rich source of supply of the necessary constituents of the pigment portion of hemoglobin. Consequently it is desirable in the case of both

breast-fed and artificially fed infants to add green vegetables to the diet at an early age. It is safe and advantageous to begin the feeding of green vegetable purées (especially spinach) at or about the sixth month of life. Under such circumstances the infant is much less likely to be anemic during the latter part of the first year. Liver, red meats and the yolks of eggs are also sources of pigment of the type discussed.

CHAPTER VII

THE VITAMINS

There are at least five vitamins which are essential for the normal nutrition of the infant. The requirements for certain of the vitamins are especially great because of the rapid growth occurring during the first years of life. The vitamins which are known to be essential for the infant are the two fat-soluble vitamins A and D, the antineuritic vitamin B_1 (vitamin F), the growth or antipellagric factor B_2 (vitamin G) and the antiscorbutic, or C vitamin. The E, or reproductive, vitamin has not been shown to be essential in the dietary of the infant, but this latter vitamin possesses certain properties such as a favorable influence on regeneration of blood that may render it a valuable addition to the diet.

The exact mode of action of the vitamins is unknown. There is a question as to whether they stimulate chemical reactions within the body or supply certain necessary cell constituents. Some have compared the vitamins to the lubrication in a machine. The vitamins exert effects in extremely minute quantities, and the exact chemical nature of most of them is as yet unknown, although recently considerable information has been obtained as to the general nature of vitamins A, B and D.

The distribution of the vitamins in those foods which may enter into the infant's dietary is given in Table III.

Vitamin A

Vitamin A is referred to as one of the "fat-soluble" vitamins because of the fact that it occurs in association with the fats of milk, egg yolk and of glandular organs. It is present also in the leafy vegetables, in carrots, and to a somewhat lesser extent in the pericarp of the cereal grains. Relatively small amounts of vitamin A occur in the ordinary subcutaneous fat of animals, and there is little if any in most of the vegetable oils. Vitamin A is probably not synthesized in the animal body, but must be obtained originally from vegetable sources. When so obtained, it is stored in the fat depots, especially in the glandular fats. Liver fat, there-

TABLE III

VITAMINS IN FOODS

	A	B	C	D	E
Milk Products					
Human milk	++	+	+	+	?
Cow's milk					
Raw, whole	++	++	+	+	+
Pasteurized, whole	++	++	±	+	+
Boiled, whole	++	++	±	+	+
Cream	+++	+	+	++	+
Skimmed	0	++	+	0	0
Dried, whole	++	++	±	+	+
Dried, part skimmed	+	+	±	±	±
Evaporated	++	++	0	+	+
Casein powder	0	0	0	0	?
Butter	+++	0	0	+++	++
Carbohydrates					
Cane sugar	0	0	0	0	0
Milk sugar	0	0	0	0	0
Karo syrup	0	0	0	0	0
Dextri-maltose	0	0	0	0	0
Vitavose	0	++++	0	0	0
Malt extract	0	++	0	0	0
Starch	0	0	0	0	0
Cereals, whole	0	++	0	0	±
Cereals, milled	0	0	0	0	0
Cereals, irradiated	0	0	0	+	0
Vegetables					
Beans, string	++	++	++	±	
Cabbage	+	++	+++	?	
Carrots	++++	++	±	?	
Lettuce	++	++	++	+	++
Onions	±	+	+++	?	
Peas, green	++	+++	±	?	
Potatoes, white	±	+	+	?	
Potatoes, sweet	++	+	+	?	
Spinach	++++	+++	++	+	
Tomatoes	++	++	++++	0	
Fruits					
Apples	±	++	+	0	
Bananas	++	++	++	0	
Grapes	±	++	+	0	
Lemons	±	++	++++	0	
Oranges	±	++	++++	0	
Meats					
Beef	+	++	?	?	
Lamb	+	++	?	?	
Pork	±	++	?	?	
Liver	++	+	?	+	
Eggs					
White	0	0	0	0	
Yolk	+++	+++	0	+++	
Oils					
Cod liver oil	++++	0	0	++++	0
Cottonseed oil	?	0	0	0	+
Olive oil	?	0	0	0	+
Maize oil	+	0	0	?	++
Cocoanut oil	?	0	0	+	+
Yeast					
Fresh	0	+++	0	0	
Extract	0	++++	0	0	
Irradiated	0	+++	0	+	

fore, contains the vitamin, provided the animal has received it in the diet. Subcutaneous fat contains much smaller quantities, especially in the case of animals, such as hogs, which have been fattened up by high carbohydrate feeding. The fat of cattle given green feeding contains larger amounts of vitamin A.

An especially rich source of vitamin A is cod liver oil. The original source of this vitamin in cod liver oil is marine vegetation, which is consumed by small fish and crustaceans, which are, in turn, consumed by the codfish. There is a great variation in the amounts of the vitamin present in various fish oils depending presumably upon the feeding habits of the fish.

In cod liver oil and in other natural fats, vitamin A is associated with the nonsaponifiable fraction. When cod liver oil is treated with alkali so as to convert all of the neutral fats into soaps, an insoluble residue consisting of less than one-half of one per cent of the total remains, which consists of sterols, substances having some of the physical properties of fats, but which are in reality higher alcohols. All of the vitamin A of the original oil is contained in this very small fraction, and of this vitamin A constitutes only a small portion.

Vitamin A is fairly stable to heat and is but little, if any, affected by ordinary cooking or canning processes, especially if free access of air is avoided. Although vitamin A appears to be produced in nature under the influence of sunlight, an excess of sunlight or exposure to ultraviolet light results in a partial destruction of the vitamin. Irradiation of cod liver oil or of foods leads only to a decrease in the vitamin A content.

When vitamin A is deficient in the diet or is completely withdrawn, the effects are not noted immediately, because of the fact that the body possesses stores of this vitamin in the fatty depots. After a period of time, which may be a matter of weeks, the effects become manifest. The first effect noted in growing individuals is a decrease in the rate of growth or a complete cessation of weight gain. Changes in the superficial mucous membranes occur which render these membranes susceptible to infection. The conjunctiva become keratinized and a condition known as xerophthalmia develops. This is accompanied by a nonspecific conjunctivitis. Later there may be associated keratomalacia and staphylomata with resulting loss of vision (Fig. 3).

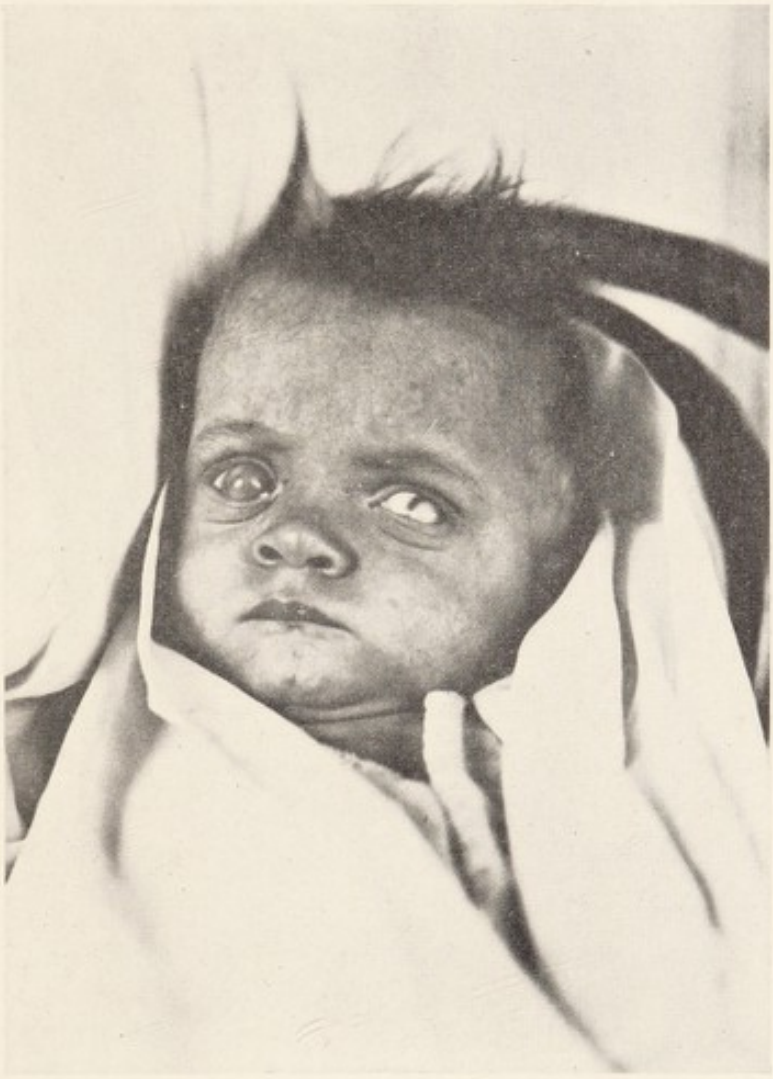


Fig. 3.—Xerophthalmia, the result of a diet deficient in vitamin A.

Due to the mucous membrane changes there is an increased liability to infections in the rhinopharynx, sinuses, lungs and gastrointestinal tract. It has been stated that a deficiency of vitamin A in the diet during infancy may result in a decreased resistance of the mucous membranes to infection for a number of years following.

Infants fed at the breast may or may not receive an adequate amount of vitamin A, depending upon whether or not the mother's diet is adequate. If the mother's diet contains an abundance of green vegetables, butter, and eggs, the vitamin A content of the milk may be entirely adequate for the infant's needs; otherwise it becomes necessary to supplement the breast feeding by the administration of cod liver oil.

Cow's milk, like human milk, is somewhat variable in its vitamin A content. In general, however, there is about as much vitamin A in cow's milk as in human milk. In both forms of milk, the entire vitamin A content is in the fatty portion or cream of the milk. There is some evidence that vitamin A is better absorbed from human milk than from cow's milk. An excess of poorly digestible fat in the diet or the administration of mineral oil results in a loss of this vitamin by way of the bowel. Artificially fed infants receiving partially skimmed cow's milk, or cow's milk so greatly diluted that the total volume of whole milk taken is small, may suffer from deficiency of this vitamin.

Vitamin D

Vitamin D usually occurs in nature in association with vitamin A, but not in fixed proportions; thus cod liver oil contains relatively more vitamin D in proportion to A than does butter. Vitamin D, like vitamin A, is associated with the nonsaponifiable fraction of natural fats. The chemical nature of vitamin D seems to have been fairly definitely established as being a sterol closely related to cholesterol and known as "ergosterol." This particular sterol occurs in cod liver oil, in egg yolk, in the leaves of green vegetables, and in the fat of milk. Small amounts are present in the skin. The richest sources of ergosterol appear to be yeast and ergot. Pure ergosterol obtained from these latter sources, however, does not have the characteristic physiologic action of vitamin D until it is exposed to ultraviolet radiation either from sunlight or from artificial sources. The degree of activity depends

upon the conditions of irradiation. Preparations have been made having one million times the vitamin D potency of standard cod liver oil.

Irradiated ergosterol is now prepared commercially and marketed in dilute oily solution (viosterol 100-D) which is standardized to have a vitamin D potency one hundred times that of U.S.P. standard cod liver oil. These preparations of irradiated ergosterol, however, contain no vitamin A and are, therefore, only partial substitutes for cod liver oil. This deficiency may, to some extent, be overcome by the use of cod liver oil fortified with irradiated ergosterol. (Viosterol 5-D.) There is a distinct danger that the present enthusiasm for irradiated ergosterol may result in the widespread substitution of this for cod liver oil. This would be most unfortunate as the vitamin A is as important for the infant as vitamin D. As many natural foods contain some ergosterol, irradiation of these results in imparting to them vitamin D potency. Irradiated milk and cereals are on the market. The actual vitamin D potency of irradiated cereals does not appear to be very great; certainly not sufficient to render them substitutes for cod liver oil.

Irradiation of the body surface leads to the same results as the feeding of vitamin D. It has been suggested that this is the result of activation of ergosterol present in the skin.

Vitamin D is very resistant to heat and oxidation and is not affected by the usual processes of cooking, drying, canning or preserving of food. The normal effect of vitamin D is to promote the deposition of calcium phosphate in developing bones and teeth. A deficiency of this vitamin in the diet is one of the possible causes of rickets. The administration of vitamin D results in prompt healing of active rickets, provided the diet is adequate in other particulars. Rickets, or a closely related condition, may be produced experimentally by the feeding of diets deficient in mineral constituents even when vitamin D is present. This phase of the subject is discussed more fully in the chapter on Rickets. (Chapter XXIX.)

An excess of vitamin D in the diet may result in abnormal calcification throughout the body. Very large doses are, however, required to bring about this harmful effect. Untoward symptoms consisting of lack of appetite, drowsiness and an apparent toxemia have been noted to occur following moderate overdosage.

There is some evidence that vitamin D may promote healing of fractures, and its use has been suggested in the treatment of tuberculosis of the bones and of certain other tuberculous lesions to promote calcification. There is evidence that vitamin D is effective in preventing dental caries. Vitamin D administration results in an increase in blood platelets but does not appear to be effective in controlling the bleeding in purpura.

Vitamin D, like vitamin A, occurs in variable amounts in both human and cow's milk, depending upon the diet of the mother or of the cow. There is, on the average, as much in cow's milk as in human milk, but none too much for the needs of the infant in either, especially during the winter months. It is, therefore, advisable to supplement the diet of all infants, whether breast fed or artificially fed, with additional vitamin D in the form of cod liver oil or irradiated ergosterol. A suitable dosage of cod liver oil is one-half teaspoonful three times a day during the first four months of life and one to one and a half teaspoonfuls three times a day after four months. Large or rapidly growing infants require more than smaller ones who are growing slowly. Preparations of the unsaponifiable residue of cod liver oil containing both vitamin A and vitamin D may be used. Acceptable preparations are "Oscodal" and "Cod-liv-X." Some alleged cod liver oil concentrates are of very low vitamin potency.

Vitamin B

This vitamin was originally supposed to be a single factor, but has recently been separated into two factors "B_1" and "B_2" or vitamins F and G, respectively. B_1 is sometimes referred to as the antineuritic factor and B_2 as the growth factor, or occasionally as the antipellagric factor; these latter designations are unfortunate, since B_2 is not the only growth factor, and deficiency of this vitamin is apparently only one of the causes of pellagra. Vitamin B_1 is partially destroyed when heated to high temperatures in the presence of alkali, whereas vitamin B_2 is remarkably heat-stable and is not affected by aging or drying. The B vitamins usually occur together and until recently no differentiation has been made. Most of the experimental work deals with the effects of the combined vitamins. We shall, therefore, consider them as one in this discussion under the single term of "vitamin B." The chemical

nature of vitamin B is not definitely known, although it is believed to be a nitrogen-containing ring compound.

Vitamin B is widely distributed in nature. It is present in both human and cow's milk, but is considerably more abundant in cow's milk. The vitamin is soluble in water so that it occurs in the whey of the milk and not in association with the fat. The amount present in human milk appears to be sufficient for the needs of the average infant and as there is considerably more of this vitamin in cow's milk, symptoms of deficiency are not to be expected in the case of artificially fed infants, except under unusual circumstances when very small amounts of cow's milk are given. Vitamin B occurs in very large amounts in the germs of all cereals, but is not present in milled cereals. It is present in most vegetables and fruits. A particularly rich source of vitamin B is yeast. Vitamin B is not stored in the body in any appreciable amount, so that when withdrawn from the diet, some effect is almost immediately observed. The effects of deficiency of vitamin B are lack of appetite, slow growth followed by decline in weight, lack of tone of skeletal and intestinal musculature and ultimately a condition of polyneuritis (beriberi). The intestinal atony which may involve the entire gastrointestinal tract, results in lack of gastric motility and also in atonic constipation. Marked colonic dilation simulating that of Hirschsprung's disease has been observed. According to Goldberger, deficiency of vitamin B is also a factor in the causation of pellagra. There is some evidence that deficiency of this vitamin in the diet of the pregnant mother is one of the factors responsible for a hemorrhagic diathesis in the newly born.

In animals fed on vitamin B deficient diets, ulcers resembling decubital sores have been observed. Vitamin B deficiency in animals results in disturbance of the carbohydrate metabolism leading to hypoglycemia. Another symptom of vitamin B deficiency is anhydremia. It has been suggested that deficiency of this vitamin may result in disturbance of function of some of the endocrine glands.

It has been claimed in the case of certain very undernourished infants suffering from loss of appetite that the addition of extra amounts of vitamin B in the form of yeast extract or preparations of cereal germ results in improvement in the appetite and gain in weight. In our experience it has been possible to supply sufficient

vitamin B in the form of milk without resorting to additional sources of supply and we have rarely, if ever, observed beneficial effects which could be attributed to the administration of extra vitamin B.

Vitamin C

Vitamin C, or the antiscorbutic factor, is widely distributed in nature, occurring in especially high concentration in the citrous fruits, and in tomatoes, onions and cabbage. Apples, bananas, grapes, string beans, and turnips contain the vitamin, but in smaller amounts. Vitamin C is present in both human milk and cow's milk; the quantity, however, is subject to wide variations. Vitamin C can apparently not be synthesized in the animal body. When a mother subsists on a diet containing a liberal allowance of vitamin C containing foods, her milk contains much more vitamin C than when the diet is deficient in those foods. The milk of cows fed on green food contains more vitamin C than that of stall-fed cows.

Vitamin C is the most unstable of the known vitamins, being rapidly destroyed by heating in neutral or alkaline solution, by drying or aging. In acid solution, vitamin C is more stable and may resist a considerable degree of heat; for this reason cooked or canned tomatoes, have almost as great a content of vitamin C as raw tomatoes or orange juice. Heated lemon or orange juice is likewise a rich source of vitamin C. The vitamin is less likely to be destroyed by heat if air is excluded; hence some canned vegetables contain more of the vitamin than those which are home cooked. The exact chemical nature of vitamin C is unknown.

When the amount of vitamin C in the diet of the infant is below the optimum requirements, growth is retarded, the infant becomes irritable, lacks energy, and shows diminished resistance to infection. These symptoms may not be observed until the infant has been fed the deficient diet for a considerable time, inasmuch as some storage of vitamin C in the body seems to occur. One of the remote effects of vitamin C deficiency is a change in the structure of the teeth. (See Chapter XXXIII.)

The long-continued feeding of diets markedly deficient in vitamin C results in development of the acute symptoms of scurvy. This condition is discussed in detail elsewhere (Chapter XXXI). The breast-fed infant obtains sufficient vitamin C for his needs,

provided the mother's diet is one containing reasonable amounts of fruits and green vegetables, but not otherwise. Scurvy in exclusively breast-fed infants occurs but rarely. Artificially fed infants may or may not receive sufficient vitamin C. Cow's milk contains a variable amount of this vitamin when fresh and raw. When the milk is pasteurized or boiled, a portion of the vitamin is destroyed. Larger amounts are destroyed when the milk is autoclaved, as in the process of canning. Some processes for the drying of milk destroy a large proportion of vitamin C; other processes, a smaller proportion. Inasmuch as raw cow's milk is not to be advised for the feeding of infants, and as any form of heat-treated milk may be deficient in the antiscorbutic vitamin, every artificially fed infant should be given an extra amount of vitamin C. In practice, orange juice is most frequently selected as the source of antiscorbutic vitamin, one to two tablespoonfuls a day being sufficient for the average artificially fed infant. Canned tomato juice is an equally valuable source of vitamin C and may be substituted for orange juice as a measure of economy.

Vitamin E

Vitamin E, sometimes known as "vitamin X," or the fertility factor, is found in wheat and maize oils, and to a lesser extent in olive and cottonseed oils and in butter fat. It is present in such leafy vegetables as lettuce. There is little, if any, in cod liver oil. Besides the function of this vitamin in the processes of reproduction, it seems to have some influence on the metabolism of iron and to promote blood regeneration after anemia. The importance of this vitamin in the diet of the infant has not yet been appraised.

It is likely that vitamins other than those now known will ultimately be discovered. Most of our knowledge concerning these elusive but important food substances has come during the last ten or fifteen years and the field is as yet incompletely explored. The whole subject of vitamins has assumed immense importance in the popular mind. Manufacturers of proprietary products have made capital of this recent knowledge, and a great many half truths have been stated and restated. A favorite method of exploiting certain foods or vitamin preparations is to present pictures of animals undernourished and sickly side by side with pictures of healthy animals, the latter having received a certain

amount of the food or vitamin preparation in question, the conclusion being drawn that a small amount of the particular vitamin supplied in a certain preparation will lead to growth and normal health. The fallacy lies in the fact that the control animals have been fed on highly purified synthetic diets *entirely* lacking in the vitamin and not on average normal diets.

CHAPTER VIII

SUMMARY OF THE NUTRITIONAL REQUIREMENTS OF INFANTS

For ready reference, the nutritional requirements of the infant as detailed in the preceding six chapters are here briefly summarized.

Calories

During the first year of life the normal infant should receive an average of 50 to 55 calories per pound of body weight per day (110 to 115 calories per kilogram). The caloric requirement is somewhat greater than the above during the first three months of life and somewhat less after the sixth month. The caloric requirement of the undernourished or overnourished infant should be calculated on the basis of the *expected* weight for the age. In the case of the normal breast-fed infant, the caloric requirement is met if the infant receives approximately 2.5 to 3 ounces of milk per pound of body weight per day (150 to 180 c.c. per kilogram). In the case of the artificially fed infant, approximately two-thirds of the total caloric requirement should be met by milk and one-third by added carbohydrate.

Proteins

The protein requirements of the normal infant are adequately met when he receives approximately 2.5 ounces of breast milk per pound of body weight (150 c.c. per kilogram), or 1.5 ounces of cow's milk per pound of body weight per day (100 c.c. per kilogram). This corresponds to approximately 2 grams of protein per kilogram in the case of the breast-fed infant and 3.5 grams per kilogram in the case of the artificially fed infant. A moderate excess of protein does no harm. Undernourished infants should receive an amount of protein proportionate to the expected rather than the actual body weight.

Carbohydrates

An infant should receive not less than one per cent of his body weight in carbohydrate per day (0.15 ounces per pound) (10 grams per kilogram). The breast-fed infant receives this amount in the

milk. In the case of the artificially fed infant, approximately one-third of the carbohydrate should be derived from the milk and the remainder added in the form of sugar or starch. For most infants the proportion of carbohydrate added to cow's milk in the diet should be approximately one part of carbohydrate for each 10 to 15 parts of milk. Carbohydrates of the dextrin and maltose types are to be preferred during early infancy. Cane sugar may, however, be used. After the sixth month of age, a portion of the carbohydrate should be given in the form of starch.

Fats

Fats are not absolutely essential to nutrition, but are desirable. The breast-fed baby receives sufficient fat when he gets enough milk to meet the caloric requirements. The artificially fed baby, taking sufficient whole cow's milk to meet the protein requirements, also receives sufficient fat. Milk from Jersey or Guernsey cows, containing a high percentage of fat, should not ordinarily be used for the feeding of infants, nor should top milk or cream be used for the preparation of the usual formulas. Skimmed milk is not indicated except in the presence of gastrointestinal disorders.

Mineral Salts

The breast-fed infant, receiving 2.5 ounces of milk per pound of body weight, or the artificially fed infant receiving 1.5 ounces of cow's milk per pound of body weight per day, obtains sufficient mineral constituents with the possible exception of iron.

Water

The infant's requirement for water is variable and depends upon activity, outside temperature, and the presence of diarrhea or vomiting. The daily requirement for the normal infant varies from 10 to 15 per cent of the actual body weight (1.5 ounces to 2.5 ounces per pound). Breast-fed infants usually receive sufficient water in the milk. Artificially fed infants, given undiluted formulas, should be offered additional water between feedings, especially in warm weather.

Pigment

Pigment for the formation of hemoglobin is a desirable addition to the diet after the sixth month of life. One tablespoonful of

purée of spinach once a day supplies sufficient pigment as well as iron for the average infant's needs.

Vitamins

The fat soluble vitamins A and D should be added to the diet of all artificially fed infants and most of those who are breast fed. From one-half to one and one-half teaspoonfuls of standardized cod liver oil three times a day supplies sufficient of these two vitamins. Vitamin D alone may be supplied by the administration of 2 to 5 drops of viosterol 100-D three times a day. Cod liver oil is, however, distinctly preferable.

Vitamin C should be given to all artificially fed babies. From one to two tablespoonfuls of orange juice or tomato juice daily is sufficient to supply this vitamin.

There is sufficient vitamin B in either human milk or cow's milk to meet the needs of the average infant provided the amounts of milk are adequate to meet the other nutritional requirements.

CHAPTER IX

DIGESTION IN INFANCY

The food of the infant is of necessity simple, consisting chiefly of milk and carbohydrate. The infant's digestive organs are well adapted to care for human milk and for somewhat lesser amounts of cow's milk. The infant's capacity for the digestion of certain other foods is distinctly limited and is only developed as the infant grows older. On the other hand, the digestive capacity of the infant is not so limited as we might be led to suppose. For example, if an adult were to take as much cow's milk in proportion to his weight as a normal infant, he would be drinking from 8 to 10 quarts of milk a day and would, in addition, be eating from 1.5 to 2 pounds of sugar.

On account of the proportionately greater food requirements of the infant, the digestive functions are taxed closer to the limits of capacity than in the case of the adult, and there is a smaller margin of safety. Furthermore, the digestive functions of the infant are especially likely to become impaired as the result of such influences as malnutrition, infection, fever, high external temperatures or pain. Not infrequently the infant's digestive capacity is lowered to such an extent that the optimum amounts of food required for nutrition are not capable of utilization.

Salivary Digestion

Salivary digestion of food is relatively unimportant during early infancy, as the saliva has no effect upon the constituents of milk. When starch is fed, slight digestion may be accomplished by the enzyme ptyalin, and such digestion continues for a short period after the food reaches the stomach. Salivary digestion assumes relatively little importance until the time the infant begins to chew starchy foods, such as toast, crackers or cereals.

Gastric Digestion

The gastric glands are functionally active at the time of birth and capable of secreting hydrochloric acid and the pepsin-rennin ferment. The concentration of acid secreted by the infant is, how-

ever, much less than that of the adult. There is a gradual increase in the strength of acid secreted throughout infancy and childhood. Considerable individual variation exists, some very young infants having a fairly concentrated gastric juice.

The total volume of the gastric secretion, as well as its acid content, is influenced by a variety of factors. The amount and concentration is greatly decreased in the presence of infections accompanied by fever. It is decreased in excessively hot weather and also as the result of pain or fright. The gastric secretion of undernourished infants is, in most instances, less than that of normal infants of the same age. As the result of pharyngeal, esophageal or gastric irritation, much alkaline mucus is secreted and this, when mixed with the gastric contents, partially neutralizes the acid and renders the gastric juice relatively inefficient. Gastric secretion is increased by the feeding of certain types of food, notably those with a high protein content. Some of the amino-acids, especially beta-alanin, have a marked stimulating effect on gastric secretion. Commercial beef extract contains a considerable amount of beta-alanin and other substances capable of stimulating gastric secretion. Acidified foods have a similar effect. Psychic factors also play an important rôle in influencing gastric secretion.

Of the two constituents of the gastric juice, hydrochloric acid and the pepsin-rennin ferment, the acid appears to perform the more important functions during infancy. In the case of the normal breast-fed infant, the amount of hydrochloric acid secreted is sufficient to render the stomach contents distinctly acid at the height of digestion. The average degree of acidity attained corresponds to a hydrogen-ion concentration of pH 3.6. This degree of acidity is sufficient to inhibit markedly the growth of the majority of the bacteria likely to be introduced into the stomach. The acidity of the gastric contents is also one of the factors in regulating the pyloric reflex. On the passage of the stomach contents into the duodenum, the acid present under normal conditions is sufficient when coming in contact with the duodenal mucosa, to bring about the production of "secretin," a hormone which is carried to the liver and pancreas by way of the blood and stimulates the flow of bile and pancreatic juice.

The amount of acid normally present in the stomach of the breast-fed infant is sufficient to activate the pepsin present so that

some peptic digestion is possible; the digestion of the proteins of milk, however, is very incomplete in the stomach, even in the case of breast-fed infants. Small amounts of both casein and lactalbumin are converted into albumoses and peptones, but the greater part of the digestion of protein is completed in the small intestine.

Coagulation of casein by rennin is usually fairly complete in the stomach. The casein curd from human milk is very small and fine, whereas the curd from raw or pasteurized cow's milk is very much larger and tougher and may be a single, large, jelly-like curd, almost completely filling the stomach. Large curds entangle in their meshes such bacteria as may be present and remove them from the antiseptic action of the gastric juice. These large tough curds may be somewhat broken up by the gastric contractions, but even then they cannot pass through the pylorus as readily as the finer curds of human milk. Boiling of cow's milk so changes the character of the casein that it is precipitated in smaller curds in the stomach. Heating cow's milk to a still higher temperature, as is done in the preparation of unsweetened evaporated milk, still further alters the character of the casein so that the curds are very fine and soft resembling closely those of human milk. Drying of milk produces an effect intermediate between boiling and evaporation. When cow's milk is acidified previous to feeding, the casein curds are precipitated in finely divided form and very little if any further change is effected through the action of the rennin of the stomach. Addition of alkalies or of sodium citrate to milk before feeding or dilution of the milk with water or especially with starch gruels, results in the formation of smaller curds in the stomach. The size of the curds produced in the stomach is an important factor in the digestibility of milk mixtures.

Mention has been made of the fact that the stomach contents of the normal infant receiving human milk are distinctly acid. When equivalent amounts of undiluted sweet cow's milk are fed, the stomach contents do not reach the same degree of acidity, the average concentration of acid at the height of digestion not exceeding a pH of 5.3. This degree of acidity is not sufficient for peptic digestion nor is it sufficient to exert very much inhibitory action on the growth of bacteria. The acidity of the stomach contents passing into the duodenum is also below the optimum for secretin

formation. If the feeding of undiluted sweet cow's milk is continued, there occurs a gradual increase in the concentration of acid secreted. Some normal infants secrete such a concentrated gastric juice that even when they are fed undiluted sweet cow's milk the gastric acidity may approximate that of normal breast-fed infants.

The difference in behavior between cow's milk and human milk in the stomach is explained by the higher "buffer" content of cow's milk. "Buffers" are substances capable of resisting a change in reaction of the medium when either acids or alkalies

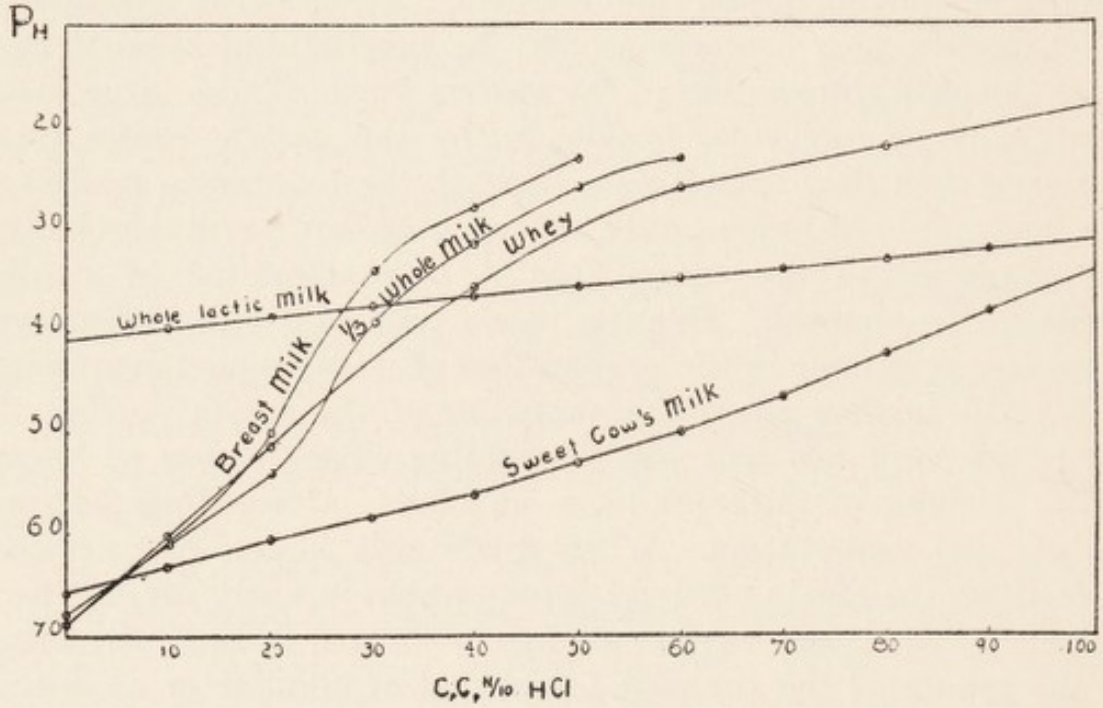


Fig. 4.—Titration curves of breast milk, sweet cow's milk, lactic acid milk, cow's milk diluted one-third and whey. These curves show the hydrogen-ion concentration resulting when varying amounts of hydrochloric acid are added to milk. The figures for hydrogen-ion concentration in the column marked pH represent the negative exponents of hydrogen-ion concentration. A pH of 7.0 indicates neutrality; figures below this, varying degrees of acidity. The lower the pH the *greater* the degree of acidity.

are added. Cow's milk contains approximately three times the amount of buffer substance as human milk, so that three times as much acid must be added to a given amount of cow's milk to bring it to the optimum acidity for gastric digestion. This difference in the two milks is shown in the titration curves. (See Fig. 4.) These curves show the gradual increase in acidity as varying amounts of diluted hydrochloric acid are added. When one part of cow's milk is diluted with two parts of water, the buffer value of the dilute milk is, of course, only one-third that

of the concentrated milk and such dilute milk shows a titration curve approximating that of human milk. When one-third diluted cow's milk is fed, the gastric contents at the height of digestion average the same as in the case of human milk. Dilution, therefore, is one of the means of rendering cow's milk more digestible. There are, however, better means of accomplishing the same purpose, for example, acidification or heat treatment. These methods are discussed in a subsequent chapter.

No appreciable digestion of fat occurs in the stomach, although a small amount of lipase is occasionally present. Carbohydrates are not acted upon by the gastric juice except for possible slight conversion of disaccharides to monosaccharides at the temperature and acid reaction of the stomach. The ptyalin of swallowed saliva may continue to act in the stomach for a short time on any starch present in the food.

Very little absorption takes place from the stomach and that mostly from the pyloric end. A small amount of dextrose, amino-acids or salts may be absorbed, but very little if any of other substances. Water is probably not absorbed except in cases of extreme dehydration.

The stomach contents begin to empty through the pylorus during the time that feedings are being taken. The pylorus opens and closes intermittently, allowing but small quantities to pass through at one time. The stomach thus acts as a protective mechanism, tending to prevent the flooding of the intestinal tract with food in excess of the digestive capacity. The length of time required for the complete emptying of the stomach is variable, and dependent upon the size of the meal, the character of the feeding and the tonicity of the pyloric and gastric musculature. In general, the stomach empties more quickly in the case of breast-fed infants, being empty usually by the end of two hours. In artificially fed infants, the emptying time may be prolonged to three hours or more. Dilute milk mixtures are emptied more rapidly than concentrated ones and small feedings more rapidly than large. Cow's milk which has been treated so that the curds are very small leaves the stomach more quickly than milk which forms large curds. The more liquid portions of the stomach contents are the first to pass through the pylorus. Sugars leave the stomach before proteins and fats. An excess of fat in the food delays the emptying time of the stomach. In the presence of

fever, gastric motility is sluggish; it is also sluggish in undernourished infants. When any degree of pylorospasm or pyloric stenosis exists, the emptying time of the stomach is prolonged. If the infant is hungry at the time of the feeding, gastric motility is more active than if the infant is fed at such short intervals that the pangs of hunger are not present. The stomach empties more rapidly if the infant lies on the right side or is held in a semi-erect position.

Intestinal Digestion

When the chyme passes through the pylorus into the duodenum, it remains for a short time before passing along toward the jejunum. While in the duodenum, the food becomes mixed with pancreatic juice, bile and some succus entericus.

The pancreatic juice is strongly alkaline in reaction and contains the enzymes trypsinogen, amylopsin (pancreatic diastase) and lipase (steapsin) capable of acting on proteins, carbohydrates and fats, respectively.

The bile is neutral or slightly alkaline and contains besides pigments, cholesterol and lecithin, and the salts of the bile acids (glycocholic and taurocholic) which latter are effective in promoting the emulsification of fats. The bile also contains a substance which accelerates the action of pancreatic lipase. Bile is capable of dissolving fatty acids and to a certain extent the soaps of calcium and magnesium which are ordinarily insoluble in water.

The succus entericus is alkaline in reaction and is secreted by the crypts throughout the small intestine. It contains the enzyme enterokinase, capable of activating the trypsinogen of the pancreatic juice to trypsin, a proteolytic enzyme erepsin, and an enzyme or group of enzymes capable of converting the disaccharides lactose, sucrose and maltose into monosaccharides.

The combined secretions of the duodenum although alkaline in reaction do not normally neutralize completely the acid chyme coming from the stomach so that the duodenal contents as well as the contents of the upper part of the jejunum are weakly acid in reaction. When gastric secretion is decreased or when foods of a high buffer content are fed (such as undiluted sweet milk) the duodenal contents may be alkaline. When the duodenal contents are alkaline, bacterial flora are likely to be present in large numbers.

Protein digestion, which may occur to a slight extent in the stomach, is continued in the duodenum through the action of the enzyme trypsin of the pancreatic juice, which breaks the protein molecule into smaller remnants, the peptones, polypeptides and amino-acids. Protein digestion is further continued by the action of erepsin which converts to amino-acids those remnants of the protein molecule which have escaped tryptic digestion. It is only in the form of amino-acids that protein is absorbed under normal conditions.

Protein digestion and absorption during infancy is remarkably complete, even in the presence of diarrhea and in the case of sick or poorly nourished infants, only an insignificant amount of protein or of protein derivatives appearing in the stools. Such protein as is present in the stools represents chiefly secretion from the intestinal mucosa together with bacterial proteins.

The simple carbohydrates, dextrose, levulose and galactose require no digestion and are rapidly absorbed by the small intestine. The disaccharides, maltose, sucrose and lactose, are converted into the monosaccharides through the action of enzymes secreted by the small intestine and are absorbed in the form of monosaccharides. When a large amount of carbohydrate is fed or when absorption is impaired, some sugar may reach the large intestine and here be attacked by the bacteria present. Sugar itself rarely appears in the stools, it being decomposed to form acids and gases.

Starch digestion is effected by the enzyme amylopsin of the pancreatic juice. Amylopsin has the same action as the ptyalin of the saliva or the diastase of germinated grain. It converts starch first to dextrin and then to maltose. Amylopsin is present in the pancreatic juice of the young infant only in small quantities. When starch is fed, however, the secretion of amylopsin appears to be stimulated so that even a young infant accustomed to the feeding of starch may digest a considerable amount. Relatively little carbohydrate is normally destroyed through bacterial action in the intestinal tract. If, however, the digestive and absorptive capacity of the gastrointestinal tract is impaired, bacterial destruction of a large portion of the carbohydrate may occur. The products of bacterial action on carbohydrate are chiefly organic acids, some of which have a distinctly irritant effect on the intestinal mucosa. Excessive fermentation of carbohydrate results in impaired absorption not only of carbohydrates but also of other food elements.

The digestion of fats begins in the duodenum, the first step being emulsification or the breaking up into very fine globules. This process is aided by the presence of bile salts and by the churning action in the duodenum. Milk fat is already in a fine emulsion, but other fats require emulsification before much digestion is possible. The next step in the digestion of fats is saponification, which is accomplished by the enzyme lipase (steapsin) of the pancreatic juice. The activity of this enzyme is enhanced by the presence of bile. Through the action of lipase, the emulsified fat is converted into fatty acids and glycerin and absorbed as such. When the duodenal contents are alkaline or when the partially digested fat reaches the lower portions of the small intestine where the reaction of the contents is alkaline, soaps are formed by the combination of fatty acids with the bases present. Some calcium soaps are also formed, especially when the food contains a good deal of casein (calcium caseinate). These calcium soaps are partially dissolved by the bile and absorbed, the remainder pass through the intestinal tract and appear in the stools as curds or as solid, putty-like masses.

The fat of the food, whether from human or cow's milk, is almost completely saponified under normal conditions. Absorption, however, is more complete in the case of human milk fat. In the presence of diarrhea, the intestinal contents may be hurried through so rapidly that some fats escape saponification, appearing in the stools as neutral fat. When the flow of bile into the intestinal tract is obstructed, saponification and absorption of fats is not complete and large amounts of soaps and some neutral fat pass through the intestinal tract unutilized. In the condition of celiac disease (see Chapter XXI) saponification of fats is fairly complete, but absorption is extremely poor, and a large portion of the ingested fat may reappear in the stools in the form of soaps.

In general the capacity of infants to digest and absorb fats is more limited than for either proteins or carbohydrates. Even a slight digestive disturbance is likely to result in diminished absorption of fat. Unabsorbed fatty acids, particularly the lower members of the series, are capable of causing gastrointestinal irritation.

The large intestine secretes an alkaline juice, but apparently no digestive enzymes. Any digestion that occurs in the large in-

testine is merely a continuation of digestive processes in such mixtures of food residues and enzymes as have previously escaped absorption.

In the large intestine bacterial activity is usually vigorous and results in the decomposition of certain food remnants, especially carbohydrates. Under normal conditions more absorption of water probably occurs from the large than from the small intestine. The contents of the small intestine are normally liquid; those of the lower portion of the large intestine, semisolid. Very little absorption of food materials occurs from the large bowel.

Bacteriology of the Gastrointestinal Tract

At birth the gastrointestinal tract is sterile, but it does not remain so for long. The bacterial flora differ a great deal in the different portions of the intestinal tract, and the number and character of the organisms present are influenced profoundly by the character of the diet, the nature and amount of gastrointestinal secretions, and by the presence of fever. Almost any organism present in the air, in water, or in milk may at times be found in the gastrointestinal tract. The most important and most frequently found organisms are the B. bifidus, B. lactis aërogenes, B. acidophilus, B. coli and Mic. ovalis. Occasional inhabitants of the intestinal tract are the B. aërogenes capsulatus (gas bacillus, or Welch bacillus) staphylococci and streptococci. Certain other organisms, such as the members of the dysentery group, typhoid, parathyroid, tubercle bacillus and the bacilli of Flügge are occasional invaders of the intestinal tract and give rise to disease.

Under normal conditions, considerable inhibition of bacterial growth is brought about through the acidity of the gastric juice, so that the food entering the small intestine is relatively free from bacteria. The gastric acidity is sufficient to inhibit the growth of B. coli, the hemolytic staphylococcus, enterococci and many other organisms. Most spore bearers and anaerobic organisms are but little affected by the gastric juice. The upper portion of the small intestine usually contains relatively few organisms, those present being chiefly enterococci and a few members of the lactis aërogenes group. When the gastric secretion is decreased, however, as the result of fever, high external temperatures or is neutralized by the giving of an excess of alkali or of large amounts of sweet cow's milk with its high buffer value, the upper portion

of the intestinal tract may contain large numbers of bacteria. Colon bacilli are often found in the stomach and duodenum in the presence of severe gastrointestinal disturbances with diarrhea. Whether these organisms have ascended from the colon as the result of gastrointestinal disturbance or whether they have been introduced in the food and having found suitable conditions for growth have by their action led to the diarrhea is an unsettled question. It is our own belief that this invasion of the upper intestinal tract with colon bacilli and related organisms is an important factor in the causation of diarrhea.

Growth of microorganisms in the upper intestinal tract is favored by an excess of food which furnishes a suitable culture medium. It is also favored by damage to the intestinal mucosa or by decrease in the normal digestive secretions. The type of organisms which predominate is determined, to a large extent, by the character of the food. Some bacteria thrive in a medium rich in carbohydrate; others grow better in the presence of protein.

In the lower portion of the small intestine the bacterial flora are more numerous, the B. bifidus, Mic. ovalis, B. coli, B. acidophilus and B. lactis aërogenes predominating.

In the large intestine enormous numbers of bacteria are normally present. In the case of breast-fed infants, the predominating organism present in the lower bowel is the B. bifidus, a gram-positive acid-producing organism. A smaller number of B. coli and B. acidophilus are found and occasionally the B. aërogenes capsulatus. The Mic. ovalis may be present in large numbers. In the colon of the artificially fed infant the flora is more complex, gram-negative bacilli of the colon and lactis aërogenes group predominating. The feeding of considerable amounts of lactose together with relatively small amounts of protein, however, tends to cause an increase in the numbers of B. bifidus present. In the large intestine such carbohydrates as have escaped digestion are fermented and converted into gases and organic acids. Protein remnants are also broken down by bacterial action to form ammonia, amines, indol, skatol and a variety of other products.

The growth of bacteria in the intestinal tract may either aid or interfere with the processes of digestion. The growth of such organisms as the B. acidophilus and other harmless lactic acid producers in the upper portion of the intestinal tract brings about a condition of acidity which inhibits the growth of certain

potentially harmful organisms. A moderate degree of acidity also promotes the absorption of calcium salts. Other organisms which ferment carbohydrates or decompose fats with the production of large amounts of acetic, formic and other organic acids exert a harmful influence, as certain of these acids are irritating to the intestinal tract.

Some strains of B. coli, B. aërogenes capsulatus and certain streptococci growing under favorable conditions produce large amounts of the protegenous amines, such as histamine. Under normal conditions, histamine is detoxified on passage through the intestinal mucosa and the liver, so that it does not exert a harmful effect. Apparently considerable amounts of histamine can be produced in the colon without causing damage to the body as a whole. When, however, histamine is injected subcutaneously or is absorbed from the mucosa of the mouth, very toxic symptoms are produced which are of a shock-like nature, often accompanied by vomiting, diarrhea and anhydremia. There is evidence that histamine and similar substances may be absorbed through an injured intestinal mucosa and not be completely detoxified. It appears likely that the growth of histamine-producing organisms in the upper portion of the small intestine is capable of producing severe gastrointestinal disturbances. It has been shown, furthermore, that when the contents of the upper portion of the small intestine are strongly alkaline, colon bacilli may pass through the mucosa and enter the blood stream.

The B. aërogenes capsulatus (gas bacillus) decomposes sugar with the formation of hydrogen and butyric acid. This latter is irritating and may, when present in large amounts, lead to diarrhea.

The relationship of intestinal bacteria to diarrhea is discussed further in Chapter XX.

CHAPTER X

THE STOOLS IN INFANCY

Meconium

The first stools of the infant consist of dark, brownish-green, semisolid meconium. The first passage is likely to occur during or very shortly after birth. The meconium consists of partially dried intestinal secretions which have accumulated in the large intestine from the fourth fetal month on. The amount of meconium thus accumulated is usually sufficient to fill the distended sigmoid and descending colon and amounts to from 5 to 7 ounces (150 to 200 grams). The gradual passage of this meconium during the first three or four days of life accounts for much of the initial weight loss of the newly born. The meconium is usually passed from three to five times daily. By the third or fourth day there occurs a gradual transition to the usual type of infant stool. In cases in which the food intake is very small, the meconium-like character of the stools persists for a longer period.

Stools of the Breast-Fed Infant

The normal breast-fed infant usually passes from two to four stools a day. These stools are soft and golden yellow or greenish-yellow in color, acid in reaction, and have a slightly sour, but not unpleasant odor. The number of stools is subject to considerable variation. Some perfectly normal infants who are thriving may have only one stool a day and others as many as seven or eight. The consistency may also vary from semiliquid to salve-like. The color may vary from yellow to definite green. Any sudden change in the character or number of the stools is of significance.

Stools of the Artificially Fed Infant

Infants fed on cow's milk usually pass a smaller number of stools than those who are breast fed. The stools are, however, more bulky and contain more solid material. The color differs

from that of the breast milk stool, being lighter yellow or grayish brown. The reaction is usually either neutral or alkaline as compared with the acid reaction of the breast milk stool. The odor is more unpleasant, especially when relatively large amounts of milk and small amounts of sugar are fed. When foods other than milk and sugar are added to the diet, the character of the stools changes until with a general mixed diet the stools are of the same character as those of the adult.

The Number of Stools

The number of stools depends upon the total amount and character of the food and the presence or absence of infection in the intestinal tract or elsewhere, and the individual tonicity of the gastrointestinal musculature. A large proportion of fermentable carbohydrate in the food, especially of lactose, leads to an increase in the number of stools. An excess of protein, especially casein, leads to a condition of alkalinity in the intestinal tract and decreased peristalsis, so that the number of stools is less frequent. An excess of fat, together with an excess of carbohydrate, leads to frequent movements, but a relatively large amount of cow's milk fat, together with considerable protein and minimal amounts of fermentable carbohydrate sometimes leads to constipation with the passage of large, firm soap stools. An insufficient intake of food may result in constipation but is as likely to lead to the passage of numerous small dark-green stools—the so-called starvation diarrhea.

Infestation of the intestinal tract, especially the upper intestine with bacteria capable of decomposing food with the production of irritating products leads to the passage of numerous liquid stools. Dysentery infections leading to ulceration in the lower intestinal tract result in the passage of very numerous stools containing mucus, pus and often blood. Infections outside the intestinal tract may also lead to diarrhea. The influence of such infections is further discussed in Chapters XX and XXVIII.

Some infants have constitutionally atonic intestinal musculatures and in consequence sluggish peristalsis and suffer from constipation. Other infants are constitutionally of the hypertonic type, have active peristalsis and suffer from a tendency to diarrhea.

The Color of the Stools

The color of the stools depends chiefly upon the character of the diet and the rate of peristalsis. Bile pigment gives the predominating color to most stools. The bile as secreted in the upper intestine is green, due chiefly to biliverdin. When peristalsis is active, the bile mixed with the food passes through the intestinal tract, largely unaltered and the stools are consequently green in color. When food remains in the intestinal tract for a longer period of time, reduction of the green biliverdin to yellow or brown bilirubin occurs. When the contents remain in the intestine for a still longer time, bilirubin may be in part reduced to colorless hydrobilirubin. The stools are then very light colored. The presence of oxidizing bacteria in the intestinal tract favors oxidation of yellow bilirubin to green biliverdin, especially in the presence of acid. The stools, under these conditions, are green. The swallowing of considerable amounts of air also tends to bring about oxidation and the passage of green stools. These facts explain the green color of certain diarrheal stools, the yellow color of the stools of the normal breast-fed infant when moderate peristalsis is present, and the light color of the constipated stools of the artificially fed infant. A stool which is yellow when passed may change to green on exposure to the air due to oxidation of bilirubin. Diminished or absent secretion of bile results in lack of color of the stools. A large proportion of lime soaps in the stools also causes them to be light in color. When cereals and certain malt preparations are fed, the stools assume a brownish shade. Occasionally a pinkish ring is seen at the edge of stools which have remained for some time on the diaper; this color is usually due to altered bile pigment or occasionally to urates, it is without significance but may be mistaken for blood. When spinach is fed, the stools may be green, and the feeding of tomatoes or beets may cause occasional red specks in the stool. Bismuth, iron or argyrol color the stools dark brown or black. Blood which has been swallowed or passed high in the intestinal tract will also color the stools dark reddish brown or black. Chemical tests may be necessary to differentiate blood coloration from that caused by drugs. Small streaks of blood on the outside of a constipated stool are generally indicative of anal fissures. Bright blood mixed with the stool is usually indicative of ulceration in the colon or

lower ileum and is seen characteristically in the presence of bacillary dysentery. Large amounts of bright blood appear immediately following intussusception.

Curds

The solid material of the stool consists largely of lime soaps, calcium phosphate, mucus and dead bodies of bacteria. When raw milk is fed, casein curds may also be present. When peristalsis is sluggish, a good deal of the water from the intestinal contents is absorbed, and the solid material of the stool is compressed into firm masses consisting largely of lime soaps. When peristalsis is active, less water is absorbed, and the churned up lime soaps appear in the stools as soft, white or bile-stained curds. The presence of such curds indicates merely that fat has been present in the diet and that peristalsis has been active. Soap curds are often found in the stools of normal breast-fed infants and are of but little significance.

In the stools of infants fed on raw cow's milk and occasionally those fed on pasteurized milk hard, yellow, bean-like masses of undigested casein may occur. These casein curds are not seen in the case of infants fed on breast milk nor on boiled, evaporated, dried or acidified milk.

Small stringy curds or partially dried balls of mucus are occasionally seen, especially in the stools of breast-fed infants.

Mucus

A small amount of mucus is present normally in the stools. The amount is increased whenever there is intestinal irritation. Especially large amounts of mucus occur in the presence of bacillary dysentery. The mucus is secreted chiefly by the mucosa of the lower bowel.

Starch

When young infants are fed on large amounts of starches or cereals, a portion of the starch escapes digestion and reappears in the stools, giving them a slimy consistency. When such stools are brought into contact with iodine solutions the starch particles are stained a deep blue. The passage of undigested starch through the intestinal tract rarely causes harm, but occasionally excessive fermentation of starch occurs in the intestine, and it then becomes necessary to reduce this element of the diet.

Cellular Elements

A few leucocytes and epithelial cells may be found microscopically in most stools. Any great excess of pus cells sufficient to cause the appearance of macroscopic pus is abnormal and is indicative of intestinal lesions. The appearance of pus in the stools is, in most cases, indicative of intestinal ulceration or dysentery.

Membrane or large numbers of desquamated epithelial cells are most frequently seen in the earlier stages of bacillary dysentery. Red blood cells do not appear normally in the stools.

Bacteriology of the Stools

Bacteriologic examination of the stools gives definite information concerning the bacteriology of the lower bowel only and does not necessarily serve as an indication of conditions in the upper intestine. Bacteriologic examination of the stools is of especial value in arriving at a diagnosis of bacillary dysentery. For technic see Chapter XXXIV. Some pediatricians consider the finding of the gas bacillus (B. aërogenes capsulatus) (Welsh bacillus), in the stools to be of pathologic significance. This organism is frequently found, even in normal stools, but when very large numbers are present, the products of fermentation, especially butyric acid, may possibly cause irritation of the mucosa and consequently diarrhea.

In the alkaline soap stools of artificially fed infants, the B. ammoniagenes (Cooke) is not infrequently present. This organism is capable of decomposing urea with the formation of ammonia. The presence of this organism is the cause of the "ammoniacal diaper" so often observed in the case of artificially fed infants. It has been shown by Cooke that soaking the napkins in 1:5000 bicholoride of mercury or in boric acid solution before drying results in the inhibition of the growth of this organism. Changing the character of the diet by reducing protein and fat and increasing carbohydrate so as to cause an acid condition of the contents of the lower bowel also results in inhibition of the growth of B. ammoniagenes as it thrives only in an alkaline medium. Changes in the diet are, however, not necessary in the treatment of the condition.

Significance of Stool Examination

From what has been said, it is evident that the stools may vary greatly even under normal conditions. If an infant is gaining in weight, has no fever, and is healthy in appearance, it makes very little difference whether the stools number one or seven a day, whether they are firm or semi-solid or whether they are light yellow, orange or green and whether or not they contain fat or protein curds. The general health of the infant is of far more significance than the character of the stools. If, however, an infant who has normally been having one or two stools a day begins to have six or seven with an increased amount of mucus and the stools become watery, the probability is that some infection either within or without the gastrointestinal tract has occurred or that some unsuitable article of diet has been taken. This subject is further considered in Chapter XX. Very large, light colored foul stools containing much soap, and some neutral fat are seen in cases of chronic intestinal indigestion or celiac disease. This condition is discussed in Chapter XXII. The presence of macroscopic pus and blood in the stools is indicative of definite pathologic conditions. Microscopic examination of the stools of infants usually gives but little information that cannot be obtained by simple inspection.

CHAPTER XI

BREAST FEEDING OF THE NORMAL INFANT

General Considerations

The milk of a healthy mother, who is receiving an adequate mixed diet, contains all the food elements necessary for the nutrition of the infant, for at least the first half year of life. The caloric value of human milk is such that when the infant is fed at reasonable intervals and takes as much as he desires, the total energy requirements are met. There is little danger of over- or underfeeding, provided the mother's supply of milk is adequate. The milk in the breasts is normally free from harmful bacteria, and if reasonable cleanliness is observed and there is no infection of the nipples, the milk as received by the infant will also be free from harmful bacteria. Volume for volume, human milk is more readily digested by the human infant than is undiluted cow's milk. It has been assumed that breast milk contains certain "immune bodies" which are of value in rendering the infant resistant to infection, but there is no convincing proof of this fact and furthermore most immune bodies are destroyed during the processes of digestion.

In general, infants who are breast-fed are larger, healthier, and suffer less from disease than those who are artificially fed. The mortality during the first few months of life is, according to most statistics, lower among infants who are entirely breast fed. The reasons for this are evident. The breast-fed infant is likely to receive more total food than the one who is artificially fed. Human milk contains on the average 20 calories per ounce; many of the cow's milk formulas in common use contain fewer calories per ounce. The infant nursing a mother with an abundant milk supply is not as restricted in his intake of food as is the bottle-fed baby, he takes enough to meet the demands of hunger, and this is often in excess of the volume of food which it has been artibrarily decided that he should receive from the bottle. In consequence, a breast-fed baby receives an adequate amount of food and the artificially fed one only too often an inadequate amount. Human milk contains all of the necessary constituents of the diet, whereas an artificial formula may be deficient in certain constituents due to

improper construction of the formula. There is but little chance of bacterial contamination of the milk when the infant receives it directly from the breast; in the preparation of a formula, there is always the chance that harmful bacteria may be introduced. With the modern knowledge of nutrition, an infant *may* be satisfactorily nourished by a prepared formula and be as healthy in every respect as the breast-fed infant. There is, however, a greater chance of error. The total amount of food may be insufficient, certain essentials may be omitted or bacterial contamination may occur. As a matter of fact, a very large proportion of artificially fed babies have, in the past, been underfed. At the present time there are fewer infants who are underfed but still entirely too many. Ignorant parents cannot be depended upon to prepare the formulas under aseptic conditions, although formulas made from cow's milk may be so constructed, as for example by the addition of acid, that they are poor culture media.

Breast feeding is not always successful. The total amount of milk may be inadequate for the infant's needs so that supplementary or complementary feedings with an artificial formula may be required. The milk may be of poor quality because of ill health on the part of the mother or because her diet is lacking in certain essentials. It is, however, advisable, whenever possible, to have the baby nurse at the breast, and it is possible, for at least the first few months of life, in something over 85 per cent of cases. On the other hand, it is not advisable to allow a baby's nutrition to suffer because the mother has an insufficient supply of milk, or to urge a delicate mother, having a scanty supply of milk, to nurse an infant who is in good health and capable of thriving on an artificial formula. Breast feeding should especially be encouraged among the poor and ignorant, as it is difficult for people of this class to carry out satisfactory artificial feeding. Many babies are unnecessarily weaned because of minor complaints. The milk of a healthy mother usually agrees with the baby, provided the milk is sufficient in quantity and the baby is nursed at the proper intervals.

Contraindications to Breast Feeding

There are certain definite contraindications to maternal nursing. A mother with active tuberculosis should not nurse her baby, nor should she come in contact with it in any way. Tuberculosis is

probably not transmitted directly through human milk, but the close contact with the mother necessitated by nursing exposes the baby to infection, and young infants are extremely susceptible to tuberculous infection. We have seen a young infant succumb to tuberculosis who had only been handled twice by a mother in the active stage of the disease.

A mother who is herself in poor general health and suffering from severe chronic illness such as advanced nephritis, cardiac disease, or cancer should not nurse her infant. A mild degree of nephritis without uremia or nonprotein nitrogen retention is not a contraindication to nursing. Diabetes is usually not a contraindication; in fact, the mother's carbohydrate tolerance is higher during the nursing period because of the secretion of carbohydrate in the milk.

In the presence of mastitis, nursing of the affected breast should be discontinued. With double mastitis, complete weaning is necessary. Acute infections on the part of the mother necessitate *temporary* weaning, but the breasts should be emptied by manual expression (for technic see page 113) and the infant should nurse again as soon as the mother's temperature has returned to normal. More prolonged infections, such as typhoid, necessitate permanent weaning. If the mother has to undergo a surgical operation, the infant should be taken from the breast temporarily and the breasts pumped until after the mother has recovered from the effects of the anesthetic.

Syphilis in either the mother or the baby is no contraindication to nursing. The nutrition of the syphilitic infant is likely to be poor, even under the best circumstances, and it is these infants who especially require mother's milk. Certain infants, especially those born prematurely, are too weak to nurse satisfactorily; others are unable to nurse because of such deformities as very wide cleft palate and harelip. Under such circumstances, the breasts should be emptied mechanically and the milk fed to the infant from a bottle or medicine dropper.

The occurrence of menstruation during the period of lactation is not an indication for weaning. When pregnancy occurs, it is advisable to wean the infant gradually, but many mothers are able to nurse their babies satisfactorily during the first few months of pregnancy, provided they are themselves in good health.

Vomiting, colic, diarrhea, or failure to gain in weight are not necessarily indications for weaning. Such symptoms may call for more frequent or less frequent feeding, for attention to the diet and hygiene of the mother or occasionally for complementary or supplementary feeding from the bottle.

CHARACTERISTICS OF HUMAN MILK

Colostrum

During the first few days *postpartum* the milk secreted differs considerably in character from that secreted later. The first milk, known as colostrum, is a thin, yellowish fluid containing more protein and salts and less fat and sugar than the later milk. It contains numerous large lymphocytic cells—the "colostrum corpuscles." Unlike milk, the colostrum is readily coagulated by heating. It appears to have a certain laxative effect and may aid in bringing about evacuation of the meconium. Experimental work on animals indicates that globulin is present in colostrum which may be absorbed unchanged into the blood and facilitate the development of immunity. It has not been shown that this occurs in the case of the human infant.

The amount of colostrum secreted is relatively small, usually not more than 5 to 10 ounces in the course of a day. About the third or fourth day the milk "comes in." The breasts become distended and tender, and there is a marked increase in the amount of secretion, which gradually assumes the character of mature milk. For at least one or two weeks, however, the milk still retains some of the characteristics of colostrum. By the end of the first month, the character of the milk is essentially the same as that which is secreted during the remainder of the nursing period.

Composition of Human Milk

The chief constituents of human milk are fat, sugar, protein, mineral salts, water and the four essential vitamins. There are present also other substances of uncertain composition, the functions of which are not fully known. The average composition of mature breast milk is given in Table IV.

The caloric value of breast milk is approximately 20 calories per ounce, or 650 calories per liter.

TABLE IV

COMPOSITION OF HUMAN AND COW'S MILK

	FAT	SUGAR	TOTAL PROTEIN	LACT-ALBUMIN	CASEIN	TOTAL MINERAL	CaO	MgO	K_2O	Na_2O	P_2O_5	SO_3	Cl	Fe_2O_3
Human	4	7.0	1.25	0.75	0.50	0.25	0.05	.005	.086	.020	.036	.004	.038	.00018
Cow's	4	4.5	3.50	0.50	3.00	0.75	0.20	.022	.145	.074	.250	.054	.076	.00007

The total amount of fat in human milk is more variable than that of any other constituent. Although the usual variation is from 2.5 to 5.0 per cent, as low values as 1.0 per cent and as high as 10 per cent have been observed. The first portions of the milk removed from the breast at a nursing are low in fat; the last portions, or strippings, considerably higher. No conclusions as to the fat content of the milk should be arrived at unless the breast is entirely emptied, and the fore portions and last portions mixed.

The fat of human milk is present in the form of minute globules held in a state of emulsion. The fat globules of human milk are smaller than those of cow's milk. The chief fats are tripalmitin tristearin and triolein, of which triolein comprises approximately 50 per cent. About 2.5 per cent of the fats of human milk are glycerides of volatile fatty acids; this is a much lower proportion than in the case of cow's milk. This latter fact is of some importance in infant feeding, as the volatile fatty acids are especially irritating to the gastrointestinal tract.

The total amount of protein in human milk is not subject to very wide variation. The protein content of the colostrum is relatively high (2.5 to 5.0 per cent) but in mature milk it is fairly constant, being between 1.0 and 1.5 per cent. The proteins of human milk are casein and lactalbumin, two-thirds of the total protein present being lactalbumin. The characteristics of these proteins are discussed elsewhere (see Chapters III and IX).

The total amount of protein in human milk is much less than in cow's milk, but because of the larger proportion of lactalbumin, it has a higher nutritional value. The amount of protein in human milk is sufficient for the needs of the average normal infant. It is, however, not always sufficient for the needs of the premature or greatly undernourished infant.

The sugar of human milk is exclusively lactose. The amounts present are remarkably constant, varying less than those of any other constituent. Such variations as are reported in the literature are to be explained largely on the basis of the different analytical methods used.

Human milk contains all the mineral salts necessary for the nutrition of the normal infant, with the possible exception of iron. There are, however, larger amounts of iron in human than in cow's milk. Human milk contains less phosphate and calcium than cow's milk but the calcium may be more completely utilized by the infant.

The vitamin content of human milk has been discussed elsewhere (see Chapter VII).

The "buffer" value of human milk, or the capacity for neutralizing gastric acid is very low as compared with cow's milk.

The bacteria usually found in human milk have no pathogenic significance for the healthy infant. Such bacteria as are found are chiefly nonpathogenic cocci derived from the external milk ducts. In the presence of a suppurative mastitis the causative organism may be present in the milk in large numbers. Any organism which occurs in the mother as a cause of septicemia may pass into the milk. Tubercle bacilli are not found in human milk unless the breast itself is the seat of the disease.

The Hygiene of the Nursing Mother

In order that there may be an adequate secretion of milk of good quality, it is essential that the mother herself be in good physical condition and that she receive an adequate diet. The mother should have plenty of sleep and exercise in the open air. If the feeding intervals are not too short, the mother can have more time free for rest and recreation. It is especially desirable that she should have, if possible, unbroken sleep during the night. After the infant is a few weeks old, it is often possible to omit the night feedings. The mother should rest for at least an hour each afternoon. She should completely relax and rest for a while after each nursing.

Any form of outdoor exercise which the mother enjoys is good for her, provided it is not carried to the point of fatigue. A mother who is overworking around the house and is tired out is not likely to be benefited if forced to take additional outdoor

exercise. Sitting outdoors in a comfortable chair or hammock or motoring will be of more benefit in such a case. The mother should, as far as possible, be relieved of worry and mental strain. If her supply of milk is insufficient in quantity and the baby is consequently always hungry and fretful, the mother will get little rest and her milk supply will not increase. Under such circumstances, it is better to give the baby additional food in the form of a suitable artificial feeding.

Amounts of Milk Secreted

The total amount of milk secreted daily depends upon the demands of the infant and the capacity of the breasts to secrete. The chief stimulus to milk secretion is complete emptying of the breasts. A healthy, vigorous infant will be likely to empty the breast rather completely and this results in the stimulation of milk secretion. The amount of milk secreted by a healthy mother usually increases in proportion to the infant's demands. Some mothers are able to nurse more than one infant. Regular and complete emptying of the breasts either by the infant or by manual expression may increase the amount of milk enormously. Wet nurses, especially negroes, have been known to produce more than a gallon of milk a day. It is impossible to state beforehand whether or not a mother will be able to produce enough milk to meet entirely her infant's needs. Some thin, but healthy mothers, with small breasts, produce much larger quantities of milk than large, well-developed fat mothers. A mother who has been unsuccessful in nursing previous infants is not likely to be able to secrete enough milk.

If the mother's diet is already adequate in every particular, forced overfeeding will not increase the secretion of milk, but the milk secretion of the underfed mother may be increased by the giving of sufficient food. The amount of human milk secreted in a day to meet the needs of the nursing infant will have a food value of from 500 to 1000 calories. This extra food must come from the mother's body or be supplied in her diet. The nursing mother should consequently take more food and more fluids than under ordinary conditions. Milk, may, however, be secreted even at the expense of the mother's own tissues.

The nursing mother does not require a very special form of diet. A diet which is suitable for her under normal conditions

will be suitable during the period of lactation except that she will need to take a somewhat larger amount. There seems to be no good basis for the idea that the nursing mother cannot take acid fruits, vegetables or salads. These are all good for her if they do not upset her digestion. Certain foods such as onions and garlic give a taste to the milk to which older infants may object. The daily diet should contain milk, butter, eggs, two green vegetables, one of which should be raw, and a fresh fruit, preferably orange.

Milk is especially valuable as it supplies the materials from which the mother may produce milk. The nursing mother should take from 1½ pints to one quart of milk a day. It is not necessary that she take this in the form of liquid milk. To many adults, milk is distasteful; in such cases the milk may be cooked in the food or served in the form of cocoa, or as ice cream or custards. Evaporated milk lends itself well to the preparation of various dishes and has essentially the same value in nutrition as ordinary bottled milk. One pound of evaporated milk is equivalent to a quart of bottled milk, and this amount can easily be incorporated in the daily diet.

Butter contains the vitamins A and D of the milk. Not less than one ounce should be taken daily. Oleomargarin should not be substituted for butter as it is deficient in the vitamins. Full cream cheese is almost as valuable as the milk from which it is made, containing most of the fat, protein, and fat soluble vitamins.

Eggs are valuable because of their high vitamin D and calcium contents. One or preferably two eggs should be taken daily. These, like milk, may be incorporated in other foods.

Green vegetables should have an especially important place in the diet because of their contents of vitamins B and C and mineral salts, as well as their laxative effects. Recent work has shown that the vitamin B requirements for lactation are high. A leafy vegetable should be served daily as salad. Tomatoes are a valuable source of vitamin C.

The juice of one large or two small oranges should be taken daily.

When the diet contains the articles mentioned, it is rather immaterial what goes to make up the remainder; it is essential, however, that the total calories be sufficient to provide for the needs of the mother and for the secretion of milk as well. The nursing mother should take about 1000 more calories than she would other-

wise. She may make up the calories in almost any way she prefers, so long as the essential foods, milk, green vegetables and fruits are taken in sufficient amount. She may take meat once a day or three times a day, she may take carbohydrate as potatoes, white bread, whole wheat bread, rye bread or cereals, hot or cold. She may eat hot cakes and syrup and a reasonable amount of candy and cakes, provided her own digestion is not thereby upset.

A sufficient intake of fluids is essential. A nursing mother should drink at least a quart of water a day in addition to the quart of milk. The water may be taken with fruit juices or other flavored beverages.

Mothers whose diets are insufficient in total amount to meet their own needs may secrete milk which is produced at the expense of the mother's body, but the amount secreted is likely to be scanty and to decrease gradually. Overstuffing the mother with food will, however, not increase her milk secretion. It is doubtful whether the proportions of fat, carbohydrate, and protein in the milk are very greatly influenced by changes in the character of the mother's diet, although undoubtedly the vitamin content is susceptible to considerable variation. There is no evidence that the taking of moderate amounts of alcoholic beverages or of tea and coffee or smoking in moderation affects the character of the milk adversely, provided the mother is not affected.

Relatively few drugs taken by mouth are excreted in the milk in sufficient amounts to have an effect on the infant. Atropine, morphine, iodides, bromides, arsenic, salicylic acid and some of the coal tar antipyretics are secreted into the milk in small amounts. The taking of laxatives by the mother is generally believed to produce laxative effects on the nursing infant. This is difficult to explain.

Care of the Breasts and Nipples

The breasts should not be compressed by close fitting dresses or brassiers, but pendulous breasts should be loosely supported. Cleanliness of the breasts and nipples is essential. The nipples should be gently washed with clean (preferably boiled) water before and after each nursing. This should be done with clean cotton cloth or gauze. The nipples should not be handled with the fingers. A clean piece of cloth should cover the nipples between nursings.

If the nipples become tender or chapped, they should be greased with boric ointment, lanolin or 10 per cent balsam of Peru ointment.

The chief thing to be feared and avoided in connection with the mother's nipples is that they become chapped and fissured. Such fissures make nursing a very painful process and at the same time offer abundant opportunity for entry of pyogenic bacteria which may cause inflammation or abscess formation to such an extent as to make nursing not only temporarily, but even permanently impossible. If the nipples have become fissured, it is well to allow the baby to nurse only through a nipple shield, keeping the breasts empty, if necessary, by manual expression or a breast pump. Healing of fissures is promoted by the application of a 5 per cent solution of silver nitrate. In the case of abscess formation, it is usually necessary, at least temporarily, to wean the baby from the involved breast. The breast should be emptied by manual or mechanical means and the milk discarded until healing has occurred, when the nursing may be resumed.

When the milk secretion is first established, the breasts may become painful, due to engorgement. This same condition of engorged or "caked" breasts may occur from time to time subsequently. In such instances the milk flow may be diminished through the wearing of a tight binder and through restriction of fluids. At times it is necessary to relieve the engorgement by manual or mechanical expression of the milk. (For technic, see page 113.)

Technic of Breast Feeding

The baby should first be placed at the breast about twelve hours following delivery and thereafter every six hours during the next twenty-four hours. Very little colostrum may be obtained by the infant, but the nursing act will tend to stimulate the flow of milk and will accustom the baby to the nursing act. The practice of putting the baby to the breast sooner than twelve hours after delivery has not been shown to be an advantage.

Beginning on the third day the infant should nurse both breasts every four hours. Very little milk will be obtained during the first few days so that after each nursing the infant should be given sterile water, sugar solution, or a milk formula from a bottle. A ten per cent solution of Karo syrup or a five to seven per cent

solution of cane sugar or milk sugar is suitable. Although sugar solutions have been very generally used for complemental feedings, our own preference is for a milk mixture. A suitable formula consists of whole lactic acid milk or one-half diluted evaporated milk with 7 to 10 per cent of added corn syrup or dextrimaltose. (See Chapters XV and XVI.) If by the fourth or fifth day the amount of milk obtained by the infant from the breast is found to be insufficient, the complemental feedings of a cow's milk formula should be continued. Such feedings may only have to be kept up for a few days. In our experience this practice has not had any deleterious effects and is preferable to permitting the infant to starve. The complemental feedings should always be offered *after* the baby has been put to the breasts and has taken as much milk as he can obtain.

When the feeding interval is four hours, it is usually advantageous for the infant to nurse both breasts at each feeding. In this way more milk can usually be obtained than with three-hour nursings of alternate breasts. Occasionally when the milk secretion is scant, nursing both breasts at three-hour intervals is an effective means of increasing the flow. At times the infant is unable to empty completely the breasts because of the presence of depressed nipples or because of weakness or mouth deformity on the part of the infant. In such cases the breasts should be emptied mechanically and the expressed milk fed to the baby until the nursing of a sufficient amount becomes possible.

A few infants fail to do well on a four-hour feeding schedule. Some become very hungry at the end of two and one-half or three hours, and cry until the feeding is given. This may be simply an indication of underfeeding. On the other hand, it may mean that the infant's gastric motility is rapid and his stomach quickly emptied. Small or weak infants who do not nurse vigorously often do better on a three-hour schedule. If a three-hour feeding schedule is adopted, the breasts should usually be nursed alternately except during the period when the milk flow is being established. It is not advisable to have the infant nurse more often than every three hours.

The four-hour nursing interval has a number of advantages over shorter intervals. The infant is hungry at the end of four hours, nurses vigorously, and more completely empties the breasts. This in itself is the best stimulus to milk production. The gastro-

intestinal tract of the infant, especially the stomach, has a chance for rest between feedings and is not constantly taxed to capacity. The infant fed at four-hour intervals takes more food at a feeding and sleeps longer following the feeding, thus getting more rest in the course of a day. Vomiting is less frequent in infants fed at longer intervals than in those fed at two or three-hour intervals. The long feeding interval gives the mother a chance for rest and recreation and makes it unnecessary for her to spend her entire time with the baby. Some mothers, who are unwilling to nurse their babies at short intervals, do not object to the longer intervals.

The number of feedings in twenty-four hours will depend upon the adequacy of the milk supply. When the mother has an abundant supply of milk, even a young infant may receive sufficient food in four or five nursings to meet all of his requirements. As soon as the infant will sleep through the night without waking and is gaining weight at a satisfactory rate the 2 A.M. feeding may be dropped and later the one at 10 P.M. Occasionally, when the night feedings are dispensed with, the infants awaken at night, either through force of habit or because of thirst, and cry. The giving of a bottle of water is likely to satisfy them. Later during the first year, when cereals and vegetables are added to the diet, the infant may take less milk at some of the nursings during the day so that it may become advisable to resume one of the night nursings.

A feeding schedule once adopted should be adhered to regularly, and the infant should be awakened at feeding times during the day. Occasionally infants who are fed at four-hour intervals become very hungry at the end of three or three and one-half hours, especially when they have been active and when the previous feeding has been a small one. Under these circumstances the infant should not be allowed to cry for a half hour or hour, but should be fed. The following feeding, however, should be at the regular hour. It is very easy for a mother to get in the habit of feeding the baby every time he cries and thus make the feeding interval shorter and shorter. This is especially likely to happen in the case of babies who are definitely underfed because of a deficient milk supply. The amount of milk actually taken by the baby should be determined and if insufficient should be complemented by a cow's milk formula. Babies with irregular nursing habits

are likely to be fussy and colicky, and the mother who feeds the baby whenever he cries becomes tired and nervous and her milk supply consequently diminishes. An especially pernicious habit is that of having the baby sleep with the mother and nurse at frequent intervals during the night. The baby should be in a crib by himself, preferably in another room.

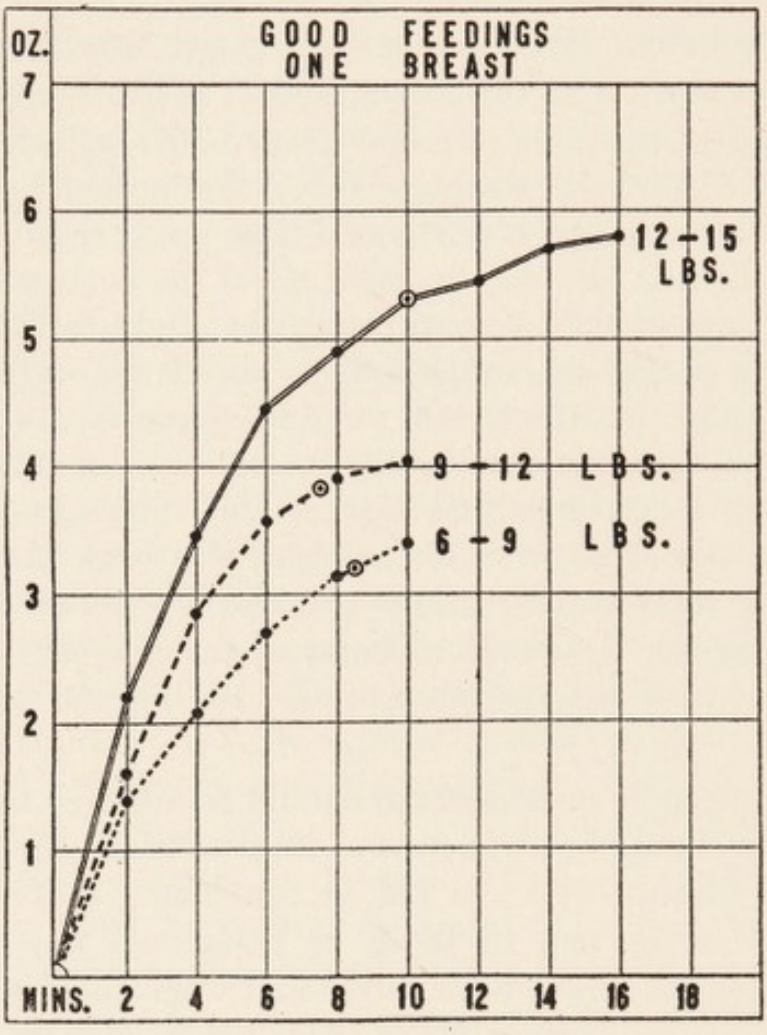


Fig. 5.—Curves showing rate at which milk is taken at a nursing by normal infants of varying weights. Average time after which no more milk is obtained is shown by circle surrounding dot. (Smith, C. H., and Merritt, K. K., Am. J. Dis. Child. xxiv, 413, Nov. 1922.)

The number of minutes that an infant nurses depends upon the sucking strength of the infant, the amount of milk available, and whether the breasts are difficult or easy to empty. Under normal conditions, a vigorous infant will obtain as much as one-half of the milk in the first two or three minutes and three-fourths of the total within the first five minutes. Very little milk is ob-

tained after ten minutes. Small and weak infants obtain milk at a considerably slower rate. These facts are well illustrated in the accompanying chart of Smith and Merritt (Fig. 5).

A healthy baby nursing both breasts should not nurse longer than ten minutes at each, and one nursing a single breast, not longer than fifteen minutes. Some very weak infants may nurse as long as twenty minutes. Infants who remain at the breast for long periods are likely to swallow air instead of milk, and this tends to cause vomiting and colic.

The position of the baby during the nursing is of importance. During the first two weeks, the mother will, of necessity, have to lie in a recumbent position while nursing. After she is up and around, however, it is better that the mother sit up while the baby is nursing and that the baby be held in a semi-erect posture; the swallowing of air is then largely avoided and the air already in the stomach tends to pass out as the milk is swallowed. It has been shown that infants held in a semi-erect posture usually take more milk at a feeding than those who are fed while recumbent. This is due to the fact that the intake of food is not interfered with by distention of the stomach with air. All babies swallow a certain amount of air, and roentgenograms regularly show an air bubble in the stomach. There may be sufficient air almost to fill the stomach. In all instances before the nursing, the baby should be held upright over the mother's shoulder and patted on the back until any swallowed air is belched up. The infant should also be held up immediately after the nursing. In some infants, it is necessary to interrupt the nursing to allow swallowed air to escape.

The amount of milk obtained at a single nursing is not uniform and depends upon the appetite of the infant and the amount of milk secreted by the mother. A healthy normal infant may take eight ounces at one feeding and three at another. If an infant has slept for three or four hours between feedings, he is likely to take less than if he has been awake and active. After a long night interval a much larger amount of milk is likely to be taken than at the other feedings during the day. It is consequently quite impossible to state even approximately the number of ounces a breast-fed infant should be expected to take at a single nursing, although the total amounts of milk required per day may be stated

more definitely. No idea as to the adequacy of the milk intake can be obtained from an observed single feeding. To determine the total daily amount of milk received by the infant, he should be weighed before and after several feedings on successive days and the results averaged. Weighings should be performed with clothes and diapers on, for if the diaper is changed the loss of weight due to passage of urine or stools may lead one to erroneous conclusions as to the intake of milk.

The average normal infant requires from two and one-half to three ounces of breast milk per pound of body weight per day to insure an adequate energy quotient. This is based on the assumption that the breast milk is of average quality and has a caloric value of twenty calories per ounce. If an infant fails to gain, is fretful and hungry, and it is found that he is receiving an amount of milk considerably below the quantity mentioned, it may reasonably be assumed that the feeding is inadequate. If, on the other hand, an infant fails to do well despite the fact that he is receiving as much as three ounces of breast milk per pound of body weight, the probability is that the trouble is not due to underfeeding.

Characteristics of the Normal Breast-Fed Infant

The normal breast-fed infant is well nourished, his flesh is hard and firm, his eyes are bright and his cheeks and nails pink. He is happy and active when awake. He cries only when hungry or uncomfortable from such causes as cold, a wet diaper, improperly adjusted clothing or distention of the stomach from swallowed air. He has from two to four stools daily during the first few months and from one to three daily after that time. The normal breast-fed baby gains from six to eight ounces a week throughout the first year (see weight curves, Chapter I). A healthy breast-fed baby usually sleeps for twenty to twenty-two hours a day during the first two months and from sixteen to twenty hours a day during the remainder of the first year. Some babies, however, sleep considerably less and thrive.

Infants nursed at the breast usually do well provided the mother has a sufficient supply of milk and the baby is fed at proper intervals. Failure to do well is more likely to be due to the presence of infections or to congenital anomalies on the part of the infant than to the character of the feeding.

Underfeeding

Certain breast-fed infants fail to receive a sufficient amount of milk from the mother and are consequently underfed. The symptoms of underfeeding depend upon the degree of deficiency of the diet. There may be only a small gain in weight, or an actual loss. The total volume of stools passed in a day is small, but the individual movements may be frequent and green. The diarrhea of starvation is often mistaken for overfeeding. The underfed infant is fretful, cries a great deal, and often suffers from colic. In fact, contrary to the usual belief, colic is more frequently due to underfeeding than to any other cause. The underfed baby sucks his fingers and anything he can get to his mouth and consequently swallows a great deal of air and often vomits from this cause. Hunger causes painful contractions of the stomach and gastrointestinal tract. If the total amount of milk obtained by the baby is found to be small, complementary feedings from the bottle should be given. At the same time, every effort should be made to improve the mother's physical condition and her diet, if inadequate, should be corrected. This phase of the subject has been discussed previously in this chapter.

There are no drugs which regularly increase the flow of milk. A temporary increase in the milk occurs when pituitrin is adminministered, but this appears to be due merely to the forcing out of milk from the breasts.

If, despite all efforts, the amount of milk obtained by the baby is insufficient for his needs, the complementary cow's milk formula is continued, or else the baby is allowed to nurse only for a few feedings a day and a bottle is given at the remaining feedings. It is usually inadvisable to wean the baby completely during the first few months of life if the mother is able to give as much as one-half of the milk necessary for his nutrition. When no more than this can be obtained, even after making every effort, it is not worth while to continue the breast feeding and the infant should be weaned completely.

Overfeeding

If an infant is nursed at intervals of four hours, there is little danger of overfeeding at the breast. Occasionally the mother has an abundant supply of milk which flows so readily that the infant gorges himself at each feeding and promptly regurgitates at the

conclusion of the nursing. This does relatively little harm, the spitting up of the food protecting the infant's intestinal tract from too great an excess. Sometimes the infant may retain more breast milk than his digestion is capable of caring for, and indigestion or diarrhea may result. The simple remedy is to limit the time of nursing, allowing the infant to nurse only one breast at a time and then for a short period, which may have to be only three or four minutes.

Unsuitable Breast Milk

A healthy mother's milk rarely disagrees with her baby, although it may be deficient in quantity or the baby may fail to do well because of irregular nursings. It must be admitted, however, that there are a certain very small number of babies with whom the mother's milk does not seem to agree. Analysis of the milk is often resorted to in an effort to explain the difficulty, but this usually gives little information of value.

About the only substance in the milk which varies sufficiently to cause disturbance is the fat. The fat content may be very low so that the milk has a low caloric value or the fat content may be so high as to lead to digestive disturbances. In some instances, in which the fat content is high, the difficulty may be remedied by having the infant only partially empty the breasts at a nursing. This practice, however, is likely to result in decrease in the flow of milk. It is said that the fat of the milk may be decreased by having the mother take more exercise, and various attempts at dietary regulation have been made in the effort to alter the fat content of the milk, but such means are rarely effective.

Mother's milk may be found to be average in composition and yet may disagree with the baby. Cases have been reported in which the infant appeared to be sensitive to foreign proteins present in the mother's milk. Elimination of the proteins in question from the mother's diet is indicated in such cases. Eggs appear to be the most frequent cause of sensitization.

In some instances breast-fed infants fail to do well, despite regular nursing, a sufficient food intake and proper care, and no source of infection or other trouble can be found. In such instances weaning may be necessary.

In a commendable effort to encourage breast feeding many extreme statements have been made, such as "a mother's milk al-

ways agrees with her baby," "a baby who does not do well at the breast will do worse on artificial feeding." Such statements are only relatively true. There are occasional exceptions. All human milk is not perfect any more than all human beings are perfect.

Gastrointestinal Disturbances of Breast-Fed Infants

Severe gastrointestinal disturbances occur rather infrequently in the case of infants who are exclusively breast-fed. Minor disturbances such as colic, spitting up of food, constipation or mild diarrhea are, however, of fairly frequent occurrence and call for appropriate treatment. Each of these conditions is considered in detail in individual chapters which follow.

Mixed Feedings

There are a number of conditions under which it is advisable to feed the infant partially from the breast and partially from the bottle. This may be necessary during the time that the milk secretion is being established. It also becomes necessary when the milk supply begins to diminish or becomes insufficient for the increasing needs of the growing infant. Weaning is usually accomplished by the gradual substitution of the bottle for the breast. Mixed feeding may be carried out in two ways, the artificial formula being given either as *complemental* or as *supplemental* feeding. By complemental feeding is meant the giving of a bottle to complete a single feeding. The bottle feeding in such instances should always be given after the infant has nursed. By supplemental or substitute feeding is meant substitution of the bottle for one or more of the nursings during the day.

Complemental feedings are usually used when the breast milk is insufficient for the infant's needs and an attempt is still being made to increase the amount. Supplemental feedings are used at the time of weaning when it is desired to allow the breast to dry up gradually. Supplemental feedings are also used occasionlly in the case of normal infants where the mother has sufficient milk but is required to be away from home daily for a longer time than the customary feeding interval. The giving of one bottle a day even when not necessary is occasionally advised because of the fact that this accustoms the baby to the artificial feeding so that no difficulty is likely to be experienced if the baby has to be weaned

suddenly or the mother has to miss one or two nursings. The disadvantages of this practice are those of artificial feeding in general. The milk mixture may not be suitable for the baby or it may be contaminated by bacteria due to lack of care in its preparation. A further disadvantage is that the missing of one or two nursings a day tends to diminish the mother's milk supply.

In the giving of a mixed feeding, the cow's milk formula should be one suitable in amount and composition for the age and size of the infant. The formula will be approximately the same as that used in the case of infants of the same age who are entirely artificially fed. The construction of these formulas is considered in the chapter on artificial feeding. Very young infants on mixed feeding may be given either sweet milk or acid milk. Older infants, however, will often refuse to take the bottle if it contains acidified milk because of the great difference in taste as compared with breast milk. Sweet milk formulas usually have to be used. Cane sugar, on account of its greater sweetness is the preferable carbohydrate to add to the formula for mixed feedings. It may at times even be necessary to sweeten the feedings with a small amount of saccharin.

Additions to the Diet of the Breast-Fed Infant

It is usually advisable to give cod liver oil to the breast-fed infant. This is particularly necessary in the case of premature infants, very rapidly growing infants and infants of certain races, especially negroes and Italians who are especially prone to rickets. Cod liver oil is not so necessary if the mother is taking a liberal amount of milk, butter and eggs, or if the infant is frequently exposed to sunlight. The administration of cod liver oil may be begun during the first month. One-half to one teaspoonful twice a day is sufficient for most breast-fed babies. This may be given at the time of nursing, the oil being fed with a dropper. In the case of breast-fed infants who show a marked tendency to rickets, the dose of cod liver oil may be increased or the infant may be given instead viosterol 100-D in a dosage of ten to fifteen drops a day. This latter preparation, although rich in vitamin D contains no vitamin A.

Beginning with the fifth or sixth month, the breast-fed infant should begin taking well-cooked cereal gruels, starting with one to two tablespoonfuls a day and increasing to four or five table-

spoonfuls twice a day. About the same time purées of spinach, carrots or mixed vegetables should also be given once a day in amounts of from one to two tablespoonfuls. There are a number of prepared vegetable purées on the market which are very satisfactory. By the eighth or ninth month the infant should be taking rather thick cereal from a spoon and may have a little meat broth, scraped beef, well-cooked ground liver or the yolk of an egg daily. Orange juice is not usually necessary as an antiscorbutic in the case of the breast-fed infant, but the giving of orange juice, prune pulp, apple sauce and well-ripened bananas may, to advantage, be begun between the sixth and ninth months.

Weaning

Under ordinary conditions, weaning should be begun about the eighth or ninth month. Frequently, however, insufficiency of the mother's milk may make it necessary to wean the baby partially or completely before this age. There is no particular advantage in continuing breast feeding beyond the tenth month. Infants who are breast fed for too long a period are likely to become anemic unless the diet is supplemented with green vegetables, meats or egg yolk. It is usually inadvisable to wean babies during very warm weather, because artificial feedings are more likely to be bacterially contaminated during warm weather, and furthermore the infant's digestive capacity is lower when the outside temperatures are high. The baby should, if possible, not be weaned during a period of acute illness.

Unless the infant has been accustomed to taking one or two bottles a day, weaning is sometimes difficult as the infant may obstinately refuse the bottle. In such instances, it may be necessary to starve the baby into submission, withholding all feedings except the bottle. A baby will sometimes refuse to take food from the bottle, especially if offered by the mother because he expects to be offered the breast. A stranger may be more successful in persuading such a baby to take the bottle. In rare instances, it becomes necessary to remove the baby to a hospital for weaning.

Wet Nursing

In a previous generation, the mortality of infants deprived of mother's milk was tremendous, and about the only hope of successfully raising such an infant lay in procuring milk from another

woman. All too frequently, mothers of the poorer classes abandoned their own babies and entered homes as wet nurses. Fortunately, such practices are not tolerated at the present day. It is not necessary for the wet nurse to wean her own baby. Indeed, many healthy wet nurses are able to supply sufficient milk for two or three infants. If a wet nurse is brought into a home, her baby should accompany her. This will relieve her of anxiety over the welfare of her own infant and such peace of mind is conducive to an abundant milk supply. The woman's own infant, if strong and vigorous, will stimulate the flow of milk by vigorous nursing.

In the choice of a wet nurse, it is not at all necessary that her baby should be the same age as the one who is to be fed, as breast milk does not change greatly in composition after the first month until the end of the lactation period. The wet nurse should be in good health. She should be free from tuberculosis and if there is the least suspicion of syphilis, the Wassermann reaction should be performed. She should be cleanly in her habits and her own baby should be in good health, this being a very good indication of the character of the milk. It is immaterial whether she be white or black, as biologic characteristics are not transmitted by way of the milk. Colored women are more likely to have an abundant supply of milk than white. On the other hand, they are more prone to tuberculosis and are likely to be living on poor diets so that the milk may be deficient in quality. The infant who is to be fed by the wet nurse may either nurse the breast directly or be given expressed milk from a bottle. Direct nursing of the breast is preferable unless the infant is very weak, as more milk is usually taken in this way and there is less chance for contamination.

In order that an infant may receive breast milk, it is not absolutely necessary that a wet nurse should be taken into the household. Milk may be expressed from the breasts of the wet nurse and delivered to the home. In some cities "breast milk dairies" have been established, which are distributing centers for breast milk. The chief disadvantages of such a system are that the milk is likely to become contaminated and there is difficulty in preserving it. To overcome the latter difficulty, collected breast milk may be desiccated or sterilized. At best such procedures are a makeshift substitute for maternal nursing. With an increasing knowledge of infant nutrition, the need for wet nursing has become progressively less.

Fig. 6.—Manual expression of milk, showing method of grasping the breast.

Fig. 7.—Manual expression of milk. The hand is pushed toward the chest wall and the fingers and thumb are brought together forcibly.

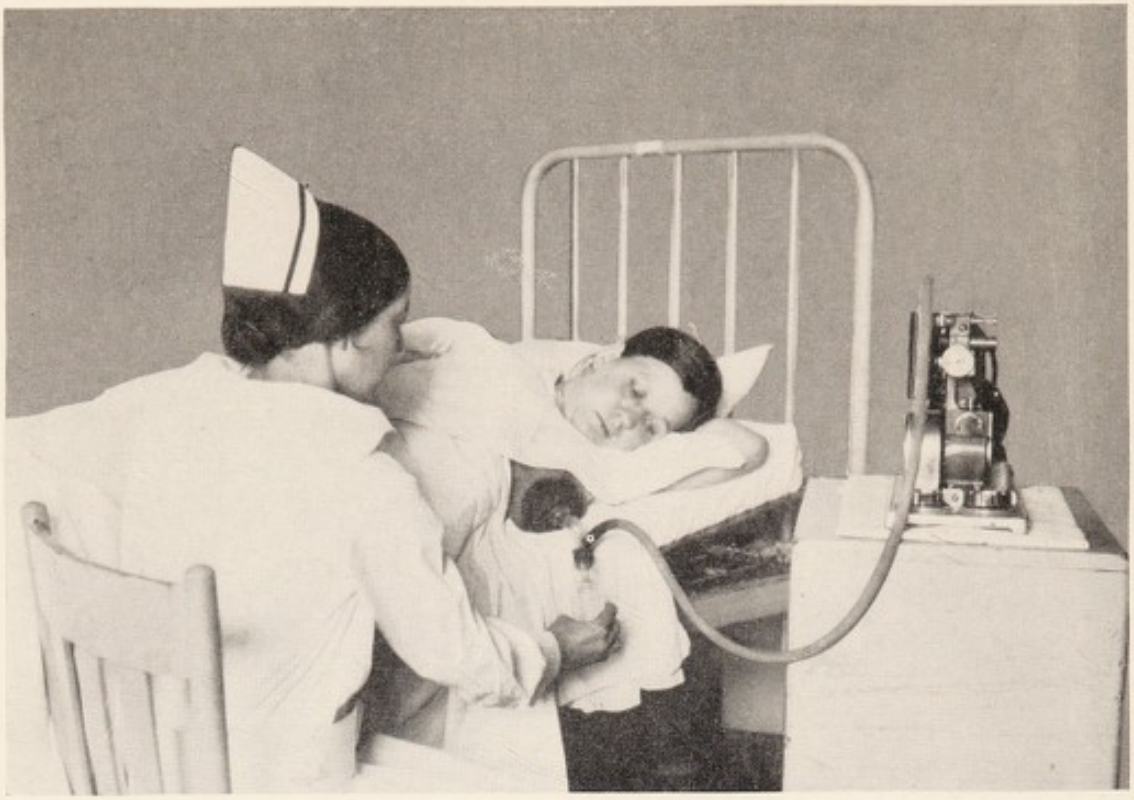


Fig. 8.—The Abt electric breast pump.

Fig. 9.—The Kieth hydraulic breast pump.

Most infants deprived of the mother's milk may be fed artificially with as great success as with collected breast milk. Premature infants are occasionally an exception. In general, wet nursing is now rare in private practice and wet nurses are not employed as often in children's hospitals as was the case a few years ago.

Manual or Mechanical Expression of Breast Milk

There are a number of conditions under which it is necessary or desirable to express the milk from the breasts. Complete emptying of the breasts is the best stimulus to milk secretion, and when the infant is unable to drain the breasts because of weakness or mechanical defects, forcible expression of the milk often serves to maintain or to increase the amount of secretion. It is particularly during the early months of the infant's life that mechanical expression of the milk is indicated.

When the nipples are fissured or infected the infant should not be nursed, but the milk should be drawn off. Mechanical emptying of the breasts is also sometimes indicated in the case of engorged or caked breasts. The milk of a wet nurse may be expressed for the purpose of feeding infants.

Milk may be expressed manually or by the use of a variety of mechanical appliances. Manual expression, when properly performed, is very effective, and the technic is not especially difficult. The principle of manual expression is to empty the milk sacs which hold the accumulated secretion. These sacs lie *behind* the nipples so that mere pulling, squeezing or stripping of the nipple is not sufficient. The proper technic is for the mother to grasp the breast about an inch and a half from the nipple with the fingers below and the thumb above. Firm pressure is then made backward against the breast and with the hand in this position the fingers and thumb are brought together forcibly. This motion should be repeated from 60 to 100 times per minute. The fingers should not be allowed to slip forward. The milk is caught in a sterilized glass, which is held in the other hand. The glass need not touch the breast, as the milk is forced out in streams to a considerable distance. (Figs. 6 and 7.) A mother who is skillful in the manual expression of milk can often remove a considerable amount of milk from the breast after the infant has obtained all that he can.

Breast pumps are of several varieties. The common glass type with collapsible rubber bulb is not very efficient and its use is often

painful. Electrical or water pressure pumps are, however, very effective. The electrical breast pump of Abt* has found wide use, especially in hospital practice. The principle is that of an intermittent sucking motion, the funnel placed at the breast being of such shape that compression of the breast occurs coincident with the suction (Fig. 8).

An inexpensive pump which is almost as effective and very suitable for home use is the water power breast pump of Kieth.† This instrument depends upon the use of a water suction pump attached to an ordinary bathroom faucet (Fig. 9). The technic of its use is simple and may be carried out by the most ignorant mother.

*The Abt pump is obtainable from Edward Lasker, Wrigley Building, Chicago, Illinois.
†The Kleth pump is obtainable from the A. S. Aloe Co., St. Louis, Mo.

CHAPTER XII

ARTIFICIAL FEEDING

General Considerations

When an infant is deprived of human milk, the natural substitute is milk from some other species. Cow's milk is usually employed in this country, but the milk of goats or other animals is occasionally used. Infants have even been fed successfully on mixtures containing no milk at all.

The milk of each species differs from that of others in the relative amounts of the organic and mineral constituents. There is also variation in the caloric value, in physical properties, and in the degree of digestibility. If one has a complete knowledge of the nutritional requirements and the digestive capacity of the infant, it is possible to prepare feedings from cow's milk or from the milk of some other species which meet all of the requirements and which are within the limits of capacity of the infant's digestion. It is also possible to render the feedings free from harmful bacteria by sterilization. In the preparation of artificial feedings, there is always the chance that some essential constituent of the diet may not be present in adequate amount or that the feeding as a whole may be deficient in caloric value. There is the additional danger of bacterial contamination after the feedings have been prepared.

The earliest attempts at infant feeding consisted merely in the giving of cow's milk or the milk of some other animal in such amounts as the infant would take. It was found that some infants could be raised successfully in this way, but many succumbed to gastrointestinal infections or nutritional disturbances. Most of the high mortality among artificially fed infants in the past has undoubtedly been due to bacterial contamination of the food. Before the advent of modern methods of milk production and preservation, much of the milk obtainable was badly contaminated and this was especially the case during the warmer seasons of the year. It has not been until comparatively recent times that the importance of bacterial contamination of milk has been appreciated.

Failure of infants to do well on artificial feeding was originally believed to be due primarily to differences in the composition of cow's milk which rendered it undigestible and otherwise unsuitable for the needs of the infant. Attempts at modification of milk by dilution or the addition of various substances met with relatively little success until the factor of bacterial contamination was considered and methods adopted to insure the absence of harmful bacteria. It was found when infants were fed on milk obtained under sanitary precautions and used fresh, that good results were obtained in a much larger proportion of cases than when unclean and carelessly handled milk was used. Subsequently it was found that boiled milk was better tolerated even than clean raw milk, but that infants fed on clean, boiled cow's milk without other modification did not thrive so well as those fed on human milk.

Many attempts have been made to explain the causes of failure in the case of artificially fed infants. Almost every constituent of cow's milk has been incriminated as the causative factor in explaining this difference. For many years it was assumed that the high protein content of cow's milk rendered it less suitable for the human infant, and attempts were accordingly made to lower the protein content of the milk by dilution or by removal of the casein by precipitation. These procedures probably did make the milk somewhat more digestible because when milk is diluted or has a portion of its casein removed, it forms smaller curds in the infant's stomach. On the other hand, the procedures adopted necessarily resulted in a decrease in the fuel value of the milk so that many infants fed in this way were underfed and consequently failed to gain in a normal manner. It was believed by some that the protein of cow's milk, being a foreign protein, was not as suitable for the infant as protein from the same species.

The fact that some infants did not do well even when the protein was reduced in the milk led to the belief that perhaps the fat might be a disturbing factor. Formulas were accordingly prepared from partially skimmed milk or occasionally from milk which was not only skimmed but also diluted. Such modified milk was found to be digested without a great deal of difficulty but the infants failed to thrive because of the low caloric value of the feedings. To overcome this latter difficulty, additional carbohydrate was added. Mixtures of this type led to better results because of the higher food values, but rarely to as good results as in the case of breast-

fed infants. It was noted that some infants receiving large amounts of sugar, developed diarrhea which ceased when the sugar was omitted. Accordingly it was postulated that an excess of carbohydrate was the primary cause of many of the nutritional disturbances of artificially fed infants, so entirely different types of formulas were devised in which the carbohydrate was reduced by dilution or separation of the whey, the protein and fat being undisturbed except for dilution. It was found that a certain number of infants could be fed satisfactorily in this way, provided a sufficient total volume of food was given.

The underlying idea of all of the earlier methods of modification of cow's milk for infant feeding was that some harmful substances were present which must be reduced in amount or removed. This conception culminated in the comprehensive system of Rotch, usually referred to as the "percentage method" of feeding. The theoretical basis of this system was that the nutritional disturbances of artificially fed infants resulted from the harmful effects of either fat, carbohydrate or protein; that an excess of each of these elements caused characteristic disturbances and that the remedy lay in the reduction of the harmful element or elements in the diet. Reduction of protein was effected by dilution of the milk, the fat and sugar contents being restored by additions of cream and milk sugar. The appearance of certain symptoms called for reduction of either the sugar or fat, in addition to reduction of protein. Fat alone was reduced by removal of the top portion of the milk. In intelligent hands such methods of modification of milk have led to successful feeding results, but all too often misinterpretation of symptoms has resulted in a reduction of the food elements to a point below the nutritional needs of the infant, and as a consequence infants have been fed digestible but insufficient food.

It has long been known that the curds produced by the action of acid or rennin on cow's milk are larger and tougher than those from human milk and this is generally believed to be one of the factors in rendering cow's milk less digestible. Dilution of the milk with water leads to smaller curd formation. Dilution with cereal gruels or lime water is more effective still in reducing the size of the curds. Heating of the milk to high temperatures changes the physical properties of the casein so that the curds produced are smaller.

Certain other physical properties of cow's milk have also been implicated as factors in rendering it less digestible. Cow's milk normally is very slightly more acid than human milk, much larger amounts of acid, however, are required to bring it to the degree of acidity attained during gastric digestion. This high "buffer value" of cow's milk is believed by some to interfere with the processes of digestion by neutralizing gastric acid. Based on this theory, cow's milk is modified by the addition of various acids to neutralize the buffer substances.

In contradistinction to the older theories that cow's milk contains an excess of some harmful substance, there is the more recent viewpoint that cow's milk is deficient in certain essential constituents, such as mineral salts and vitamins.

Without question, all of the theories which have been advanced to explain the relative unsuitability of cow's milk for the feeding of the human infant contain certain elements of truth and every attempt at modification based upon the various theories has served to increase our knowledge of the nutritional needs and digestive functions of the infant and has brought us nearer to the solution of the problem of satisfactory artificial feeding. There is no one method of artificial feeding which is the only correct one. Infants may be fed artificially in a variety of ways and good results obtained. On the other hand, any system, unintelligently used, results in numerous failures. No matter what method is used for the preparation of the formulas, certain essential requirements must be fulfilled.

The Requirements of a Satisfactory Artificial Feeding

The nutritional requirements of the infant are fairly definitely known and the capacity of the infant to digest the various food elements either singly or in combination is also known. The effects of bacteria introduced by way of the milk are understood, as are the methods for rendering the milk free from harmful bacteria.

With our present knowledge it is possible to formulate the essential requirements of satisfactory artificial feeding. These requirements may be stated as follows:

(1) Sufficient calories.

(2) Sufficient protein, carbohydrate, mineral salts, water and the vitamins A, B, C and D (fat and pigment are desirable but not absolutely essential).

(3) There must be no harmful bacteria present.

(4) The food must be digestible.

Any form of food which meets the above requirements will be successful whether it be prepared from cow's milk or mother's milk, whether the milk be in the fresh liquid form, evaporated or dried; whether it be sweet or sour, or whether there be no milk at all present. It will make little difference whether the basis of the diet be a proprietary food obtained from the drug store or simply milk and sugar obtained from the grocery store. Good results may be obtained provided only that each of the above requirements are met. Failure to meet any one of these requirements will result in failure of the feeding as a whole.

Only too often, a food which is readily digestible is so dilute or given in such small quantities that an insufficient number of calories are provided. Attempts to render the feeding digestible may result in the giving of too little protein with consequent small gain in weight and poor nutritional results. This is the common fault of many of the high carbohydrate proprietary foods which contain relatively little milk. A formula suitable in respect to all elements may be contaminated by harmful bacteria. In the effort to meet all the requirements, a food may be prepared which is quite beyond the capacity of digestion of the infant. So much attention, however, has been centered on the digestibility of the food that this error is not one which is frequently made.

If one keeps the essential requirements clearly in mind, it is not difficult to prepare a satisfactory artificial feeding adapted to the needs of the individual infant. A clear understanding of these fundamental principles is also of the greatest aid in determining the causes of unsuccessful feeding. A review of the history of "difficult feeding cases" usually reveals the fact that previous feedings have failed to meet one or more of the essential requirements. Once having determined the essential error or errors, correction of the discrepancy may be expected to remedy the feeding difficulties. It should, however, be pointed out that improper feeding is only one of the causes of the failure of infants to thrive. Only too often blind efforts are made to find a suitable formula for an infant who is not thriving when the real difficulty is organic disease or undiscovered infection. If the feeding is one which meets all the requirements, it may confidently be assumed that

some other factor is at fault and a careful search should be made to determine this disturbing factor.

The requirements as to calories, protein, carbohydrate, mineral salts, water, vitamins, fat and pigment have been considered in detail in Chapters II to VII and are summarized in Chapter VIII.

Bacterial Contamination of the Infant's Food

Harmful bacterial contamination of milk may be prevented by scrupulous care in production, distribution and handling. Certified milk is the cleanest form of raw milk available and is usually free from harmful bacterial contamination. It is, however, safer in all instances to subject milk or milk formulas used for infant feeding to a sufficient degree of heat to insure destruction of any pathogenic organisms. Pasteurization of milk, when properly carried out, is an effective method of rendering it safe from the bacterial standpoint. Evaporated milk is absolutely sterile and dried milk is usually, but not always, free from harmful organisms. Condensed milk is not sterile. The prepared baby foods contain varying numbers of organisms, depending upon the care taken in the processes of manufacture. Acidification of milk mixtures to a sufficient degree results in inhibition of bacterial growth and tends to prevent contamination after formulas have been prepared.

For a more detailed discussion of the bacteria of milk and of the methods of reducing bacterial contamination, see Chapter XIII.

Milk is not the only source of bacterial contamination of the infant's food. Contamination may occur from the use of impure water, from lack of cleanliness of bottles and nipples or from accessory foods.

Digestibility of Milk Formulas

Raw cow's milk is less easily digested by the infant than is human milk. The reasons for this have been discussed elsewhere. (Chapters IX, XI, and XIII.) Cow's milk which has been heated is more digestible than raw milk. The higher the degree of heat and the longer continued, the more digestible it becomes; hence, boiled cow's milk is more easily digested than pasteurized, and evaporated milk more digestible than boiled milk. Dried milk

is intermediate in digestibility between boiled and evaporated milk. Acidified milk is more digestible than sweet milk because of the formation of smaller curds and the lower buffer value. Diluted cow's milk is more digestible, volume for volume, than undiluted because of the smaller amount of milk present and the smaller size of the curds formed.

It is not possible to state precisely how much cow's milk an infant can digest, nor is it possible to state accurately the relative digestibility of different forms of modification of milk. From clinical and laboratory investigation, however, it may be stated in very general terms that boiled cow's milk is approximately two or three times as difficult to digest as human milk. One-third or one-half cow's milk is, therefore, about as digestible as human milk. Acidified, undiluted cow's milk approximates human milk in digestibility. The same may be said of unsweetened evaporated milk diluted with an equal volume of water so as to render it the approximate equivalent of whole milk. Acidified evaporated milk is more digestible than unacidified. Whole dried milks are intermediate in digestibility between boiled milk and evaporated milk, and partially skimmed dried milks are more digestible, in proportion to the amount of milk present, than whole dried milk. Goat's milk is about as digestible as cow's milk.

The capacity of the infant to digest cow's milk varies a great deal with age and physical conditions as well as with the character of the previous feeding. Infants who have been fed cow's milk from an early age become accustomed to it and are able to digest more than infants who have previously received only human milk. In general, the average normal infant, during the first month of life, is capable of digesting about one-half as much boiled cow's milk as human milk; or in other words, can take a volume of half diluted cow's milk equivalent to the volume of breast milk ordinarily taken. Very weak or premature infants cannot take more than one-third as much cow's milk; or, in other words, one-third diluted milk. By the age of four months, an average infant accustomed to artificial feeding may be expected to digest well two-thirds diluted whole milk and by seven or eight months, undiluted milk. There is a good deal of variation in digestive capacity, some infants during the first month or two of life being able to take undiluted boiled cow's milk with impunity. Most normal infants

can take acidified whole cow's milk, or half-diluted evaporated milk (equivalent to reconstituted whole milk) as early as the first month of life.

Partially skimmed milk is somewhat more readily digested than whole milk. Carbohydrates of the dextrin and maltose types are more digestible than cane sugar or milk sugar. Starch is incompletely digested during early infancy, but rarely leads to digestive disturbances.

CHAPTER XIII

COMPOSITION AND CHARACTER OF COW'S MILK

All cow's milk is not the same. The chief variations are in the fat content and in the number and character of contaminating microorganisms. The milk from Holstein and other common breeds of cows is best suited for infant feeding, its composition being more uniform and its fat content lower than that of highly bred cows, such as Jerseys or Guernseys. There is some individual variation in the milk of cows of the same breed and of the same cow at different times. The mixed milk from a herd is far more uniform in composition than the milk from a single cow. Furthermore, the effect of any deleterious changes in the milk of any one cow in the herd is minimized by dilution of this milk with that of the remainder of the herd. Milk which is distributed by large dairies in cities is usually more uniform than country milk or milk from small dairies because of the fact that it is "standardized" to a definite fat content, which is usually just a trifle above the minimal legal requirements. The amounts of the other elements in milk, the protein, sugar and mineral salts, are remarkably constant. The average composition of dairy milk is

Fat 3.5 to 4 per cent
Lactose 4.5 per cent
Protein 3.5 per cent
Minerals 0.75 per cent

The average caloric value of cow's milk is 20 calories per ounce, or 650 calories per liter. Further details as to composition are given in Table IV, page 96.

The fat of cow's milk exists in somewhat coarser emulsion than that of human milk. The emulsion is much finer, however, if the cow's milk has been "homogenized." The chemical composition of the fat of cow's milk differs from that of human milk in that there is a larger proportion of the more difficultly digestible tripalmitin and tristearin and less of the readily digestible triolein. There is also a larger proportion of the glycerides of the irritating

volatile fatty acids. In general, the fat of cow's milk is somewhat less completely absorbed from the gastrointestinal tract and is somewhat more likely to produce gastrointestinal irritation than an equal amount of the fat of human milk.

The sugar of cow's milk is identical chemically with the lactose of human milk, although the total amount present is less.

The protein of cow's milk is mostly (85 per cent) casein, the remainder being whey protein (lactalbumin and lactoglobulin). The characteristics of these two proteins have been considered elsewhere. (See Chapters III and IX.)

The casein (caseinogen) is present in a fine suspension, but is precipitated as a tough gelatinous curd by the action of rennin and acid. The curd from diluted milk is finer than that from undiluted milk. The curd from milk which has been heat-treated is smaller than from raw milk. The greater the degree of heat, the smaller the curd. Only very small and fine curds may be precipitated from evaporated milk. The curds from dried milk are intermediate in texture between those of boiled and evaporated milk. The addition of such protective colloids as starch gruels to milk results in the precipitation of a finer curd. Gelatin has some effect on the size of the curd, but is relatively inefficient as a means of modifying curd formation. The addition of lime water, sodium bicarbonate or sodium citrate causes some modification of the size of the curd. The slow addition of acid to milk, whether produced by bacterial fermentation or added in dilute form, causes precipitation of fine casein curds. Strong acid, added rapidly, throws down large curds.

The mineral constituents of cow's milk are more abundant than those of human milk and exist in different proportions. There is more calcium, magnesium and phosphorus present in cow's milk and less iron. (See Table IV, page 96.)

All four of the essential vitamins, A, B, C and D, are present in cow's milk. The amounts of the fat soluble A and D vitamins present vary with the type of feeding of the cow and the time of the year. The amounts present may or may not be sufficient for the needs of the infant. The B vitamin is present in sufficient amounts, there being usually three or four times as much as in human milk. Raw cow's milk may or may not contain sufficient C vitamin to protect the infant against scurvy. Heat-treated milk, whether pasteurized, boiled, dried or evaporated, contains minimal

amounts of this vitamin and therefore cannot be depended upon to protect against scurvy.

The "buffer value" of cow's milk, that is, the capacity to absorb considerable quantities of acid and alkali without significant change in chemical reaction, is much higher than that of breast milk. It is necessary to add approximately three times as much acid to cow's milk in order to bring it to the optimum point for gastric digestion as in the case of human milk.

The Enzymes of Milk

Raw cow's milk always contains a number of enzymes of various types. Some of these are proteolytic, resembling either trypsin or pepsin in their action. Carbohydrate splitting and fat splitting ferments are also present as well as oxidizing and reducing enzymes. There is a question as to whether these enzymes are natural, normal constituents of the milk or whether they are derived from the bacteria present. Available evidence would indicate that bacteria are the chief source of the enzymes. The enzymes in question exist in too small quantities to be of any practical significance in the subsequent digestion of milk by the infant. They are all destroyed by heating and this destruction does not make the milk any less digestible.

Miscellaneous Constituents of Milk

Cow's milk occasionally contains substances capable of causing disturbances in those drinking the milk. When cows are first turned out to pasture in the spring, and overeat, the milk occasionally causes gastrointestinal disturbances in infants. Certain poisonous plants may be eaten by cows, the most dangerous of which is probably snakeroot. The harmful substance in this is secreted into the milk and gives rise to the disease known as "milk sickness." Epidemics of milk sickness are mostly seen toward the end of a dry summer when the grazing has become poor and cows eat plants which they would not ordinarily touch.

Bacteriology of Cow's Milk

The bacteria of cow's milk vary in character and number, depending upon the source of the milk and the conditions under which it is kept before use. Freshly collected milk contains relatively few bacteria (100 to 1000 per c.c.). Milk, however, is an

excellent culture medium for most bacteria so that such microorganisms as may be accidentally introduced multiply rapidly unless the milk is kept at a low temperature. Milk is easily contaminated by stable dirt, unclean utensils or the hands of milkers.

When milk is produced under clean conditions, kept at temperatures below 50° F. during transportation and delivered to the consumer without unnecessary delay, the bacterial count ordinarily varies from 10,000 to 50,000 per c.c. Milk having a bacterial count of less than 50,000 is ordinarily considered of good quality. Milk which has not been obtained under cleanly conditions, or which has not been cared for properly, may, on delivery, have a bacterial count of several million. The bacterial count of milk serves as an indication of the care taken in its production. Milk with a high bacterial count is not necessarily harmful to health as the bacterial flora may be entirely innocuous. On the other hand, milk may have a low bacterial count, but contain pathogenic organisms and therefore be unsuitable. In general, milk with a high bacterial count is likely to contain numerous types of organisms, some of which may lead to serious digestive disturbances. It has been shown by actual statistics that infants fed on market milk of low bacterial counts are less subject to gastrointestinal diseases than those fed on milk with high bacterial counts.

In general, the bacteria of milk fall into three main groups:

(1) **Harmless, Lactic Acid Producing Organisms.**—These, under ordinary conditions, make up well over 90 per cent of the total bacterial flora of raw milk. Included in this group are the B. acidophilus, B. bulgarius, Streptococcus lacticus, Micrococcus ovalis, and B. lactis aërogenes (B. acidi lacti). Strains of the colon bacillus of bovine origin, growing in a medium rich in sugar ordinarily produce only lactic acid and other harmless products; other strains, however, growing under different conditions, may produce harmful products.

The harmless acid producers, when growing in milk, tend to overgrow other types of organisms because of the fact that they are, as a group, more resistant to the acid they produce than are most pathogenic bacteria. The harmless lactic acid producers grow at room temperatures, whereas pathogenic organisms usually do not multiply rapidly except at incubator temperature, hence the importance of keeping milk cold.

(2) **Harmful Saphrophytic Bacteria.**—These organisms decompose milk to form products which are capable of causing gastrointestinal disturbances. It is not necessary that the organism invade the body and cause specific infectious disease. Many of the organisms of this type have marked proteolytic properties. Examples of this group are certain strains of B. coli, B. faecalis alkaligenes, B. enteritidis, a group of organisms described by Flugge and certain spore bearers, including the gas bacillus.

Among the harmful saphrophytes should probably be included certain cryo-flora or organisms which grow only at very low temperatures; and which in consequence develop in frozen milk. The products of the growth of these organisms are believed to be one of the factors in causing the gastrointestinal disturbances sometimes observed when infants are fed on milk which has been frozen. The offending substances, however, seem to be destroyed in greater part by subsequent boiling of the milk.

(3) **Pathogenic Organisms** capable of producing specific infectious disease.

Included in this group are typhoid, paratyphoid, dysentery, hemolytic staphylococci, B. tuberculosis, B. diphtheriae and B. abortus (very similar to or identical with B. melitensis).

In raw milk organisms of the lactic acid group usually grow rapidly and greatly outnumber all other types—indeed, the numbers of the latter may diminish or they may almost completely disappear when a certain degree of acidity is reached. For this reason milk which tastes sweet may contain more harmful organisms than milk which is definitely sour. When milk is pasteurized most of the lactic acid producing organisms are killed, whereas certain proteolytic spore bearers survive. When pasteurized milk is poorly refrigerated, the growth of putrefactive organisms is not greatly checked by acid production, so that spoiled pasteurized milk may be more dangerous than sour raw milk.

The bacteria present in milk are not evenly distributed. They tend to become attached to the fat globules so that the cream often has a higher bacterial count than the remainder of the milk. This is especially true of gravity cream. When cream is separated by centrifugalization, the bacteria tend to be thrown down in the sediment.

Methods for Reduction of the Bacterial Count of Milk

Milk intended for infant feeding should be free from pathogenic bacteria and from potentially harmful saprophytes. There are a number of methods by which the harmful organisms in milk may be reduced in number or eliminated:

1. Prevention of contamination.
2. Heat treatment to destroy such organisms as may accidentally have gained entrance.
3. Acidification.
4. Use of preservatives.

It has been shown that if extraordinary precautions are taken in the production of milk, contamination with harmful bacteria is unlikely to occur. Milk produced under the strictest sanitary conditions, known as *certified milk,* can usually be depended upon to be safe from the bacterial standpoint. Destruction of harmful or potentially harmful organisms by heat of varying degrees is a safer means of insuring against bacterial contamination. The application of a moderate degree of heat, known as pasteurization, destroys practically all known pathogenic organisms and over ninety per cent of all organisms present in milk. Boiling of milk effects a more complete sterilization than pasteurization, but alters the taste of the milk more. Autoclaving the milk at temperatures above the boiling point, as in the preparation of unsweetened evaporated milk, brings about complete sterilization. The drying of milk is about as effective as pasteurization, although occasionally pathogenic organisms escape the drying process, or the product is contaminated subsequently.

Acidification of milk, by implanting it with active lactic acid producing organisms or by the addition of a sufficient amount of acid renders it a poor culture medium for most pathogenic bacteria.

Chemical preservatives such as formaldehyde, benzoic acid or hydrogen peroxide have occasionally been used to lower the bacterial content of milk. The use of such preservatives is very generally prohibited by law, although it is doubtful whether the small amounts of preservatives necessary for the purpose are really harmful.

Certified Milk

Certified milk is the purest form of raw milk obtainable. It is produced in accordance with regulations prescribed by the American Association of Medical Milk Commissions. The regulations require that the barns in which the cattle are milked shall be of such construction that they may be easily cleaned, that they be screened and that there be adequate ventilation. The cows must all be tuberculin tested at regular intervals and must be free from other diseases as determined by veterinary examination. The milkers must be free from disease and milking done with scrupulous care as to cleanliness. After drawing, the milk must be cooled immediately to below 45° F. and kept below this temperature until delivered. The milk must be bottled at the dairy and must at the time of delivery contain less than 10,000 bacteria per c.c.

Certified milk is necessarily expensive, the cost being usually twice that of ordinary milk. Even with the extreme precautions used in the production of certified milk, contamination is possible. It should, therefore, be boiled before being used for infant feeding. At one time certified milk was the only really clean milk obtainable in cities and was consequently largely used for infant feeding. With the improvement of the general milk supply, the almost universal introduction of pasteurized milk, and the availability of sterile preserved milk, the need for certified milk is not so great.

Pasteurized Milk

Pasteurization consists in heating milk to a temperature of 140° to 145° F. and holding it at this temperature for thirty minutes. It is then immediately cooled and kept cold until delivery. It is possible to pasteurize milk only when it is reasonably fresh, as milk which is sour is curdled by the pasteurization process. When pasteurization is properly carried out and the milk is protected against subsequent contamination, harmful bacteria are not present. If the pasteurizing process is incomplete, the temperature being too low or the milk not being held at the required temperature for a sufficient length of time, pathogenic bacteria may escape destruction. Furthermore, unless care is taken in the cleanliness of the pipe lines and bottles, contamination subsequent to pasteurization may occur. Pasteurized

milk, bottled at the dairy, is much less likely to be contaminated than that dispensed from large cans or bottled by the storekeeper or milk wagon driver. In general, pasteurized milk is far safer than ordinary grades of raw milk and properly pasteurized milk is as safe as certified milk. It should, however, be boiled before being fed to young infants. Pasteurization of milk affects the taste slightly, but not to the same extent as boiling. The heating causes some precipitation of insoluble calcium phosphate, but this loss is of no practical importance. The only vitamin seriously affected by pasteurization is the antiscorbutic or C vitamin.

Boiled Milk

Boiling of milk serves much the same purpose as pasteurization, but is somewhat more effective in destroying such organisms as may be present. Boiling also destroys the true bacterial toxins. Boiling of milk for three minutes is sufficient to render it safe from the bacterial standpoint. Boiling affects the taste of milk and also causes the formation of a scum on the surface which consists of coagulated lactalbumin, calcium phosphate, and a few enmeshed fat globules. Less of a scum is formed if the milk is heated in a closed receptacle or in a double boiler instead of over a free flame. The scum formed on the milk contains very little of value from the nutritional standpoint and should be removed before the milk is fed. Boiling, like pasteurization, leads to some precipitation of calcium phosphate and brings about destruction of the C vitamin, but this latter may readily be supplied by the addition of orange juice or tomato juice. Boiled milk is more digestible than raw milk because of the change in the physical character of the protein. It is popularly supposed that boiled milk is constipating in its action, probably because diarrhea is less frequent in babies fed on boiled milk. In view of the distinct advantages of boiled milk, it is advisable that all formulas prepared from sweet cow's milk, whether certified or pasteurized, should be boiled before feeding.

CHAPTER XIV

FEEDING OF THE NORMAL INFANT WITH WHOLE SWEET MILK MIXTURES

The fundamental nutritional requirements of normal infants may be met in most instances by the use of simple mixtures of whole sweet cow's milk, sugar and water. There are, however, certain limitations to the use of such mixtures.

Infants vary considerably in their ability to digest cow's milk. Many are able to take undiluted boiled cow's milk in amounts to satisfy the appetite without suffering from digestive disturbances. Others with lower digestive capacity are unable to take such large amounts of cow's milk so that the milk needs to be diluted. Dilution of milk serves the purpose of giving the infant less milk without at the same time too greatly limiting his fluid intake. Most normal infants can take at least one-half as much cow's milk as human milk at a feeding during the first month, so that the cow's milk usually need not be diluted more than with an equal volume of water. By four months, most infants can take two-thirds cow's milk and by seven or eight months, undiluted cow's milk in customary quantities. In very hot weather or when the infant's digestive capacity is impaired by the presence of infection, smaller amounts of sweet cow's milk have to be given. Not infrequently the infant's digestive capacity is so low that the cow's milk given must be diluted for a considerable portion of the first year. Such dilute milk mixtures have a low caloric value and there is danger of underfeeding unless large volumes are given at a feeding and frequent feeding intervals instituted.

In the preparation of the whole milk formulas, certain general principles should be followed but there will necessarily be variations for individual infants dependent upon the digestive capacities and appetite. The amount of whole milk given should approximate 1.5 to 2 ounces to each pound of *expected* body weight. The amount of carbohydrate added should bear a definite relationship to the amount of milk, preferably one of carbohydrate to about eleven of milk. This proportion of carbodydrate may be added

with safety provided a sugar of the maltose dextrin type is used. When cane sugar or lactose is used the proportion of sugar may have to be reduced somewhat.

The amount of water to be added will depend upon the degree of dilution required to render the mixture digestible and upon the total volume of food taken.

The total volume given at a feeding should be regulated more by the child's appetite than anything else. The bottle-fed infant should not be expected to take the same amount at each feeding any more than the infant nursing at the breast. Nature is often a better guide as to the food requirements than man-made rules. In the case of the sick infant there may, of course, be definite contraindications to the giving of unlimited amounts of food.

The total calorie value of the food should approximate 55 calories per pound of expected body weight. At times it may not be possible to give this much food and still keep within the limits of digestive capacity. Under such circumstances, it will be necessary to resort to the use of acid milk or evaporated milk. These types of feeding are discussed subsequently (Chapters XV and XVI).

Construction of Formula

In laying out the formula, the first step is to decide how many feedings are to be given and how much at a feeding. During the first month or two of life six feedings are usually necessary in the case of infants fed on whole milk mixtures, later five or even four feedings, depending upon the degree of concentration of the milk and the infant's rate of gain. The more concentrated the feeding mixture, the fewer the feedings need be. An infant who will gain well on four or five feedings a day need not be fed more often.

The feedings should preferably be given at four-hour intervals. (For discussion of feeding intervals, see Chapter XI.)

In preparing the feedings, it is well to put a larger volume in each bottle than the average amount that the infant usually takes, so that when he is especially hungry he will have sufficient. There is little danger of overfeeding in this way provided the formula is a reasonable one. This practice tends to duplicate conditions when the infant nurses at the breast.

In starting an infant on a feeding with sweet cow's milk, it is best to give a mixture that is somewhat dilute until the infant's

capacity for digestion has been determined. After that the concentration may be increased. The application of these principles can best be illustrated by a specific example:

Normal infant, one month old, weight 8 pounds. (Birth weight 7½ pounds.) Previously breast fed.

Six bottles a day should be given at four-hour intervals. The usual volume taken at a feeding at this age will be in the neighborhood of four ounces. The minimum amount of milk needed at 1.5 ounces per pound of body weight is 12 ounces. The proper amount of sugar in proportion to the milk used is approximately one ounce. The addition of 12 ounces of water brings the total volume up to 24 ounces, which is sufficient for the six bottles of four ounces each. The caloric value of the feeding is readily calculated as follows:

12 oz. of milk	at 20 calories per ounce	=	240 calories
1 oz. of sugar	at 120 calories per ounce	=	120 calories
	Total		360 calories.

The formula will then be

Milk	12 oz.	240 calories
Sugar	1 oz.	120 calories
Water	12 oz.	
Total	24 oz.	360 calories.

6 bottles, 4 ounces each.

This formula is adequate in most respects. It provides sufficient milk with its protein and mineral salt content and sufficient carbohydrate. It may be expected to be within the limits of capacity of digestion of the infant, being one-half diluted milk. It should be rendered free from harmful bacteria by boiling. The feeding is somewhat low in caloric value, providing only 45 calories per pound. The formula would, however, be a safe one on which to start and the infant might possibly gain on it.

If the infant fails to gain and shows signs of hunger, it is necessary to increase the calories. This is accomplished by adding more milk and proportionately more sugar. The amount of milk may be increased, for example, to fourteen ounces and the sugar to 1¼ ounces. The total volume of the mixture may be allowed to

remain the same by decreasing the amount of water, provided the infant does not show a desire to take more than four ounces at a feeding, or on the other hand the water may be somewhat increased if the infant readily takes a larger volume. The formula would now be:

Milk	14 oz.	280 calories
Sugar	1¼ oz.	150 calories
Water	10 oz. to 13 oz.	
Total	24 to 27 oz.	430 calories.

6 bottles, 4-4½ ounces each.

This would probably give the infant adequate calories and also meet all of the other requirements with the possible exception of the vitamins, this latter requirement being met by the addition of cod liver oil and orange juice to the diet as described elsewhere (Chapters VII and VIII).

If the infant shows no digestive disturbances on such a mixture and gains weight, no change need be made for a few weeks until the infant's food requirements increase with increase in weight. If, on the other hand, the infant shows a reluctance to take as large a volume at a feeding, yet shows no evidence of digestive disturbance, the amount of water in the formula is decreased, the proportions between milk and sugar, however, being constantly maintained.

By the age of two months, the infant might normally be expected to have attained a weight of 9½ to 10 pounds and might be given the following formula:

Milk	16 oz.	320 calories
Sugar	1½ oz.	180 calories
Water	7 oz.	
Total	23 oz.	500 calories

5 bottles, 4½ ounces each.

This supplies the necessary amount of milk and carbohydrate and also calories. If the infant shows no digestive disturbances, there would be an advantage in continuing this more concentrated formula and giving only five feedings instead of six, provided good

gains in weight were being made. If, on the other hand, the infant shows digestive disturbance, there is an advantage in giving a more dilute formula containing the same amounts of milk and sugar, but more water, for example:

Milk	16 oz.	320 calories
Sugar	1½ oz.	180 calories
Water	10 oz.	
Total	26 oz.	500 calories

6 feedings, 4½ ounces each.

This more dilute mixture would give the infant less milk to digest at each feeding. The curds would also be somewhat smaller. The same number of calories and the same total amount of milk per day would be taken.

By the age of four months, the infant might be expected to weigh about 13 pounds, and a suitable formula would be:

Milk	22 oz.	440 calories
Sugar	2 oz.	240 calories
Water	8 oz.	
Total	30 oz.	680 calories

5 bottles, 6 ounces each.

The total volume taken would, of course, be regulated by the manifest need for food.

By the age of six months, when the weight has increased to about 16 pounds, a suitable formula would be:

Milk	26 oz.	520 calories
Sugar	2 oz.	240 calories
Water	9 oz.	
Total	35 oz.	760 calories

5 bottles, 7 oz. each.

At the age of six months, cereal feeding is begun, the infant being given from one to three tablespoonfuls of well-cooked cereal gruel, twice a day (barley, farina, cream of wheat or oatmeal). This is given before the 10 A.M. and 6 P.M. feedings. Some of the milk mixture is mixed with the cereal and after finishing this the bottle is offered.

Some time during the fifth or sixth month, one or two tablespoonfuls of well-cooked, strained vegetable purée is given daily. The yolk of an egg is also added to the milk mixture. Stewed fruit may be given once daily.

After cereals and vegetables are added to the diet, the proportions of sugar may be decreased gradually, the starch taking the part of the sugar. By seven months, the proportion between milk and sugar need not be higher than 1:15 or 1:20 and by eight months, 1:30. At one year of age no sugar need be added to the milk, the infant taking from 28 to 32 ounces of boiled whole milk a day in four feedings, this being supplemented by cereals, vegetable purées, egg yolk and meat broth.

From the above it will be seen that no hard and set rules need be followed in the feeding of a normal infant. One must be guided by the appetite, the digestion and the rate of gain.

So far as the milk mixture is concerned, it may be stated that the basic formula used during the first six months of life consists essentially of whole milk and sugar in the proportions of 11 of milk to one of sugar. This basic formula may be prepared by the addition of 1½ ounces of sugar to one pint of milk, or 3 ounces to the quart. The formula thus prepared is diluted with varying proportions of water, depending upon the age and digestive capacity of the infant. It is never necessary to add more than two volumes of water to one volume of milk and for most infants even during the first month of life a proportion of one of water to one of the basic formula is a suitable dilution. As the infant becomes older, the proportion of water is gradually decreased until at four months the proportion is one of water to two of milk; by six months it may be one of water to three of milk. At about six months of age starch partially replaces sugar as the carbohydrate addition, hence the basic formula is then changed to one of sugar to fifteen of milk, or one ounce to the pint; and from the ninth to the twelfth month one of sugar to twenty or thirty of milk. By the eighth month an infant may be expected to digest the milk formula well without the addition of any water. It will be seen that the formulas expressed in this simple way correspond to those constructed on the basis of the total food requirements and the digestive capacity at various ages.

When formulas are prepared in this way normal well infants may usually be given about as much as they desire. The more

dilute the formula, the larger amounts must be taken and in the case of some infants it is impossible to give a sufficient amount of dilute formula unless the feeding intervals are shortened. It is better, in such cases, to use a different type of milk mixture. It is especially during the early months of life that it is difficult to introduce sufficient food when formulas of this type are used; later there is little difficulty.

Tables giving the exact proportions of milk and the amounts taken by infants at different ages mean very little and are likely to give an erroneous impression. This is especially true in so far as the amounts taken at an individual feeding are concerned. The following table is given merely to indicate initial feedings which may be offered to infants at varying ages. The proportions of milk and carbohydrate given are those usually found most suitable and the total amount of food designated is just about sufficient in caloric value and in protein to cover the average require-

TABLE V

OUTLINE FOR THE FEEDING OF NORMAL INFANTS WITH WHOLE SWEET MILK MIXTURES

AGE	WHOLE MILK	WATER	SUGAR	FEEDINGS
1 week	6 oz.	6 oz.	1 oz.	6 × 2 oz.
2 weeks	10 oz.	8 oz.	1 oz.	6 × 3 oz.
1 month	14 oz.	10 oz.	1¼ oz.	6 × 4 oz.*
3 months	18 oz.	7 oz.	1½ oz.	5 × 5 oz.
4 months	22 oz.	8 oz.	2 oz.	5 × 6 oz.
6 months	26 oz.	9 oz.	2 oz.	5 × 7 oz.†
8 months	32 oz.	–	1 oz.	4 × 8 oz.
12 months	32 oz.	–	–	4 × 8 oz.

*In all cases the above formulas are to be supplemented with orange juice and cod liver oil from the first month on.

†Cereals, green vegetables and egg yolk are added at the age of six months.

ments at the different ages. Many infants will take more concentrated mixtures and many will take larger volumes at a feeding. In all cases the formula should be made to fit the requirements of the infant and the infant should not be expected to conform to a table.

Technic of Preparation of the Formula

All utensils used in the preparation of the formula should be scrupulously clean and preferably boiled before use. In preparing the formulas the total day's feedings are made up at one time. The water is first measured and poured into a clean vessel. The sugar is then measured out or weighed and dissolved in the water.

The sugar may be measured in a tablespoon or a graduated medicine glass; the latter is preferable as tablespoons are not uniform in size. In measuring the dried sugar preparations, the spoon should be filled without compressing and leveled with a knife blade. In measuring in a medicine glass the sugar should be shaken down by gentle tapping until level with the proper graduation on the glass. The different sugar preparations vary considerably in density so that a tablespoonful of one may weigh more than a tablespoonful of another. The approximate equivalents are as follows:

Cane sugar, 2 level tablespoonfuls = 1 ounce
Maltose dextrin mixtures, 4 level tablespoonfuls = 1 ounce
Milk sugar, 3 level tablespoonfuls = 1 ounce

Instead of measuring the dried sugars, they may be weighed on a letter scale or small balance.

In measuring corn syrup, two tablespoonfuls or one ounce by volume contains one ounce of sugar. This should be measured by pouring the syrup into the tablespoon and then rinsing the latter off in the water. The spoon should not be dipped into the can as an extra amount of the syrup will adhere. A very handy method of measuring corn syrup is first to mix the syrup with an equal volume of boiled water. This thin syrup may be bottled and kept for several days in a refrigerator. Two ounces by volume, or four tablespoonfuls of the thin syrup is equivalent to one ounce of carbohydrate.

After dissolving the proper amount of sugar in the water, the milk is stirred in. The milk should be taken from a fresh bottle which has been mixed by inverting several times.

The mixture of milk, water and sugar is then boiled for three minutes in a saucepan or heated in a covered double boiler for twenty minutes after the water in the lower portion begins to boil. The milk is allowed to cool somewhat while covered, the scum is removed, and the formula is then poured, by means of a small funnel, into clean sterilized bottles, one for each feeding. The bottles are stoppered with nonabsorbent cotton, corks, or covered with pieces of clean tissue paper secured by rubber bands. The bottles are cooled by immersion in running water and placed in the refrigerator until used.

The formula may be sterilized in the bottles instead of in a saucepan. This is a somewhat preferable method. In carrying out this procedure the formula is poured into individual bottles which are stoppered with nonabsorbent cotton and placed on a circular rack in a large covered pail containing two or three inches of water. The bottles remain in the pail for thirty minutes from the time the water in the bottom begins to boil.

The Nursing Bottle

Any form of bottle that may be cleaned easily is suitable. Ordinary small-mouthed bottles are preferable to the wider-mouthed variety, as they are more readily stoppered and the nipples are less expensive and more easily cleaned. Bottles made of "pyrex" glass are less apt to crack when heated than are those made of ordinary glass.

After each feeding the empty bottle should immediately be rinsed and filled with water. Later the bottle is scrubbed with hot soap suds and a bottle brush, rinsed and boiled for five minutes. The nipples should be washed with warm soap and water, rinsed and kept in a covered jar in a boric acid solution. Boiling the nipples after each feeding is not necessary except in hospital practice. The nipples should, however, be boiled occasionally, but frequent boiling rapidly destroys the rubber.

Technic of Feeding

The bottle should, before being offered to the baby, be warmed to body temperature by immersing in warm water. The temperature of the milk can best be tested by allowing a few drops to fall on the inner side of the wrist.

The hole in the nipple should be of such size as to allow the milk to drop rapidly, but not to flow in a large stream. A small hole may be enlarged by burning with a hot needle.

The bottle should be held for the baby throughout the feeding. It is best also that the baby be held in a semi-upright position, so as to facilitate the escape of swallowed air. The infant should not be forced to take more food than he wants. When the hole in the nipple is too large, so that the milk fairly pours into the infant's mouth, he may have to swallow to keep from suffocating and thus take more food than he should. On the other hand, the

infant should not be allowed to nurse more than twenty minutes from the bottle. Unless the infant is very weak or the hole in the nipple so small that the food is obtained with great difficulty, an infant who is really hungry will take all the food he needs within ten or fifteen minutes. At the conclusion of each feeding the infant should be held over the mother's shoulder and patted on the back in order to get rid of swallowed air. It may be necessary to hold the infant for as long as fifteen minutes before the air is belched up.

CHAPTER XV

EVAPORATED MILK MIXTURES

Unsweetened evaporated milk possesses certain distinct advantages over ordinary whole sweet milk for the feeding of infants. The chief advantages are the readier digestibility, uniformity of composition and sterility. Evaporated milk formulas are also more easily prepared and consequently there is less chance of error or contamination.

Characteristics of Evaporated Milk

In the preparation of evaporated milk, fresh mixed herd milk is heated in a partial vacuum until about sixty per cent of the water is removed. This concentrated product is homogenized; which results in the breaking up of the fat globules to almost colloidal size, so that they remain distributed in the milk and do not rise to the surface as cream. The concentrated, homogenized mixture is transferred to cans, sealed and then autoclaved at a temperature of about 240° F. This brings about complete sterilization. The milk appears to keep almost indefinitely in sealed cans. The composition of evaporated milk is constant within narrow limits and is controlled by government regulations. For practical purposes in infant feeding evaporated milk may be considered as having twice the strength of cow's milk; one volume of evaporated milk mixed with one volume of water giving reconstituted milk which is just a little more concentrated than fresh mixed herd milk.

TABLE VI

COMPOSITION OF EVAPORATED MILK

	FAT	SUGAR	PROTEIN	MINERAL	CALORIES PER OZ.
Evaporated milk	7.85	10.0	7.0	1.6	43.0
Evaporated milk and water 1 to 1	4.0	5.0	3.5	0.8	21.5
Evaporated milk and water 1 to 2	2.6	3.3	2.3	0.5	14.5

The fat of evaporated milk is in very finely divided form and for this reason more readily digestible. The sugar is unchanged

by the heating process. The protein is considerably altered so that when the milk is curdled by rennin or acid extremely fine curds are formed resembling those from breast milk. This is an important factor in rendering the milk digestible. The lactalbumin as well as the casein is partially denatured by heating so that it is not so likely to produce anaphylactic phenomena in infants sensitized to cow's milk.

The heat to which evaporated milk is subjected during the process of sterilization causes certain changes in the mineral constituents. The salts of the whey are not altered, but a small portion of the calcium phosphate is precipitated, the change being similar to that occurring in boiled milk.

The buffer value of evaporated milk closely approximates that of natural milk, being if anything slightly less.

The vitamin content of evaporated milk is about the same as of boiled milk. The C vitamin appears to be completely destroyed, the other vitamins are practically unaffected and present in approximately the same amounts as in raw milk.

Observations on a large number of infants and older children fed for prolonged periods on evaporated milk have convinced us that this form of milk is, from the nutritional standpoint, the full equivalent of pasteurized or boiled cow's milk, and that its continuous use does not lead to the development of any nutritional disturbance. Furthermore it is the safest form of milk from the bacteriologic standpoint. All things considered, evaporated milk is the most suitable form of milk at present available for the feeding of most infants. It has the further advantages that it is economical and is a nonproprietary product. Unsweetened evaporated milk should not be confused with *condensed* milk, which is prepared with the addition of approximately 45 per cent of cane sugar (see Chapter XVII).

Construction of Formulas With Evaporated Milk

Evaporated milk diluted with an equal volume of boiled water may be used for the preparation of formulas in the same manner as whole cow's milk, the formulas being constructed as described in Chapter XIV.

Instead of diluting the milk first with water and then using it as whole milk, the formula may be constructed using just half the

amount of evaporated milk as of whole milk and making up the final volume with water. For example, if the formula decided upon consists of

Whole milk	20 oz.
Sugar	2 oz.
Water	10 oz.

This may be made from evaporated milk, as follows:

Evaporated milk	10 oz.
Sugar	2 oz.
Water	20 oz.

This latter formula will have the same volume and the same caloric value.

Formulas corresponding to those recommended for whole milk dilutions may be used in the case of infants with limited digestive capacity in whom the whole milk formulas are not well tolerated. For most normal infants, however, evaporated milk may to advantage be used in considerably higher concentration than whole milk, most infants being able to take evaporated milk diluted with an equal volume of water throughout the first year, the proportions of sugar being those used in the case of whole milk; namely, one of sugar to eleven or twelve of half diluted evaporated milk during the first six months, one to fifteen of milk after the addition of cereal at six months of age, and one to twenty or thirty from the ninth to twelfth month. Such formulas have a high caloric value so that frequent feedings or large volumes at a feeding are not necessary. These formulas may not supply as much fluid as is needed during the warmer months of the year, so that additional water should be offered between feedings if the infant shows signs of thirst. There is, of course, no serious disadvantage in giving the formula somewhat more dilute provided a sufficient total amount is given to fully meet the infant's nutritional requirements. The following table indicates the way in which normal infants may be fed on evaporated milk mixtures.

This table is subject to the limitations outlined relative to the use of a similar table for whole milk dilutions. (See page 137.)

TABLE VII

OUTLINE FOR THE FEEDING OF NORMAL INFANTS WITH EVAPORATED MILK MIXTURES

AGE	EVAPORATED MILK	WATER	SUGAR	FEEDINGS
1 week	5 oz.	7 oz.	1 oz.	6 x 2 oz.
2 weeks	7 oz.	11 oz.	1¼ oz.	6 x 3 oz.
1 month	11 oz.	13 oz.	1½ oz.	6 x 4 oz.*
3 months	12 oz.	13 oz.	1½ oz.	5 x 5 oz.
4 months	15 oz.	15 oz.	2 oz.	5 x 6 oz.
6 months	17 oz.	18 oz.	2 oz.	5 x 7 oz.†
8 months	16 oz.	16 oz.	1 oz.	4 x 8 oz.
12 months	16 oz.	16 oz.	---	4 x 8 oz.

*In all cases the above formulas are to be supplemented with orange juice and cod liver oil from the first month on.

†Cereals, green vegetables and egg yolk are added at the age of six months.

Technic of Preparation of the Formula

As in the case of whole milk mixtures the total day's feedings are prepared at one time. The sugar is measured, as previously described, and dissolved in the requisite amount of boiled water. The required amount of evaporated milk is stirred in. The formula is immediately poured into the clean nursing bottles. No further sterilization is necessary. The technic of feeding and the feeding intervals are the same as in the case of whole milk mixtures.

Use of Evaporated Milk for the Preparation of Acid Milk Mixtures

Evaporated milk is especially suitable for the preparation of acid milk formulas. Formulas of this type are discussed in Chapter XVI.

CHAPTER XVI

ACID MILKS

When cow's milk is soured through the growth of lactic acid-producing organisms, or if lactic acid, acid fruit juices or other nontoxic acids are added to the point of curdling, certain changes are brought about in the milk which render it more digestible. The amount of acid necessary for this purpose is that corresponding to from 0.4 to 0.6 per cent of lactic acid, or the chemical equivalent of other acids.

Acidification of milk leads to the neutralization of a portion of the buffer substances present so that when the milk is fed the degree of acidity attained in the stomach approximates that when human milk is fed. This acidity is sufficient to inhibit bacterial growth, to favor normal functioning of the pyloric sphincter and to stimulate the flow of bile, pancreatic and intestinal juices. (See Chapter IX.) An additional important effect of acidification is the precipitation of casein curds in finely divided form. These fine curds do not enmesh bacteria as do larger ones, are more readily permeated by the digestive juices and leave the stomach more quickly than the large curds ordinarily formed when sweet milk is fed. When acid milk is fed, the contents of the stomach and of the upper portion of the small intestine are more acid than in the case of sweet milk feedings. This greater degree of acidity tends to render certain of the mineral constituents, especially the calcium salts, more soluble and hence favors absorption. The acids themselves are quickly absorbed so that the contents of the lower portion of the intestinal tract are likely to be strongly alkaline. This alkalinity is in part due to the lesser degree of carbohydrate fermentation because of inhibition of bacterial growth and in part to the increased secretion of alkaline intestinal juices.

The stools of infants fed on acid milk mixtures are characteristically firm, putty-like and alkaline in reaction. They are composed largely of lime soaps and calcium phosphate. They are light brown in color and the odor is foul rather than sour.

The organic acids which are used for the acidification of milk are well absorbed and practically completely metabolized to carbon dioxide and water. These acids are not excreted in the urine in appreciable amounts and there is no effect on the acid base balance. Mineral acids, on the other hand, such as hydrochloric acid, although well absorbed, are not burned in the body but must be neutralized and excreted. The continuous feeding of large amounts of inorganic acid such as hydrochloric necessarily leads to some disturbance of the acid base balance and may deplete the alkali reserves of the body. The small amounts customarily added to milk, however, are not likely to be harmful.

Because of the fact that acid milk is a poor culture medium for most bacteria, formulas prepared from acid milk keep better than sweet milk formulas, even though the milk be poorly refrigerated. Acid milk mixtures are less likely to become bacterially contaminated even when prepared by careless persons and put in bottles that are not entirely clean.

When acid milk mixtures are used, it is rarely necessary to dilute the milk inasmuch as the digestibility of acid milk approximates that of human milk. The caloric value of formulas made from undiluted milk being considerably higher than that of the customary sweet milk dilutions, there is much less danger of underfeeding. Mixtures of this type are of especial value for the feeding of infants who have small gastric capacities and who vomit when large volumes of food are taken. Acid milk mixtures are also of value in the treatment of undernourished infants having large food requirements and low digestive capacities. There is but little danger of overfeeding with acid milk formulas because of the fact that they are so readily digestible and likely to be free from harmful bacteria.

There are but few disadvantages in the use of acid milk. Older infants who are accustomed to sweet milk may, at the start, refuse sour-tasting milk, and infants who are partially breast-fed do not take acid milk as readily as sweet milk when given as supplemental or complemental feedings. A few infants secrete an abnormally acid gastric juice. In the case of these infants the use of acid milk may be a disadvantage and lead to vomiting. Such cases are, however, rare. For the majority of infants acid milk is preferable even though sweet milk may be well tolerated. There is no objection to continuing the use of acid milk beyond the period of infancy.

Lactic Acid Milk

Of the various forms of acid milks, that soured with lactic acid has had the widest use. Lactic acid milk feeding is not new. Buttermilk has been used in infant feeding for at least one hundred and fifty years and probably longer. It was found empirically that infants with diarrhea often tolerated buttermilk better than other forms of food.

Buttermilk is essentially skimmed lactic acid milk and a by-product in the manufacture of butter. Cream is allowed to sour spontaneously or the souring is hastened by the addition of a "starter" or culture of lactic acid-producing organisms, and the fat subsequently removed by churning. Buttermilk prepared in this way is likely to be relatively free from harmful bacteria because of the high degree of acidity but cannot be depended upon as being entirely safe.

The lactic acid milk used at the present day for infant feeding is not a by-product of the butter industry but is an especially prepared food. For the feeding of most infants the entire fat content of the whole milk is retained. Two methods of preparation of lactic acid milk may be used:

(1) Souring by acid producing bacteria.
(2) Addition of chemically pure lactic acid.

Bacterially Soured Milk

Lactic acid milk may be prepared by incubating pasteurized or sterilized milk with pure cultures of lactic acid-producing organisms such as B. acidophilus or the Bulgarian bacillus. Since the chief objective is the formation of a sufficient amount of lactic acid, it is rather immaterial what type of organism is used provided it is one which is nonpathogenic and produces no harmful products. The B. acidophilus produces acid from lactose more slowly than the Bulgarian bacillus, but on the other hand it is more resistant to acid and brings about ultimately a higher concentration of acid in the milk. Bulgarian bacilli die out quickly in the intestinal tract whereas the B. acidophilus when fed survives to a certain extent especially when fed with dextrin. It is possible that the continued growth of the B. acidophilus in the intestinal tract may exert some beneficial action, but any such effect is difficult to detect clinically.

In most larger cities dairies prepare bacterially soured whole and skimmed milk which is satisfactory for infant feeding. Such milk usually costs about five cents per quart more than ordinary pasteurized milk.

Bacterially soured milk may be prepared in the home, making use of cultures obtainable on the market. Before attempting to prepare the acid milk, the culture to be used should be tested and a stock culture prepared. This is done by boiling a few ounces of milk in a saucepan, cooling to body temperature and adding to this a small amount of one of the commercial cultures either liquid or tablet form. The inoculated milk is poured into a sterile bottle, stoppered loosely and allowed to stand in a warm place for at least twelve hours. If the milk is curdled at the end of this time it indicates that the culture is active.

In the preparation of the acid milk, one quart of milk is boiled and cooled to about 80° F. One tablespoonful of the milk previously prepared with the culture as described above is mixed in and the whole poured into a clean quart milk bottle, covered with a sterile piece of cheesecloth and allowed to stand in a warm place overnight; or the warm mixture may be poured in a clean vacuum bottle, stoppered and allowed to stand. The vacuum bottle serves to keep the milk warm. If the milk is incubated at too high a temperature, organisms other than lactic acid producers may grow and impart to the milk undesirable characteristics. Too long a period of incubation results in a product which is excessively acid.

Properly prepared cultured acid milk should have a creamy consistency; there should be no large curds present and no separation of whey. The odor should be pleasantly sour but not rancid. Milk which contains bubbles of gas or large curds separated from the whey is not suitable for infant feeding. Properly prepared acid milk contains from 0.4 to 0.6 per cent lactic acid. The curds of the bacterially soured milk are finer than those prepared by the direct addition of acid.

Use of Lactic Acid U.S.P. for the Preparation of Lactic Acid Milk

Lactic acid milk may readily be prepared by the addition of lactic acid to sweet milk. The acid milk so prepared does not contain the lactic acid organisms, but the presence of these latter

does not appear to be of any great advantage. The curds of acidified milk are not so fine as those of bacterially soured milk but from the practical standpoint acidified milk appears to be just about as effective as that which has been bacterially soured.

In the preparation of acidified milk, lactic acid U.S.P. (85 per cent) is added to whole milk in the proportion of 1½ drams to the quart (6 c.c. per liter). This is equivalent to four or five drops of lactic acid to each ounce of milk. (Drops of lactic acid are small, there being approximately 100 drops to the dram.) Pasteurized or preferably boiled milk should be used and in any event the milk must be thoroughly cold before the acid is added. The requisite amount of acid is mixed with one to two ounces of water and this dilute acid solution is poured slowly into the cold milk with constant stirring. At first no effect on the milk is observed, but as the last portions of the acid are added, curdling begins. It is especially important that the last portions of acid be added very slowly as it is only in this way very fine curds will be formed. The final product resembles bacterially soured milk in taste and odor but on standing the curds tend to separate. Unless the milk is thoroughly cooled before the acid is added, or if the acid is added too rapidly the curds are likely to be large and clumpy and the mixture not suitable for feeding.

The amount of lactic acid recommended above is sufficient to render the milk approximately as acid as milk which has been soured by bacterial action. This is a suitable proportion of acid for most infant feeding purposes. Larger amounts should usually not be added as more strongly acid milk is not well taken and is occasionally vomited. Lesser amounts of acid are not so effective in neutralizing buffer and in bringing about the formation of small curds, but milk containing as much as one-half the proportion of lactic acid recommended is nevertheless considerably more readily digested than sweet milk.

In starting infants on lactic acid milk feedings, it is sometimes desirable to increase gradually the amount of lactic acid added, beginning with one-third or one-half the full amount. The infant thus becomes accustomed to the sour taste and does not object as when fully acidified milk is fed at the start. When the proportion of acid is so small that no curd formation occurs, the milk is likely to curdle in the stomach in a manner similar to sweet milk.

Lactic Acid Milk Mixtures From Evaporated Milk

Lactic acid milk is readily prepared from unsweetened evaporated milk and the product is more digestible than that prepared from ordinary milk. It is not necessary to add quite so much lactic acid to evaporated milk. The optimum proportion is one dram to the quart of half diluted evaporated milk (4 c.c. per liter). This is equivalent to three drops of acid to each ounce of the half diluted evaporated milk, or 6 drops per ounce of undiluted evaporated milk used in the formula. In making acid milk from the evaporated product, sterilization is unnecessary, one simply mixes the milk with the proper amount of water and stirs in the acid. This may be done more rapidly than in the case of ordinary milk as the curds are small in any event.

A very convenient method of making the lactic acid milk mixtures from evaporated milk is to first add the acid to the requisite amount of water and sugar and then pour this into the milk. (See page 152.)

Dried Lactic Acid Milk

A number of the preparations of dried lactic acid milk are on the market. These are made by drying milk which has been bacterially soured or acidified by the addition of lactic acid. The best known preparations are Mead's Powdered Lactic Acid Milk, made by the Mead Johnson Co., and the Powdered Whole Lactic Acid Milk of the Merrell-Soule Co. These preparations, when mixed with the proper amount of water, make a whole lactic acid milk which is entirely suitable for infant feeding. Directions for dilution are given by the manufacturers.

Construction of Lactic Acid Milk Formulas

Whole lactic acid milk, that is, acid milk, containing the full fat content of whole milk, is so readily digestible that it may be fed without dilution to most infants from the time of birth. There is no especial objection to a moderate degree of dilution, but it should be remembered that one of the chief advantages of acid milk is that it may be fed in such concentrated form that an adequate caloric intake is assured.

The Standard Formula

Sugar is added to lactic acid milk in the same proportions as to sweet milk, that is, for infants under six months of age the

proportion of sugar to milk should be approximately one to eleven, or 1½ ounces of sugar to the pint of milk. This formula has a caloric value of approximately 30 calories to the ounce and is one which we have used as a standard formula in hospital and dispensary practice for the feeding of over 90 per cent of infants under six months of age.

After the sixth month, when the infant begins to take cereals and green vegetables the proportion of sugar may be reduced to one to fifteen, or one ounce to the pint. The caloric value of this formula is approximately 26 calories per ounce. After the ninth month the proportion of sugar may be reduced to one to twenty, or one to thirty (½ to ¾ ounce to the pint).

In feeding these concentrated formulas not more than five feedings a day are usually necessary during the first month of life. Later many infants obtain sufficient food on as few as four feedings a day. The feedings are offered at four-hour intervals except during the night, and the infant is allowed to take about as much as his appetite dictates. In the case of infants who have previously been underfed, it is desirable to give more feedings in the day, up to five or six, until normal weight is approximated, at which time the infant's appetite is likely to diminish. The fact that infants need relatively small volumes of formulas of this type to meet their needs often leads one to the erroneous supposition that the infants do not relish the acid milk mixtures.

There is little if any danger of overfeeding with these mixtures in the case of normal infants and there are relatively few conditions which render a change in the formula necessary. The same formula is used for months at a time, the only change being in the volume taken, and that being determined by the infant's increased appetite as he grows older. The formulas are of such type that individualization is perfectly possible but such individualization is not usually necessary. The statement is frequently made that no two babies can be fed in the same manner and that the formula must be individualized in each case. This may be true when certain types of formulas are used, but it is not true in the case of formulas which meet the fundamental requirements. In this connection one should not lose sight of the fact that human milk, which is admittedly the best food for a normal infant, does not vary appreciably in composition throughout the

first year, the only changes in the feeding of the breast-fed infant being in the total amounts taken and the additional foods which are added to the diet.

Technic of Preparation of Lactic Acid Milk Formulas

In preparing the lactic acid milk formulas all that is necessary is to stir the sugar into the acid milk. No further sterilization is necessary. Acid milk mixtures cannot readily be boiled.

In preparing the acid milk mixtures from evaporated milk, a practical method is first to mix the acid, sugar and water in the proper proportions and then add this solution to the evaporated milk. In order to make the formula recommended for the first six months of life, an acid-sugar solution is first prepared, as follows:

Acid-Sugar Solution

Karo Syrup 3 oz.	6 tablespoonfuls
Lactic acid, U.S.P.	1 teaspoonful
Water to make up to	1 pint

The syrup is dissolved in a portion of the water, the lactic acid added and the whole made up to volume. This acid-sugar solution, on account of its high acidity, keeps well even at room temperatures, the only organisms growing being an occasional yeast. Enough solution may be made up at one time to last for several days. When the acid-sugar solution is mixed with an approximately equal volume of evaporated milk, the product is equivalent to whole lactic acid milk with sugar added in the proportion of one to eleven, or 1½ ounces to the pint.

The feedings for the entire day may be prepared at one time, in which case the contents of a one pound can of evaporated milk are mixed with one pint of the acid-sugar solution, or single feedings may be prepared at a time by opening a small can of evaporated milk, pouring into the feeding bottle a suitable amount and then adding an equal volume of the acid-sugar solution. The whole is shaken and is then ready for feeding, after warming. This latter method of preparation is especially advantageous when traveling or when adequate refrigeration facilities are not available.

From the sixth to the ninth month the composition of the acid-sugar solution is changed by reducing the amount of sugar to two ounces (or 4 tablespoonfuls) to the pint. After the ninth month only one ounce of sugar to the pint is used. After one year of age both sugar and acid may be omitted.

In feeding lactic acid milk mixtures to infants, the bottles must not be warmed to too high a temperature, as this may cause precipitation of large curds. As acid milk formulas are much thicker than those made from sweet milk, it is essential that the hole in the nipple be enlarged; this may readily be done by burning with a hot needle.

Other Forms of Acid Milk

Acids other than lactic may be used for acidifying milk and comparable feeding results obtained.

A. Hess and Matzner have suggested the use of lemon juice as an acidifying agent, the amount of juice used being approximately 3 drams per pint (21 c.c. per liter). The lemon juice is slowly stirred into the milk. Milk prepared in this way is not quite so acid as the usual lactic acid milk, but sufficiently acid to render the milk very digestible. An additional advantage of lemon juice is that it contains the antiscorbutic vitamin, so that supplements of orange juice to the diet are unnecessary.

Orange juice in the proportion of one ounce to the pint has been recommended by Julius Hess. This gives a very slightly acid mixture, but one which is agreeable to take. This also contains the antiscorbutic vitamin.

Pure citric acid has been used for the purpose of acidifying milk by Weisenberg, Meyer and Behrens in Germany and by Gonce in this country. The proportion used is 4 grams of dehydrated citric acid to the quart of milk. This amount of acid may be dissolved in a little water and then stirred into the milk. Much less care is necessary than in the preparation of lactic acid milk mixtures, as large curds are not readily formed, even though the acid is added rapidly. The curds from milk produced by the addition of citric acid or the citric acid containing fruit juices are usually smaller than those formed by the addition of lactic acid.

Orange juice milk is usually fed diluted to about the same extent as sweet milk; lemon juice milk diluted one-third; and citric acid milk is fed undiluted in the same amounts as lactic acid milk.

Acetic acid in the form of vinegar or acetic acid U.S.P. has been used by Dunham, one ounce of cider vinegar, or one dram of 36 per cent acetic acid U.S.P. being slowly stirred into a pint of cold milk.

Faber advises the use of hydrochloric acid as an acidifying agent, the proportion being 4 ounces of decinormal hydrochloric acid to the pink of milk.

Acetic acid milk and hydrochloric acid milk may be fed undiluted.

CHAPTER XVII

SPECIAL AND PROPRIETARY FOODS

The nutritional requirements of most infants may be met fully by the use of simple mixtures of sweet or acid milk and sugar supplemented with cod liver oil, orange juice, cereals, vegetables and egg yolk. Other types of food are not necessary for the feeding of normal infants, although it is possible to feed normal infants in a variety of ways.

A number of food mixtures have been devised for the feeding of normal infants and many of these are entirely satisfactory in practice, provided the fundamental nutritional requirements are met.

The special types of food used for the feeding of normal infants may be divided into several classes:

1. Carbohydrate preparations.
2. Processed milks.
3. Carbohydrate-milk mixtures.
4. "Complete" foods.

The Carbohydrate Preparations

The types of carbohydrates used in infant feeding have been discussed in Chapter IV. Despite the large number of names of individual products, the only carbohydrates used in infant feeding are milk sugar, cane sugar, starch and the starch derivatives, dextrin, maltose and dextrose. The names given to certain forms of carbohydrates, especially the dextrose and maltose mixtures, are such as to give the impression that these are foods possessing unique properties. Mellin's Food, Horlick's Maltose Dextrin Milk Modifier, Denno's Food, Allenberry's Food No. 3, Horlick's Malt Food, and Ridge's Food are merely mixtures of the simple carbohydrates, starch, dextrin, maltose, cane or milk sugar.

Processed Milks

Certified, pasteurized, and evaporated milk have already been considered. Dried milk represents another distribution form of milk which is suitable for infant feeding.

Two methods for the preparation of dried milk are used. In one, whole pasteurized milk is sprayed into a chamber through which a stream of heated air at a temperature of 180° F. is passed. This abstracts the moisture from the spray and the dried milk falls to the bottom of the chamber as a fine, flaky powder, which is collected and marketed in sealed tins. A well-known example of milk prepared by this process is "Klim" made by the Merrell-Soule Co. The same process of drying has been applied to skimmed milk, acid milk, and protein milk.

The second method of manufacture of dried milk is the Just-Hatmaker process. In this process fresh milk is allowed to flow in a thin layer over steam-heated revolving drums, the dried flakes of milk being scraped off. The best known form of milk prepared by this process is "Dryco" of the Dry Milk Co. In the preparation of Dryco approximately one-half of the fat is removed from the milk before drying.

The temperature to which dried milk is subjected is ordinarily sufficient to destroy pathogenic bacteria. The product is not always completely sterile but rarely contains pathogenic organisms. Partly skimmed dried milk keeps somewhat better than whole dried milk.

The heating and drying processes alter the casein of the milk so that the curds formed from it are finer than in the case of raw or pasteurized milk. The roller process is more effective in changing the character of the protein. It is uncertain how much the vitamin C content of milk is affected by drying. Some samples of recently dried milk appear to have this vitamin present in approximately the same concentration as in the original milk. The other vitamins in milk are not appreciably affected by drying. The composition of typical forms of dried milk is given in Table VIII.

TABLE VIII

COMPOSITION OF DRIED MILK

	FAT	SUGAR	PROTEIN	MINERALS	CALORIES PER OZ.
Klim	28	38	27	6	160
Dryco	12	44	34	7	130

4 level tablespoonfuls of Klim = 1 ounce
8 level tablespoonfuls of Dryco = 1 ounce

Dried milk is a suitable and safe form of milk for the preparation of infant feeding formulas. The dried milk may be made up with water to give the approximate equivalent of whole milk and used as such. For example, 4 tablespoonfuls of Klim stirred into 8 ounces of water gives the equivalent of whole milk.

The equivalent of whole milk cannot be constituted by the use of Dryco because of the reduced fat content. Very satisfactory feeding formulas, however, may be prepared from Dryco, sugar and water mixtures. The details of the preparation of these formulas are given by the manufacturers.

One valuable use of Dryco or of dried skimmed milk is in the preparation of concentrated milk formulas suitable for the feeding of infants who will take only small volumes at a feeding and yet who have fairly high caloric and protein requirements. In the feeding of premature infants with breast milk, better results are often obtained if some dried milk is added to the breast milk before feeding. The caloric value and protein content may be doubled in this way without rendering the milk undigestible. (See Chapter XXVII.)

Carbohydrate-Milk Mixtures

A large number of dried carbohydrate-milk mixtures are on the market. The best known representative of this group is malted milk, which is a mixture of dried milk, maltose and dextrin. Malted milk is essentially one-third dried milk and two-thirds carbohydrate. Mixtures of dried milk and lactose are also on the market. Most of these contain too high a proportion of carbohydrate to render them suitable as complete foods. If used at all it is necessary to add additional milk. Nestle's Milk Food is a mixture of dried milk, starch, dextrin, maltose, cane sugar, calcium phosphate, iron citrate, sodium iodide, sodium fluoride and cod liver oil concentrate. It is designed as an addition to milk. There would appear to be but little reason for the use of such a preparation.

There are on the market mixtures of dried lactic acid milk with dextrimaltose or corn syrup added in such proportion that when the milk is diluted a suitable lactic acid milk formula is obtained (Mead's Powdered Lactic Acid Milk with Dextrimaltose, Merrell-Soule's Powdered Lactic Acid Milk with Corn Syrup).

Sweetened Condensed Milk

Sweetened condensed milk is whole cow's milk evaporated to a little less than half of the original volume and preserved with the addition of 40 to 45 per cent of cane sugar.

Condensed milk is not sterile but the sugar content is sufficiently high to inhibit bacterial growth until diluted. Condensed milk is not a suitable food for infant feeding. When given in sufficient amount to supply the necessary calories, there is too little milk present to provide sufficient protein, mineral salts and vitamins. Such a one-sided diet may lead to rapid increase in weight, but infants so fed are as a class flabby and have very poor resistance to infections. Condensed milk is fairly digestible because of the very small amount of milk contained and because of the heat treatment to which it has been subjected. There are, however, other foods which are equally digestible and do not possess the same disadvantages as condensed milk. The average composition of sweetened condensed milk is

	FAT	LACTOSE	CANE SUGAR	PROTEIN	MINERALS	CALORIES PER OZ.
Condensed milk	8.0	11	43	8.0	1.8	100

"Complete" Infant Foods

A number of mixtures designed as complete infant foods, in addition to those already mentioned, are available. The most scientific of these is Synthetic Milk Adapted (S.M.A.) of Gerstenberger (The Laboratory Products Co., Cleveland, Ohio).

S.M.A. represents an attempt to provide a food having the chemical and physical properties of human milk. In the preparation of S.M.A. skimmed cow's milk is diluted with two volumes of water to lower the protein and fat content to the percentages present in human milk. A small amount of potassium salt is added and sufficient lactose to bring the percentage up to that present in human milk. Special fat in the proportion of 4 per cent is incorporated by homogenization. This special fat is a mixture of vegetable and animal fats with cod liver oil having a melting point and chemical constants approximating those of the fat of human milk. The cod liver oil provides for an adequate intake of the fat soluble vitamins A and D. The percentage composition of fat, protein and sugar in S.M.A. is the same as that of human milk

and the caloric value is 20 calories per ounce. It is recommended that S.M.A. be fed in about the same amounts as human milk, or approximately 2½ ounces per pound of body weight.

S.M.A., being one-third diluted milk, is digestible, and when given in sufficient amounts, the caloric needs of the infant are met. The protein content is low for a food containing only cow's milk protein. The rather high proportion of lactose and low proportion of protein gives the food somewhat more laxative qualities than the ordinary cow's milk dilutions. S.M.A. is conveniently marketed in powdered and in concentrated evaporated forms and is readily prepared for use.

"Similac" and "Lactogen" are preparations modeled after the S.M.A. pattern. Similac has been processed so as to change somewhat the character of the calcium combination with protein. The fat content of this consists of a mixture of homogenized butter fat, cod liver oil, cocoanut oil and olive oil. Lactogen is an homogenized dried mixture of top milk and lactose. Both of these preparations, when diluted with water, give products having approximately the percentage composition of human milk.

Butter Flour Mixture (Czerny-Kleinschmidt)

Czerny and Kleinschmidt have described the preparation of a milk formula in which the fat of cow's milk is modified by heating so as to reduce the content of volatile fatty acids. The fat thus altered is emulsified with flour and sugar and added to diluted milk. This type of feeding goes by the name of "Butter Flour Mixture" (Butter mehlernährung). In the preparation of butter flour mixture, a stock butter soup is first made, the composition of which is

Butter	2 level tablespoonfuls
Wheat flour	2½ level tablespoonfuls
Cane sugar	1 level tablespoonful
Water	10 ounces

The butter is melted in a frying pan and heated at a low temperature until foaming ceases and the odor of volatile acids has disappeared. At this time the butter usually turns a light brown color. The flour is then thoroughly stirred in and the mixture again heated for four or five minutes until the flour is brown. The

water and sugar are added and the mixture brought to a boil, while stirring. The caloric value of this butter soup stock solution is approximately 32 calories per ounce.

The butter soup stock is mixed with milk in the proportion of one of milk to two of stock solution for the feeding of young infants, or equal parts of milk and stock solution for the feeding of older infants. The caloric value of the final mixtures varies from 24 to 26 calories per ounce.

The proportion of fat is considerably higher than in whole milk, but the fat is in more digestible form. The starch, being partially dextrinized, is fairly well digested. Small curds are formed in the stomach from this mixture.

In the feeding of normal infants the amount of butter flour mixture required is approximately 2 to 2½ ounces to the pound of body weight. This meets the caloric requirements but does not provide for any too much protein. The high content of butter fat provides considerable A and D vitamins. Butter flour mixtures are tolerated well by many normal infants and by some undernourished infants. The butter flour mixture is likely, however, to induce diarrhea, especially during hot weather. Most of the good results obtained with butter flour mixture are to be attributed to its relatively high caloric value.

Foods Designed to Meet Special Conditions

Under certain conditions, special types of food are of value, as, for example, in the feeding of infants suffering from diarrhea or those who are sensitive to the proteins of milk.

Foods Used in the Treatment of Diarrhea

Protein Milk (Albumin Milk, "Eiweiss Milch").—Protein milk is a special type of milk mixture, designed by Finkelstein and Meyer for the feeding of infants suffering from diarrhea.

Protein milk is skimmed lactic acid milk to which has been added the curds from whole milk. It possesses the advantages of acid milks in having a low buffer value and small curds. It contains very little of the fermentable lactose and less of the whey salts than whole milk. The fat content is also lower. Protein milk is a poor culture medium for bacteria and is readily digestible. It has a high casein content, which favors the formation of firm stools.

In the preparation of protein milk, one quart of whole milk is warmed to body temperature, rennin is added and the milk allowed to stand until coagulated. The curd is cut into small squares with a knife and the whole transferred to a cheesecloth bag and allowed to drain for one hour. During this time the curds should not be squeezed as this forces out fat. The drained curd is turned out on a wire strainer and rubbed through with a wooden spoon or potato masher. One pint of lactic acid skimmed milk is mixed with the curds and the whole again run through the sieve. Water is then added to bring the total volume up to one quart. The average composition of protein milk prepared in this way is fat 2.5 per cent, sugar 1.5 per cent, protein 3.5 per cent. The caloric value is about 13 calories per ounce.

The preparation of protein milk is time-consuming and it is difficult to prepare a satisfactory product in the home.

Protein milk is prepared commercially on a large scale and marketed in dried form. Dried protein milk when mixed with a sufficient amount of water gives a product which is as satisfactory as freshly prepared liquid protein milk. Dried protein milks are made by the Mead Johnson and Co. and the Merrell-Soule Co. The product of Mead Johnson and Co. is so prepared that it can be boiled without curdling.

The use of protein milk in the feeding of infants suffering from diarrhea is discussed in Chapter XX.

Closely similar to protein milk is "Protein S.M.A. Acidulated" of the Laboratories Products Co. This represents a modification of S.M.A. for the treatment of infants with diarrhea.

Casein Preparations.—Special foods for the treatment of diarrhea similar to protein milk may be prepared by the addition of calcium caseinate to partially skimmed sweet or acidified milk. The effects of the addition of extra amounts of calcium caseinate to the diet are discussed in Chapter III. Examples of prepared soluble calcium caseinate are "Casec" (Mead Johnson Co.), "Larosan-Roche" (Hoffman LaRoche Chemical Works) and "Protolac" (The Dry Milk Co.).

Milk-Free Foods.—Some infants are sensitive to one or both of the proteins of cow's milk. In a certain number of instances infants who are sensitive to the proteins of cow's milk may be fed satisfactorily on goat's milk or on human milk, but still others

appear to be sensitive to all forms of animal proteins. When infants are unable to take any form of milk without developing symptoms, it becomes necessary to devise a milk-free diet. This presents difficulties but infants have been satisfactorily fed on various combinations. One of the most satisfactory methods of preparing a milk-free diet is by the use of soy bean flour, which contains a high percentage of the protein glycinin. It has been demonstrated that satisfactory growth is possible when soy beans furnish the sole source of protein. In order to prepare a complete food, to meet the requirements; carbohydrate and vegetable fats may be added. Hill and Stewart, of the Harvard Medical School, have devised a complete food using soy bean as a basis. This is composed of: soy bean flour, barley flour and olive oil, together with small amounts of sodium chloride and calcium carbonate. This preparation, designated "Sobee" is marketed by Mead Johnson and Co., and is recommended for use especially in the feeding of infants suffering from eczema.

Goat's Milk

Goat's milk is not widely used for infant feeding in this country, but in some countries and among certain classes of the population fresh goat's milk is more readily obtainable than fresh cow's milk.

Goat's milk contains about 0.5 per cent more protein than cow's milk, the extra amount being chiefly lactalbumin. The fat content averages 5 to 6 per cent. The lactose content is about the same as of cow's milk. Goat's milk has a caloric value of 23 to 25 calories per ounce. Goat's milk does not appear to be any more digestible than cow's milk. Goats are less susceptible to tuberculosis than cows, but, on the other hand, goat's milk is more likely to be contaminated by the organism causing undulant fever (B. melitensis, or Brucella abortus).

There are but few instances in which goat's milk is preferable to cow's milk for the feeding of infants. The chief indication for its use is in the case of infants who are sensitive to the proteins of cow's milk and develop asthma, urticaria, eczema or other allergic manifestations when cow's milk is fed. Some of these infants can take goat's milk without showing the manifestations; others, however, are found to be sensitive to goat's milk also. In the attempt to manage difficult feeding cases, goat's milk is not

infrequently used, but there is no good basis for this practice. Infants who are unable to digest cow's milk are not likely to digest goat's milk any better.

When goat's milk is used as the basis of the feeding, the method of preparation of the formula is the same as when cow's milk is used. Like cow's milk, goat's milk may be obtained in evaporated or dried form.

The chief disadvantage in the use of goat's milk is that a certain number of infants fed exclusively on goat's milk develop a rather severe degree of anemia. The anemia does not appear to be due to any deficiency of iron or other substance in goat's milk but seems to be due to the presence of an unidentified substance capable of damaging the blood-forming organs.

CHAPTER XVIII

THE FEEDING OF NORMAL INFANTS DURING THE SECOND YEAR

During the second year of life there should be three or four feedings in the twenty-four hours, given at approximately four-hour intervals throughout the day. Nothing should be permitted between feedings except water and the allowed quantity of fruit juice. Water should be given liberally, especially during warm weather.

The diet during the second year should consist of or be selected from the following:

1. **Milk.**—At least 1½ pints a day. Boiled milk should be used up until the time the infant is eighteen months old. After this time pasteurized or certified milk may be used. During hot weather it is safer to boil the milk even for older infants. Unsweetened evaporated milk diluted with an equal volume of water may be used instead of dairy milk. It is safer and more digestible.

2. **Cereal.**—Barley, oatmeal, corn meal, farina, cream of wheat, ralston, rice, grits or hominy. The cereals should be cooked in a double boiler for at least forty-five minutes. A little salt may be added to taste. The food value is greater if the cereals are cooked in milk or in equal volumes of milk and water. The cooked cereals are served with butter and milk. It is better not to add sugar. The ready cooked package cereals such as corn flakes, shredded wheat, and puffed grain are as readily digestible as the cooked cereals and lend variety to the diet.

3. **Vegetables.**—Spinach, carrots, peas, string beans, lima beans, tomatoes, cauliflower, cabbage and potatoes. The green vegetables should be thoroughly cooked in as small an amount of water as possible and the water used in the cooking should not be thrown away but fed with the vegetables as it contains mineral salts and vitamins. Potatoes may be boiled or baked, preferably the latter. The cooked vegetables should be mashed with a fork or in case the baby has a tendency to diarrhea, should be passed through a fine sieve. Vegetables may be served with a little butter, salt and

bread crumbs. Canned vegetables are satisfactory, especially the concentrated vegetable purées prepared for infant feeding.

4. **Eggs.**—Up to the age of fourteen months, the infant should receive the yolk of one egg daily except in cases where there is an idiosyncrasy to egg. The raw yolk may be mixed with the milk or it may be boiled and fed with a spoon. After the age of fourteen or fifteen months a whole soft boiled egg should be fed daily or alternated with meat feedings.

5. **Meat.**—Beef, lamb, mutton, or chicken. The meat should be scraped finely and in the case of beef, lamb or mutton, made into a pat and slightly broiled; this is served with a little salt and butter. Chicken should be boiled or roasted and cut into fine pieces. The amount of meat given at a meal should be from three to six teaspoonfuls.

Broth made from beef, lamb, mutton or chicken should be given every few days. Vegetables may be cooked into the broth. Broths for infants should not be highly seasoned.

6. **Fruits.**—One to three ounces of orange juice should be given daily. A little sugar may be added if desired or the orange juice may be diluted with several ounces of water.

Stewed apples, peaches, apricots or prune pulp may be given as dessert after one of the morning feedings, or the infant may have a portion of a baked apple or a thoroughly ripe banana.

7. **Desserts.**—Simple custards and puddings are suitable. Custard is prepared by mixing one teaspoonful of sugar and one teaspoonful of flour together with one beaten egg and 6 ounces of milk. This is placed in a cup in a saucepan of water and boiled for fifteen minutes. Cornstarch pudding is prepared by mixing two tablespoonfuls of cornstarch with one tablespoonful of sugar to a pint of milk. This is cooked in a double boiler for one-half hour and cooled in a mold.

The daily schedule during the second year should be about as follows:

6:00 to 6:30 A.M. Milk, warmed, 8 ounces, preferably taken from a glass.
9:00 A.M. Orange juice, 2 to 3 ounces; sugar added if necessary; water up to 6 to 8 oz. if desired.
10:00 A.M. (Just after bath) 2 to 4 tablespoonfuls of cereal with a little milk and butter. One piece of toast or zwiebach and a soda or graham cracker. Eight oz. milk. Stewed fruit.
2:00 to 2:30 P.M. (After nap) 2 to 3 tablespoonfuls of cooked vegetables, egg or scraped meat. Eight oz. milk. Dessert.
6:00 to 6:30 P.M. Eight oz. milk.

CHAPTER XIX

MALNUTRITION, ATHREPSIA, MARASMUS

Malnutrition in infants may be of all grades, irrespective of the cause. The infant may be merely a little below normal weight for the age and show no particular symptoms, or he may have wasted to a skin-covered skeleton, unable to assimilate even proper food and extremely susceptible to infection.

Various names have been used to describe malnutrition in infants such as "atrophy," "hypothrepsia," "athrepsia," "marasmus" or the German term "dekomposition." By some the term "marasmus" or "dekomposition" has been applied as indicating a more or less specific condition. It would appear better to consider the various forms of malnutrition as merely stages of the same condition. For convenience of designation, one may consider an infant as essentially normal who is within ten or fifteen per cent of the average weight for the age, birth weight and length. The term "hypothrepsia" (ὑπό = under, θρέψις = nutrition) may be applied to infants twenty to forty per cent below the average weight, and the term "athrepsia" (a- = negative, θρέψις = nutrition) or "marasmus" to an infant more than forty per cent underweight. Infants in this latter severe stage of malnutrition present a more marked and characteristic symptomatology than those who are only moderately undernourished.

The Causes of Malnutrition

The chief causes leading to malnutrition in infancy are the following:

(1) Underfeeding
(2) Infection
(3) Constitutional and congenital anomalies
(4) Poor hygiene
(5) Overfeeding

Underfeeding

The chief cause of malnutrition is inadequate food intake. The inadequacy may consist in simple underfeeding with a food too

low in caloric value or there may be a specific deficiency of certain elements in the diet, such as protein or the vitamins.

Simple underfeeding may occur even in the case of breast-fed infants if the mother's milk supply is insufficient or if the infant is unable to nurse properly. Underfeeding, however, is more common in the case of the bottle-fed baby.

The most common fault in bottle feeding is the giving of too dilute milk mixtures having low caloric values per ounce. The common but erroneous belief that the nutritional disturbances of infants are due to relative or absolute overfeeding with the different food elements has undoubtedly been responsible for this frequently repeated mistake. Ill advised tampering with the formula by decreasing one or another food element on the assumption that this or that element has been responsible for causing a specific type of indigestion is especially likely to result in underfeeding. When attention is centered upon the digestibility of the food and the character of the stools, the total caloric needs are often overlooked with disastrous results. There are, of course, conditions under which it is necessary to decrease temporarily the total food intake or the amounts of certain elements in the formula, but it should be understood clearly that food which fails to meet the caloric requirements, no matter how digestible it may be, never leads to normal growth and development.

Underfeeding, if persisted in, initiates a vicious circle. The underfed infant becomes less able to assimilate food and may even suffer from starvation diarrhea. This diarrhea may be interpreted as an indication for further reduction of the food until ultimately the infant becomes so undernourished that he becomes susceptible to infections which still further limit his capacity for utilization of food. Finally the infant reaches a state where he is unable to utilize a sufficient amount even of suitable food to meet the minimal requirements. Under such circumstances death is inevitable.

In an analysis of several hundred "difficult feeding cases" coming to the St. Louis Children's Hospital, the primary cause of the difficulty in over 80 per cent was found to be underfeeding. In many of these the picture was complicated by the presence of infections, but the histories indicated that the majority were underfed for long periods previous to the onset of the infections. Next to a deficiency in calories, the most frequent cause of malnutrition

so far as the food is concerned, is protein deficiency. The effects of this deficiency are discussed in Chapter III. Deficiencies of carbohydrate or of the several vitamins may also lead to malnutrition.

Infection

Any infection accompanied by fever is likely to result in a decrease in the appetite and also in the digestive capacity. It has been shown experimentally that in the presence of fever, the secretion of digestive juices is diminished and absorption from the intestinal tract is impaired. Acute infections result usually only in a temporary retardation of weight gain. Chronic infections, on the other hand, may lead to progressive impairment of the nutrition. Chronic infections are more likely to occur in infants who have previously been underfed. In well-nourished infants the infections which occur are likely to be of short duration. The most frequent infections during infancy which interfere with the nutrition are otitis media and pyelitis. Tuberculosis, when present, often, but not always, leads to malnutrition. Fortunately the incidence of tuberculosis among young infants is rapidly decreasing in this country.

Chronic infection may interfere with the nutrition, even though there be no fever present. Syphilitic infection, for example, may lead to an extreme degree of malnutrition. It would appear that the infection so damages the cells of the body as to render them incapable of utilizing food which would ordinarily be considered adequate. An infant with congenital syphilis often begins to gain in weight coincident with the institution of specific antisyphilitic treatment, irrespective of any change in the character of the diet. For a more detailed discussion of the influence of infections on nutrition, see Chapter XXVIII.

Constitutional and Congenital Anomalies

The failure of infants to thrive is often ascribed to "constitutional weakness." In many instances this term is used as a cloak to cover one's ignorance of the true nature of the condition. There are certain instances, however, in which infants fail to do well even when the diet is apparently adequate and no evidences of disease can be discovered. In some of these cases coming to autopsy, congenital anomalies are found, such as a rudimentary

pancreas, a single small kidney, an atrophic gastrointestinal mucosa or anomalies of the vascular system. Certain congenital anomalies are readily recognized during life, such as some of the forms of congenital cardiac disease, atresias of the gastrointestinal tract, endocrinopathies and maldevelopments of the central nervous system. Pulmonary atelectasis is also an occasional cause of malnutrition. Infants with harelip and cleft palate or tongue tie may receive an insufficient amount of food because of inability to suckle and in consequence become undernourished.

In some infants no anatomical cause for the failure to thrive is discoverable either during life or at autopsy. Some of these infants are born prematurely and do not appear to have sufficient "vitality" to allow for independent existence. In such cases the term "constitutional weakness" may properly be applied.

In the condition known as "celiac disease" there appears to be a functional inability to utilize food, especially fat. The exact cause of this is not known, whether it be a constitutional disturbance, a deficiency disease or the result of a chronic infection. (See Chapter XXII.)

Poor Hygiene

It is difficult to differentiate between the effects of poor feeding and poor hygiene, as the two are frequently associated. An infant kept in crowded, poorly ventilated, overheated quarters or one who is overclothed and receives but little fresh air and sunlight is not likely to thrive in the same manner as a baby under good hygienic conditions. The same is true of babies who are always being trundled around, played with, and shown to relatives and who rarely have a chance for quiet undisturbed rest.

Overfeeding

At the time that the idea of food injury was dominant, the nutritional disturbances of infants were very generally attributed to overfeeding with some food element. We now know that some of the nutritional disturbances occurring with unbalanced diets are due more to deficiencies of certain elements rather than to an excess of others. It is, nevertheless, true that overfeeding with certain food elements may bring about digestive disturbances which interfere with adequate utilization not only of the offending article of food, but also of other constituents of the diet. An ex-

cess of fat, especially cow's milk fat, may lead to gastrointestinal indigestion and subsequent poor nutrition. Overfeeding with easily fermentable sugar, especially in the presence of considerable fat and relatively little protein, leads to increased gastrointestinal fermentation, diarrhea and poor absorption of food. Both carbohydrates and fats are much more likely to cause trouble if the milk formulas are insufficiently sterilized. The nutritional failures seen in infants fed on high carbohydrate diets are, however, as frequently due to relative deficiency of milk as to excess of carbohydrate *per se*.

An excess of protein in the diet is rarely a cause of nutritional disturbance provided adequate amounts of carbohydrate and of water are given at the same time. Infants fed on large amounts of sweet cow's milk with little or no added carbohydrate sometimes fail to thrive, and become malnourished. Malnutrition of this type is sometimes referred to as "Milchnährschaden" or milk atrophy. These infants pass large, hard, constipated stools, containing a considerable proportion of lime soaps. The symptoms appear to be due to two factors, the taking of an amount of sweet milk in excess of the digestive capacity and deficiency of carbohydrate. Infants taking the same amounts of milk as acid milk together with adequate carbohydrate do not develop the condition.

It is thus seen that overfeeding leads to malnutrition chiefly when the diet is an unbalanced one. There is but little danger of overfeeding if the diet is well balanced and properly sterilized. Severe nutritional disturbances are not seen from overfeeding with breast milk. Some vomiting may occur after each feeding, which represents merely the spitting up of excess food and there may be at times numerous stools, but the infant thrives. All too often the assumption is made that an infant is suffering from overfeeding when the actual underlying cause is an acute or chronic infection which has been undiscovered.

The Symptomatology and Pathology of Undernutrition

The pathology of undernutrition is essentially that of starvation. When the caloric value of the utilizable food is lower than the basal heat output, or when the amounts of specific elements such as protein and mineral salts in the food are less than the amounts of these elements metabolized in the body and excreted, destruction of body tissue is inevitable. In such instances the

fuel need is partly met by burning of stored fat, carbohydrate and ultimately protein. Tissues which are destroyed in the natural processes of wear and tear are not rebuilt when the food intake is inadequate.

The utilization of a certain amount of fat to supply the caloric needs causes little or no damage to the organism as a whole. Fat stored in the subcutaneous tissues and elsewhere is transported to the liver and there combined with such carbohydrate as may be available and utilized as fuel. It is only when the amount of available carbohydrate is insufficient that this process of fat utilization is accompanied by untoward phenomena. The metabolism of fat with the simultaneous metabolism of inadequate carbohydrate results in the formation of the acetone bodies. This leads to ketonuria and occasionally to definite acidosis.

As the subcutaneous fat is gradually used up, the infant becomes emaciated, the skin becomes shrunken and when a fold of the skin is lifted between the fingers, it is found to be thin and lacking in turgor. Through the atrophied skin may be seen the outlines of the ribs and coils of intestines. The eyes, no longer supported by postorbital fat pads, sink back into their sockets. The infant, however, may not appear sick, although he will, in most instances, be restless and fretful due to hunger.

After the fat depots become exhausted, more vital structures of the body are destroyed to supply energy. The protein of the muscles, blood and various organs is utilized as fuel. This necessarily leads to general atrophy. The destruction of blood protein results in a decrease in blood volume, and this in turn to poor circulation. It has, for example, been shown, in the case of severely athreptic infants, that the volume of the blood passing through a given part of the body in a unit of time may be reduced to as little as one-tenth of the normal amount. Such poor circulation necessarily exerts an effect upon almost every organ in the body. The heart muscle itself suffers functional disturbance and this still further impairs the circulation. The gastrointestinal tract supplied by atrophied and poorly circulating blood becomes functionally inefficient, so that the functions of digestion and absorption are impaired.

The peripheral vessels, especially those of the skin, tend to collapse and to become constricted. The blood corpuscles pass with difficulty through the narrowed peripheral arterioles and are held

back and stagnate on the arterial side. This stagnation of corpuscles with partial reduction of the hemoglobin results in a peculiar grey color of the skin.

Infants who have reached an extreme degree of malnutrition or athrepsia show but little resistance to infections. They are especially likely to develop otitis media and pyelitis, and are prone to infections in the gastrointestinal tract. Organisms which ordinarily are found only in the colon may invade the upper intestine and cause vomiting and diarrhea.

In severe degrees of athrepsia, the red blood cell count is diminished, but in the milder degrees the count may be within or even above normal limits. These high counts are, however, misleading, as blood obtained from the veins may have a much smaller number of red blood corpuscles than that obtained by skin puncture. The discrepancy is explained by the fact that the blood obtained by skin puncture contains corpuscles held back by construction of the arterioles. The leucocyte count is correspondingly high in capillary blood obtained by skin puncture as compared with venous blood. The total white count may be further elevated as the result of intercurrent infections. The usual white blood cell count is in the neighborhood of 14,000 to 16,000. The protein content of the blood serum is low, often being no more than 3 or 4 per cent as compared with the normal of 6 or 7 per cent.

The volume of the blood as determined by the dye method may be as low as 50 per cent of that of normal infants of the same age or body length. The blood volume in proportion to the actual weight, however, may be relatively high. This is explained by the fact that the loss of weight in these infants is chiefly of subcutaneous fat, which is a relatively nonvascular tissue.

The urine shows little if any change on ordinary examination. The total nitrogen and mineral salt outputs, however, in cases of severe athrepsia exceed the intake of these elements, this being due to the elimination of products resulting from the destruction of body tissues.

The pulse is relatively slow and readily compressible; the blood pressure normal or low. The heart sounds are weak.

The temperature, except in the presence of infections, tends to be subnormal and may fall as low as 94° or 95° F. Some infants with severe atrophy show a remarkable tendency to hydrolability, i.e., the water content of the body is subject to wide and rapid

fluctuation. An athreptic infant may for a short period gain in weight at an abnormal rate and become pasty in appearance and edematous, due to water retention. These gains in weight may be mistaken for legitimate gains until the appearance of a definite pitting edema discloses the true nature of the condition. Equally sudden losses in weight occur, due to elimination of stored water.

Nutritional edema is likely to occur in the case of infants who have received relatively little protein in the diet, especially those who are fed on foods high in carbohydrate. Accompanying the edema there may at times be observed petechial or purpuric eruptions—the so-called cachectic purpura. In some instances this appears to be the result of vitamin deficiency.

If the underlying cause of the malnutrition in an individual case can be determined and eliminated, gradual recovery is to be expected in the majority of cases. If, however, the malnutrition has gone on to an extreme degree, so much damage may have occurred to the body that recovery is impossible, even though suitable food be given and means taken to eradicate infections.

The Treatment of Malnutrition

Malnutrition is, in the overwhelming majority of cases, a preventable condition. Prophylaxis is easier than cure.

Whenever an infant fails persistently to gain in weight at a normal rate, the first step should be to ascertain whether or not the food intake is adequate in every particular. The caloric value should be checked and a calculation made to determine whether the infant is receiving sufficient protein, carbohydrate, mineral salts, and vitamins. If the feeding is a well-balanced one and if the infant is taking all of the formula offered but still shows signs of hunger, the volume of the feeding should be increased or else the feeding made more concentrated by omitting some of the water. Above all, constant changes in the formula are to be avoided. If the feeding is obviously unsuitable in some respect, the difficulty should be remedied but there should be a definite reason for the change in every case. There is no surer way of bringing about a condition of malnutrition in an infant than the injudicious switching from one type of feeding to another in the hope of finding something that will exactly agree. Any success which attends such efforts is usually to be attributed to the accidental giving of a

sufficient amount of food. When a formula has been selected which meets all of the requirements, further changes should not be made.

In the case of breast-fed infants, the total intake of milk should be determined by weighing before and after nursings for a number of days and if the total amount of milk received is less than that which an infant of the age and size should receive, steps should be taken to alter the nursing régime so as to insure an increased intake or else complementary or supplementary feedings should be given. (See Chapter XI.)

If no fault can be found in the diet, and if there are no digestive disturbances, a search should be made for possible infections. Any temperature elevation or increase in the leucocyte count should be taken as presumptive evidence of infection. It should, however, be noted that some athreptic babies with chronic purulent infections fail to show any significant temperature elevation. The leucocyte count in such cases, however, is usually increased.

The examination should certainly include observation of the eardrums with an electric otoscope and also examination of the urine for pus. An intradermal tuberculin test and a Wassermann reaction on the blood give valuable information as to the presence of tuberculosis or syphilis.

Once the condition of malnutrition has developed, the indication is to give a sufficient amount of food to cover the nutritional requirements and yet not more than can be taken care of by the functionally impaired gastrointestinal tract.

The food requirements of undernourished infants are high in proportion to the body weight and approximate those of normal infants of the same age but weighing much more. Thus, an undernourished infant who has weighed seven pounds at birth, but at the age of four months weighs only eight pounds, requires in the neighborhood of 55 calories per pound of his *expected* weight, which should be 12½ or 13 pounds. His requirement would therefore be in the neighborhood of 700 calories or almost 100 calories per pound of actual weight. In some infants the caloric requirement may even exceed this amount in relation to body weight.

This large food intake is required to meet the high energy demands and also because of the fact that only a portion of the food given is absorbed from the functionally impaired gastrointestinal tract.

Aside from the high caloric requirements, there are special needs for certain specific elements of the diet in the case of athreptic infants. Relatively more protein is required than in the case of normal infants because of the need for reconstructing destroyed body tissues. It is for this reason that it is often advantageous to add additional protein to the diet of athreptic infants who are being fed on breast milk. There is sufficient protein in human milk to meet the needs of the normal infant, but not necessarily enough for restoring the athreptic infant.

Athreptic infants require a larger amount of mineral salts than normal infants to provide for the reconstruction of damaged tissues. In moderate degrees of malnutrition, sufficient mineral matter will be obtained in the milk given, but in extreme athrepsia recovery is hastened by the addition of mineral salts to the diet, especially the salts of sodium, potassium and calcium. This additional mineral requirement may be met by the injection or feeding by mouth of Ringer's solution or one of the special salt solutions.

Inasmuch as the capacity of the athreptic infant to digest and absorb food is low, the food necessarily must be one which is readily digested and absorbed. Malnourished infants are often unable to take as large volumes of food as normal infants of the same age because gastric motility is impaired so that the stomach empties slowly, furthermore the stomach may be actually smaller than normal. For these reasons the food must provide the necessary elements in small volumes, or, in other words, it must be concentrated.

Athreptic infants usually secrete but small amounts of weakly acid gastric juice and in consequence are unable to digest very much sweet cow's milk, the buffer substances of which readily neutralize the small amounts of acid present. It is not usually possible to feed much undernourished infants satisfactorily on sweet cow's milk, for it cannot be digested in concentrated form, and a sufficient amount of food cannot be supplied when the milk is diluted. Human milk is readily digestible, but it is not always possible to give a sufficient amount to meet the caloric requirements and furthermore the protein of human milk is barely sufficient for the needs of the athreptic infant. The caloric value and protein content of human milk may be increased by the addition of dried cow's milk or casein.

Cow's milk formulas prepared from whole lactic acid milk or acidified evaporated milk with added carbohydrate are well digested, have a high caloric value, and supply sufficient protein and usually sufficient mineral salts for the needs of malnourished infants. A formula composed of whole lactic acid milk, 11 parts, Karo Syrup, 1 part, or a similar formula from evaporated milk, has a caloric value of 30 calories to the ounce. Such a feeding is readily digested and absorbed and has proved a very satisfactory one for the feeding of undernourished infants. (For preparation of formulas, see Chapters XV and XVI.)

Certain symptoms call for changes in the formula. If vomiting occurs, it may be necessary to thicken the formula by boiling with cereals. For further details, see Chapter XXIII.

In the presence of marked diarrhea, it may be necessary to remove temporarily a portion of the fat from the milk and also to reduce somewhat the proportion of added carbohydrate. One should, however, not make the mistake of reducing the food intake too greatly because of the passage of fairly numerous or moderately loose stools. A starvation diarrhea should not be confused with the diarrhea due to overfeeding. Many undernourished infants gain in weight even though they may not have a strictly normal stool for weeks. A sudden increase in the number of stools or excessive vomiting is often due to the onset of an acute infection and the indication in such cases is to look for the infection rather than to change the formula. Starvation is especially to be avoided, and the feeding should always be as concentrated and given in as large amounts as can be taken without leading to an increase in the gastrointestinal symptoms.

Feeding intervals should not be more frequent than every four hours except in the case of certain premature or very young infants who will take only very small volumes at a feeding. There should be at least six feedings in the day in order to insure an adequate food intake. In the case of infants who have been grossly underfed for a long period, it is sometimes well to begin with small feedings of somewhat diluted food, but these should be increased rapidly in concentration and amount.

Most infants who have become undernourished as the result of an insufficient food intake are ravenously hungry, but a few have become so weak that the appetite fails. The presence of infection also tends to cause a decrease in the appetite. In

such instances it may be necessary to resort to gavage or to the administration of food parenterally in the form of dextrose (glucose). In some underfed infants the appetite appears to be impaired because of a deficiency of vitamin B in the previous diet. In these cases extra amounts of vitamin B should be given in the form of yeast or wheat germ preparations. Certain amino acids, notably *beta-alanin,* when fed lead to hunger contractions of the stomach and thus increase appetite. Commercial beef extract contains a good deal of *beta-alanin* and may be given in amounts of one-half teaspoonful in a little water twenty minutes before a feeding. The injection of insulin may also increase appetite. The use of this is discussed below.

Some athreptic infants take fairly large volumes of concentrated feedings having high caloric values and containing what would appear to be sufficient amounts of all the essential elements and yet fail to gain. There may be no evidences of infection. In such cases the failure to gain is evidently due to incomplete utilization of food. Thus, we have observed, in the course of a complete metabolic experiment on an athreptic infant, a loss of over 40 per cent of the total caloric value of the food by way of the bowel. This failure of digestion and absorption is in part to be accounted for by the poor circulation dependent upon decreased blood volume. Transfusion in such cases is likely to be followed by improved digestion and absorption of food, so that weight gains may occur without further changes in the diet. Transfusion also seems to increase the resistance to infection and often is followed by improvement in appetite.

Indirect transfusions with citrated whole blood are as effective as direct transfusions and a further advantage in the use of citrated blood is that sufficient blood may be drawn from the donor to provide for two or three transfusions at daily or two-day intervals. The amount of blood given at a single transfusion should be from 20 to 30 c.c. per kilogram of body weight (⅓ to ½ ounce per pound). Larger amounts than this may be injected intraperitoneally, but the intravenous route is usually preferable. For further details as to the technic of transfusion see Chapter XXXIV.

Less effective than transfusion in increasing the blood volume and improving the circulation is the intravenous injection of dextrose solution. This results in a temporary increase in blood vol-

ume. Hypertonic solutions of dextrose, when injected intravenously tend to promote absorption from the gastrointestinal tract. An additional advantage of dextrose is that it provides extra food in readily available form. The strength of solution used may vary from 10 to 20 per cent, the total amounts given being about the same as in the case of blood transfusion. The dextrose given in this way is not completely utilized, as a variable amount is excreted by way of the urine depending upon the strength of the solution and the rapidity with which it is injected. The dextrose tolerance of undernourished infants is much higher than in the case of normal infants or of adults: that is, they can utilize more dextrose per pound of body weight per hour.

Dextrose may also be administered subcutaneously, intramuscularly or intraperitoneally. For intramuscular or subcutaneous injection, a 10 per cent solution is used; and for intraperitoneal injection a 6 per cent solution. For full discussion of the technic of the preparation and administration of dextrose solutions, see Chapter XXXIV.

The simultaneous administration of insulin appears to render dextrose injections more effective. Insulin promotes the utilization of dextrose and also its conversion into glycogen and probably its storage as fat. It has, for example, frequently been noted that diabetic children receiving insulin often become extremely fat, even though they are not given excessive amounts of food. Insulin appears to have a similar fattening effect on undernourished infants. Insulin is to be used with caution and only when an adequate amount of carbohydrate is given to protect against insulin reactions. It should not be given to undernourished infants with gastrointestinal symptoms who may be absorbing relatively little carbohydrate, unless dextrose is injected at the same time in the proportion of one unit of insulin to 1.5 or 2 grams of dextrose. The insulin may be added to the dextrose solution just previous to injection. The amounts of insulin used are usually from 5 to 10 units at a dose.

The athreptic infant, as already mentioned, shows a tendency to hydrolability and may, at any time, become desiccated as the result of vomiting and diarrhea and failure of the body tissues to retain water. Unless sufficient fluid is supplied, the symptoms of anhydremia are likely to be superimposed upon those of athrepsia. In order to prevent anhydremia, water should be adminis-

tered to athreptic infants in sufficient amounts to prevent any desiccation. It is not always possible to give sufficient water by mouth, so that resort must be had to subcutaneous or intraperitoneal injection. Furthermore, water may not be retained unless additional mineral salts are given at the same time. For further discussion, see Chapter XXXII.

In most cases of athrepsia, infections occur. In the case of an undernourished infant suffering from a chronic infection, it is often impossible to determine whether the nutritional state is the result of infection, or vice versa. The two go hand in hand, the one predisposing to the other. So long as infection exists, it is difficult to restore the infant to a state of satisfactory nutrition even with feedings which otherwise would be adequate. Furthermore, until the nutrition is improved, infections are not likely to clear up. This explains, in part, the high mortality among infants suffering from extreme undernutrition.

The athreptic infant should be examined regularly for evidences of infection. Some athreptic infants develop a low grade otitis media which is difficult to detect on otologic examination. In others, middle ear infections spread to the mastoid antrum and this results in an exacerbation of constitutional symptoms, especially vomiting and diarrhea, although local manifestations in the mastoid may be but slight. The additional infection may throw upon the already weakened infant a strain beyond his powers to resist. For a fuller discussion of the relationship of infections to nutrition and the treatment of infections, see Chapter XXVIII.

To illustrate the principles outlined above in the treatment of athrepsia, protocols of typical cases are given. It is not to be assumed that all cases respond in the same manner as those described. These are favorable cases in which good results were obtained. Equally good results and equally good "before and after" pictures could be obtained in the case of infants treated in entirely different ways. The protocols merely indicate the various types of treatment which may be applied. It is often difficult to appraise the value of any form of treatment, for there may be various factors involved that influence the nutrition. Some athreptic infants begin to gain from no assignable cause, others because of a cessation of diarrhea or the clearing up of an infection, and others because of an increase in food intake. These are only some of the factors.

Protocols

Athrepsia Due to Underfeeding

Effect of Giving Sufficient Food

J. W. Girl. Brought to the hospital at age of four months, with history of failure to gain. Infant normal at birth, weight 9 pounds, breast-fed for two weeks. Weaned because of "colic." Given formula consisting of

Whole milk	10 oz.	200 calories
Dextrimaltose	1 oz.	120 calories
Water	14 oz.	
		320 calories

Given three ounces every two to three hours. Took feedings well and had no diarrhea or vomiting, but failed to gain. Colic continued. Changed to sweetened condensed milk, diluted 1:8. Fed every two hours. Total caloric intake about 250 calories. Amount of milk in mixture about 9 ounces. Slight gain in weight, but stools were loose. Taken off milk and fed nothing but barley gruel for several days and then given mixture of equal parts barley gruel and skimmed milk. Loose stools continued. No fever. Given protein milk for a week, during which time diarrhea was less, but loss in weight continued. Finally changed to formula consisting of

Whole milk	14 oz.	280 calories
Mellin's Food	1½ oz.	180 calories
Water	18 oz.	
		460 calories

Given eight feedings of 4 ounces each. Failed to gain and stools became loose.

Child brought to hospital at age of four months, weight 8½ pounds (3900 grams). Infant undernourished but no signs of disease found. Diagnosis: simple underfeeding, starvation diarrhea.

The clinical chart of this patient is shown in Fig. 10. For explanation of signs used on the chart, see Appendix.

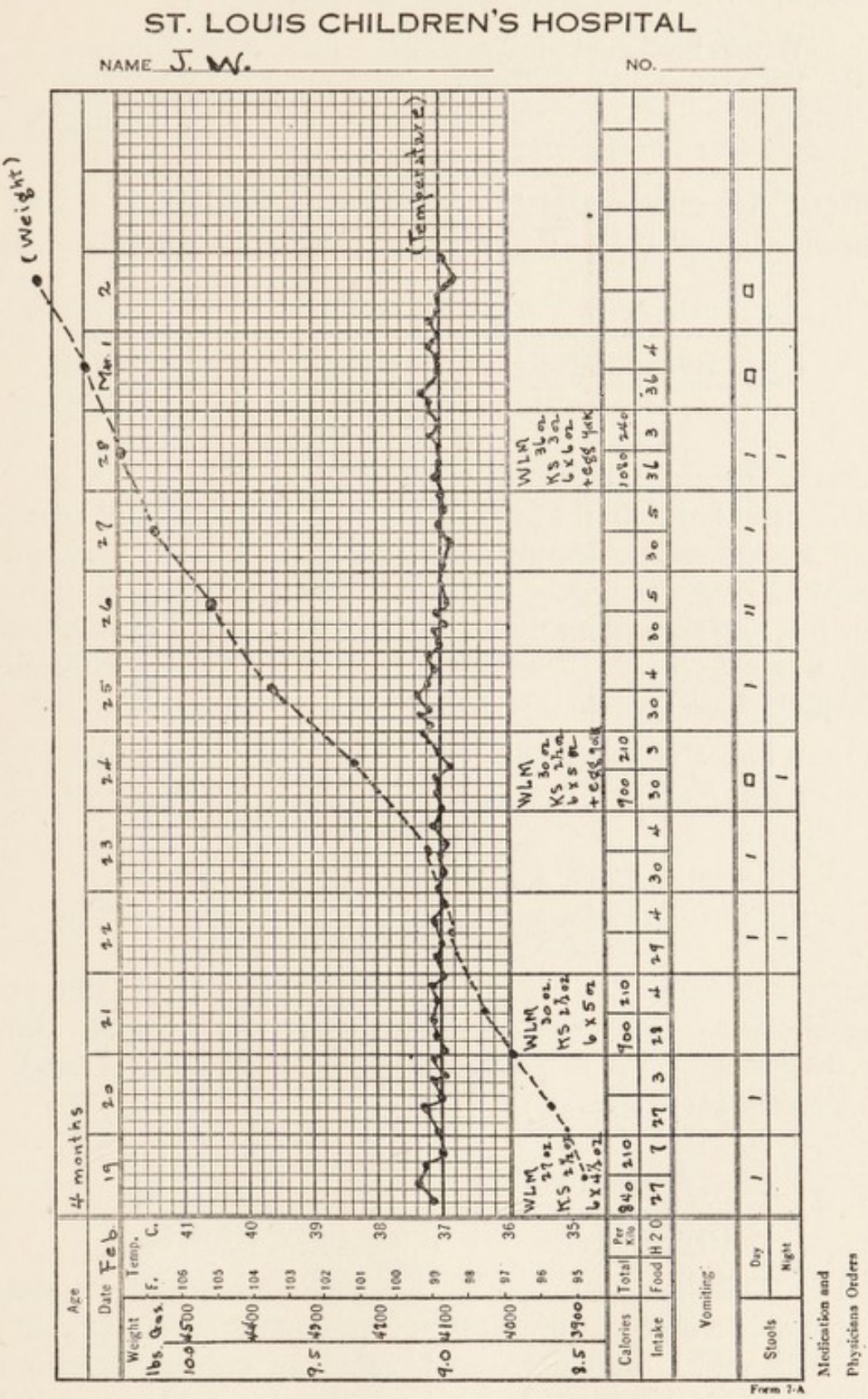


Fig. 10.—Chart of infant previously underfed, showing effects of adequate caloric intake.

The baby was given a formula consisting of

Whole lactic acid milk	27 oz.	540 calories
Karo Syrup	2½ oz.	300 calories
		840 calories

Offered 4½ ounces every four hours, six feedings.

Infant took all food offered. After two days formula changed to

Whole lactic acid milk	30 oz.	600 calories
Karo Syrup	2½ oz.	300 calories
		900 calories

Offered 5 ounces, every four hours.

Took most but not all of the formula. No diarrhea or vomiting. Rapid weight gain. Infant emptied bottle at most feedings, so amount offered was finally increased to 6 ounces, the proportions in the formula remaining the same. The yolk of one egg was added to the day's feeding. Steady gain in weight. Infant still hungry so formula increased to

Whole lactic acid milk	36 oz.	720 calories
Karo Syrup	3 oz.	360 calories
		1080 calories

Plus yolk of one egg.

Six feedings of 6 ounces each.

Infant continued to do well, although stools were at times constipated. Discharged from hospital at end of 11 days. During this time weight increased from 8½ pounds (3900 grams) to 10½ pounds (4720 grams).

Athrepsia in a Premature Infant

Use of Breast Milk With Added Protein

R. H. White male. Infant born one month before term, weight 4½ pounds. Nursed at breast every two hours but seemed to get very little milk. Failed to gain. Fed on several proprietary foods, much diluted. Lost weight steadily. Brought to hospital at age of three months. On admission the weight was 3½ pounds (1600 grams). Very athreptic. Somewhat dried out. Skin grey;

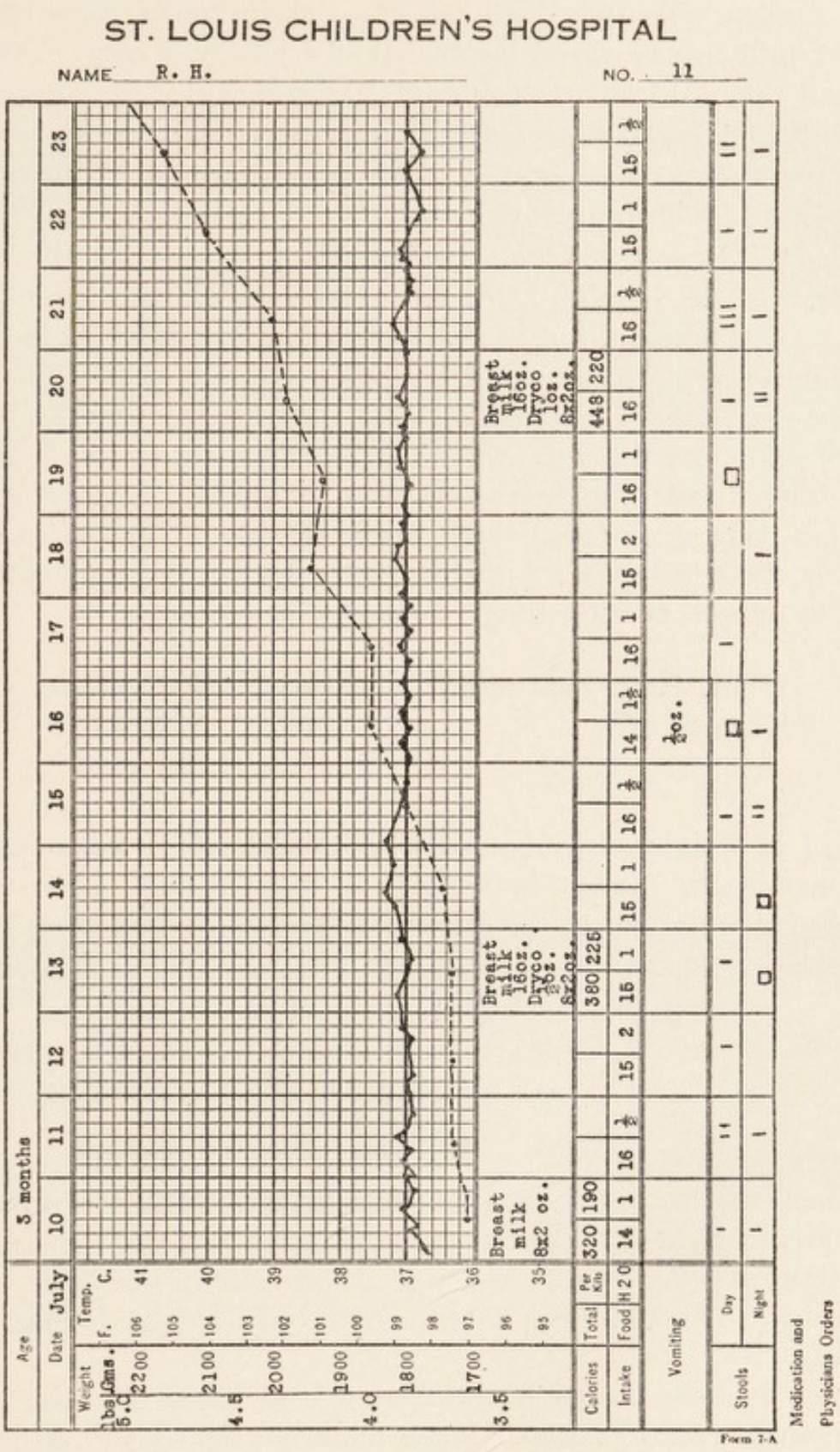


Fig. 11.—Chart of athreptic premature infant. Feeding of enriched breast milk.

eyes sunken. Temperature 96° F. Put in warm room and fed breast milk every three hours, eight feedings a day. Average amount taken, 15 ounces per day (300 calories). No vomiting or diarrhea, but very slow weight gain. At end of three weeks infant's weight had increased to only 3¾ pounds (1700 grams). (See Fig. 11.) Infant would not take any more food, hence it was decided to increase the caloric value and protein of the milk by the addition of dried milk. One-half ounce of "Dryco" dried milk added to total amount of breast milk given infant. This increased the caloric value by about 60 calories and the protein by about 50 per cent without increasing the volumes of the feedings. Baby then began to gain weight. No digestive disturbance. Amount of added dried milk increased to one ounce. Steady gain in weight. During the next three weeks the weight increased to 5¾ pounds (2600 grams). Appetite increased so that baby was taking 2½ ounces at a feeding. Formula then changed to

Whole lactic acid milk*	20 oz.	400 calories
Karo Syrup	2 oz.	240 calories
		640 calories

Gain in weight continued at approximately the same rate for the next week. Infant sent home on this formula. The clinical chart of this patient at the time that the caloric value and protein contents of the food were increased is shown in Fig. 11, page 183.

Malnutrition Complicated by Infection and Diarrhea

Use of Transfusions

F. O. Full-term infant. Weight at birth 7½ pounds.

Breast-fed for two weeks and then given a variety of formulas, no one of which supplied sufficient calories, protein, mineral salts or vitamins. At age of six weeks had cold in the head, fever, diarrhea. The diarrhea continued despite frequent changes in feeding.

Child brought to the hospital at age of three months; weight 6¼ pounds (2800 grams). Much undernourished and desiccated. The appearance of this child on admission is shown in Fig. 12.

*The whole lactic acid milk was prepared from evaporated milk, as described on page 152.

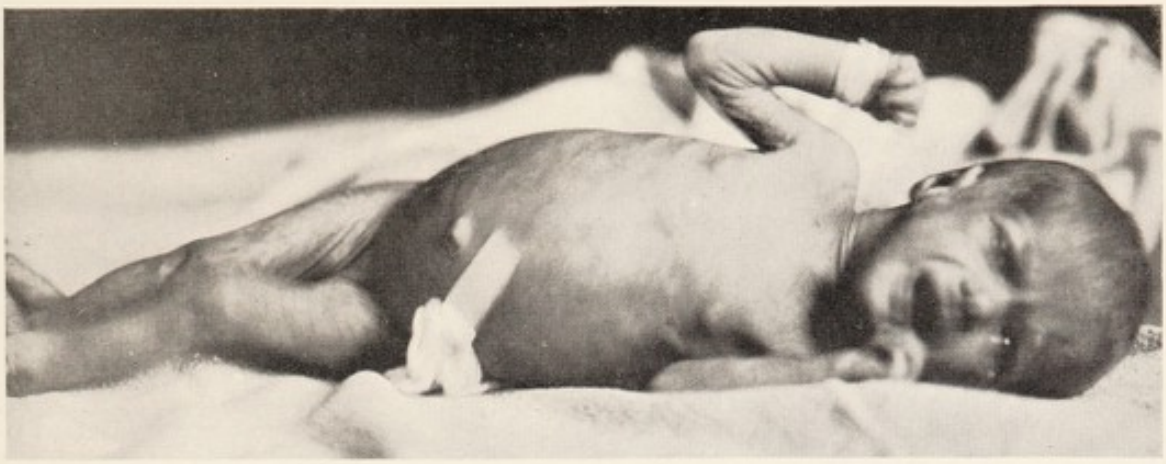


Fig. 12.—Patient F. O. on admission. Age three months, weight 6¼ pounds. Athrepsia, diarrhea, otitis media. Portions of the clinical chart are shown in Figs. 14 and 15.

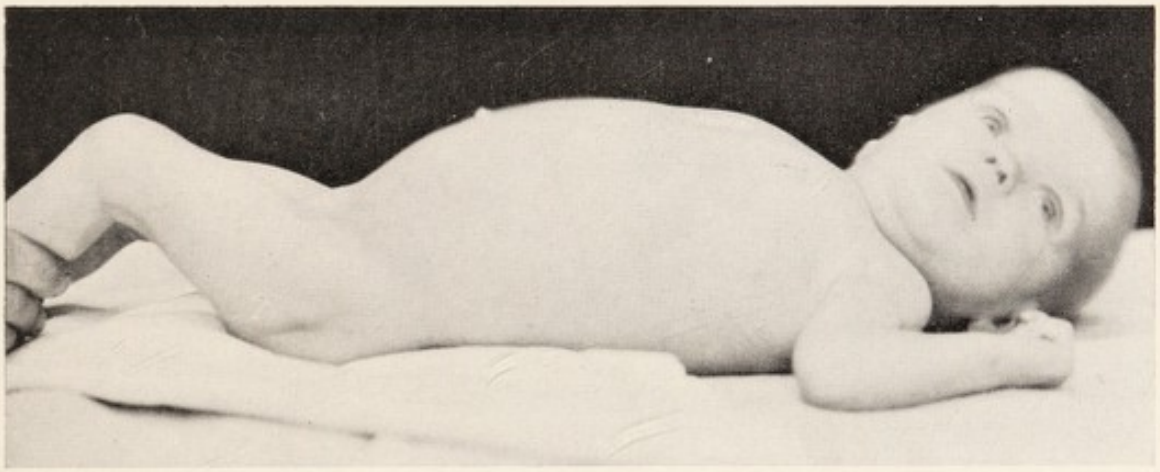


Fig. 13.—Patient F. O. at time of discharge from hospital. Age five months, weight 9 pounds. Compare Fig. 12.

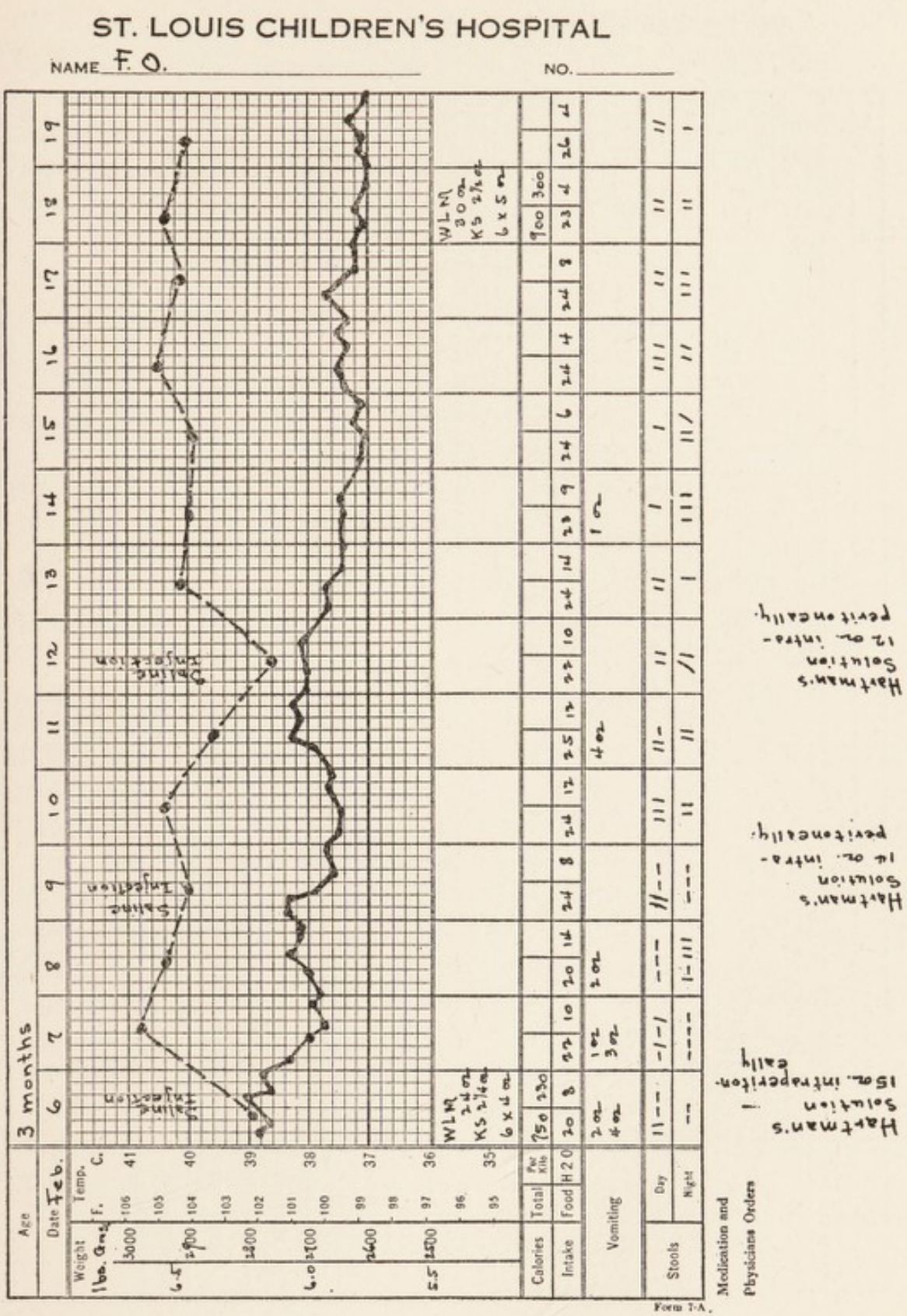


Fig. 14.—Chart of patient F. O. Athrepsia, diarrhea, otitis media. See also Fig. 15.

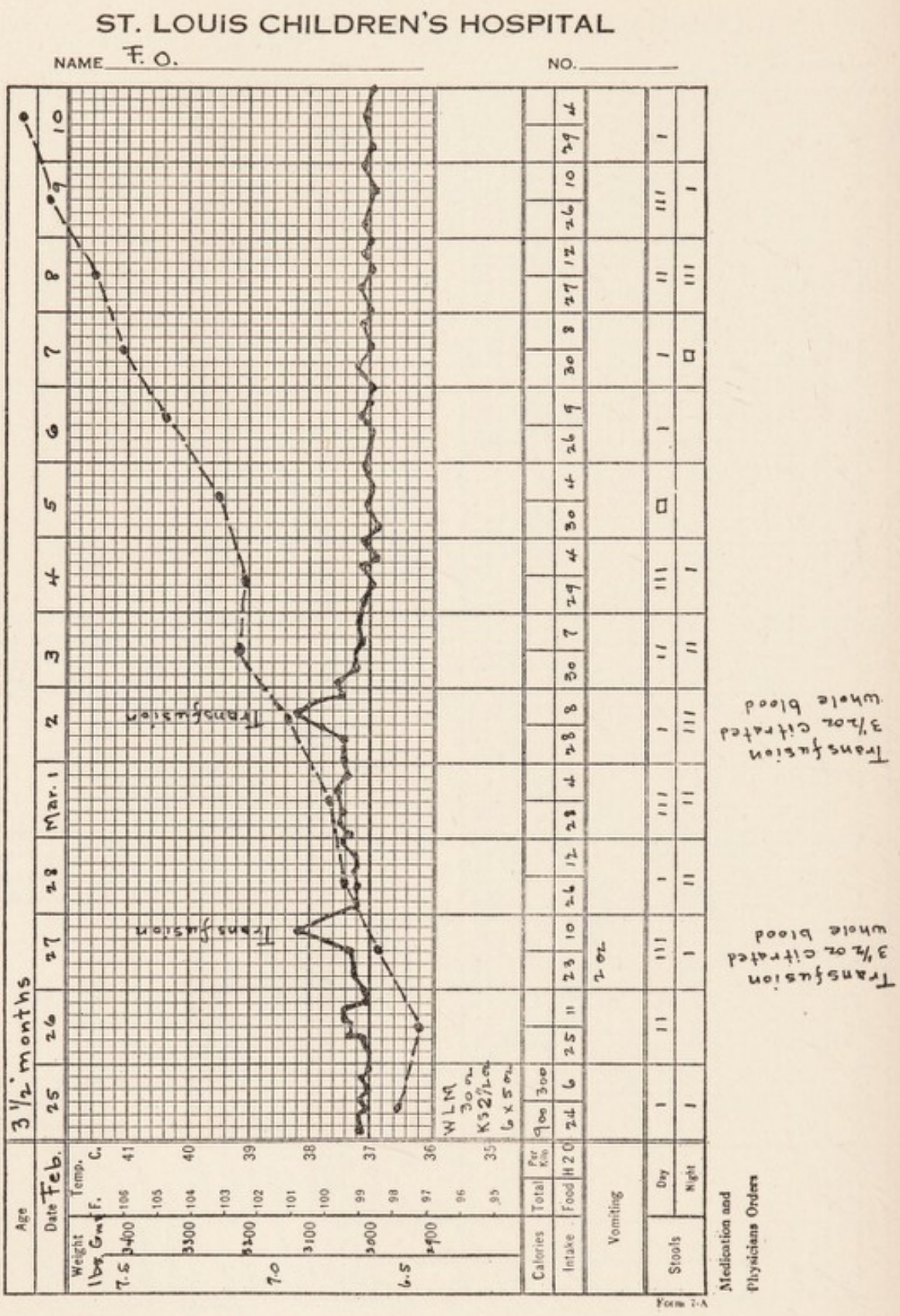


Fig. 15.—Chart of patient F. O. Athrepsia, diarrhea, otitis media. Continuation of chart in Fig. 14.

Temperature 101.8° F. Both eardrums thickened, red and slightly bulging. Diagnosis: underfeeding, athrepsia, otitis media.

Both ears opened with discharge of pus. Temperature gradually fell to normal. Because of dehydration, infant given several injections of saline solution intraperitoneally. (Hartmann's solution, see page 323.) The feeding was as follows:

Whole lactic acid milk	24	oz.	480 calories
Karo Syrup	2¼	oz.	270 calories
			750 calories

Offered six feedings of 4 ounces each.

The diarrhea continued for several days and then ceased as the temperature dropped. No gain in weight occurred however. (See clinical chart, Fig. 14.) Feeding increased to

Whole lactic acid milk	30	oz.	600 calories
Karo Syrup	2½	oz.	300 calories
			900 calories

Six feedings of 5 ounces each.

Not all of this feeding was taken, but there was no diarrhea and no vomiting. It appeared that this child was unable to utilize well the food taken. In order to improve circulation and food utilization, a transfusion of 3½ ounces of citrated blood was given. This was followed two days later by another transfusion of the same amount. The feeding was not changed. (For clinical chart, see Fig. 15.) Following the transfusions, gain in weight began. During this time the ears had continued to discharge, but the discharge gradually ceased as the infant's nutrition improved. At the end of two months the weight had increased to 9 pounds (4100 grams) and the child was discharged from the hospital. No change in the feeding formula was made. The appearance of this patient on discharge is shown in Fig. 13.

Athrepsia, Complicated by Infections

Use of Dextrose Injections and Insulin

B. S. Born at full term. Weight at birth 10 pounds.

Never nursed at the breast. Baby boarded out and very little known of details of feedings, but received mostly sweetened con-

densed milk. Had diarrhea from time to time. Brought to the hospital at age of four months. Weight 6¼ pounds (2800 grams), very much undernourished (Fig. 16). Almost constant feeble crying. Both ears discharging pus. Temperature irregular, varying from 100° to 102° F. Urine contained pus. Given formula as follows:

Whole lactic acid milk	24 oz.	480 calories
Karo Syrup	2¼ oz.	270 calories
		750 calories

Six feedings of 4 ounces each.

Given Ringer's solution intraperitoneally and two transfusions. Stools never normal, always numerous, soft and sometimes watery, but contained no blood or pus. Feeding not changed. At the end of two weeks the stools were less numerous and the temperature had fallen to normal. During the next ten days no gain in weight occurred. Infant given 3 ounces of 20 per cent dextrose solution intravenously daily. Still no increase in weight. Two units of insulin were added to dextrose solution. No weight gain. This child was given all the food she could take. She had been given transfusions and extra fluid. Some remaining infection was present, but the temperature had fallen. Child then given daily 3 ounces of 20 per cent dextrose solution to which 15 units of insulin were added. (See chart, Fig. 18.) These injections were continued for 12 days. During this time there was a rapid gain in weight and progressive increase in appetite. The dextrose and insulin injections were then stopped. At this time the infant was taking

Whole lactic acid milk	30 oz.	600 calories
Karo Syrup	3 oz.	360 calories
		960 calories

Six feedings of 5 ounces each.

The weight continued to increase after insulin administration was stopped. At the time of discharge seven weeks after the glucose-insulin injections had been started the weight was 10¼ pounds (4700 grams) (Fig. 17).

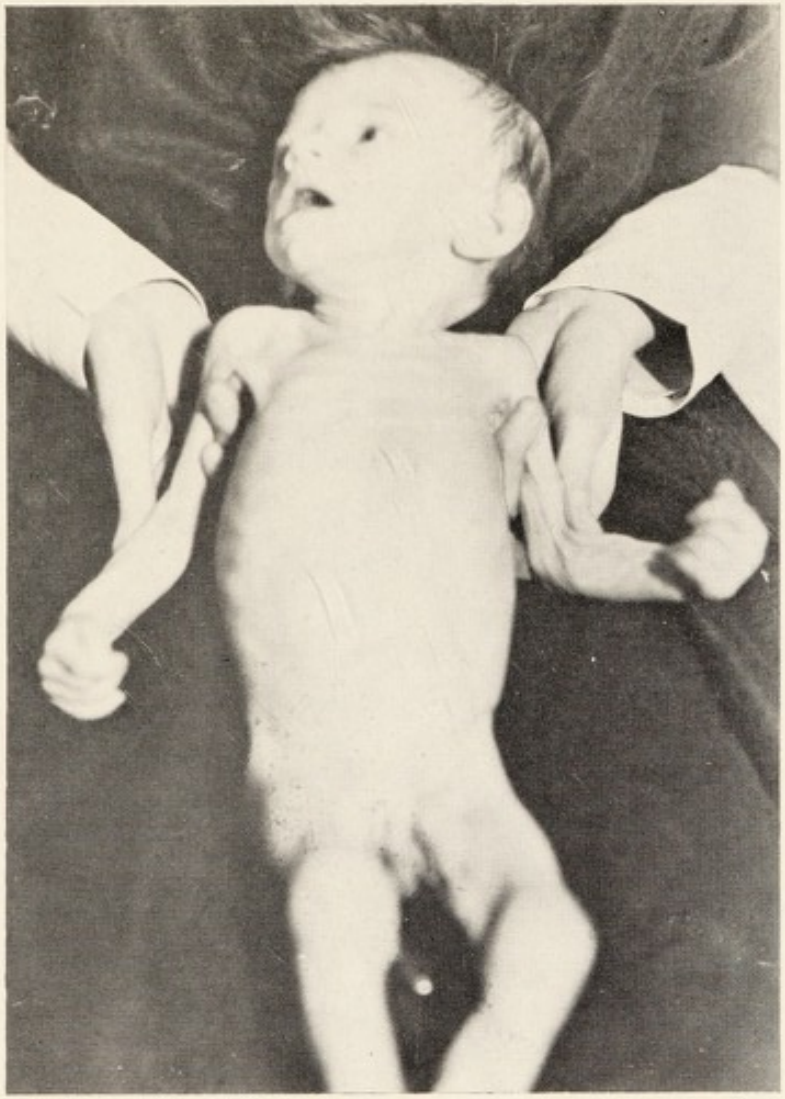


Fig. 16.—Patient B. S. on admission. Age four months, weight 6¼ pounds. Athrepsia. See clinical chart in Fig. 18.

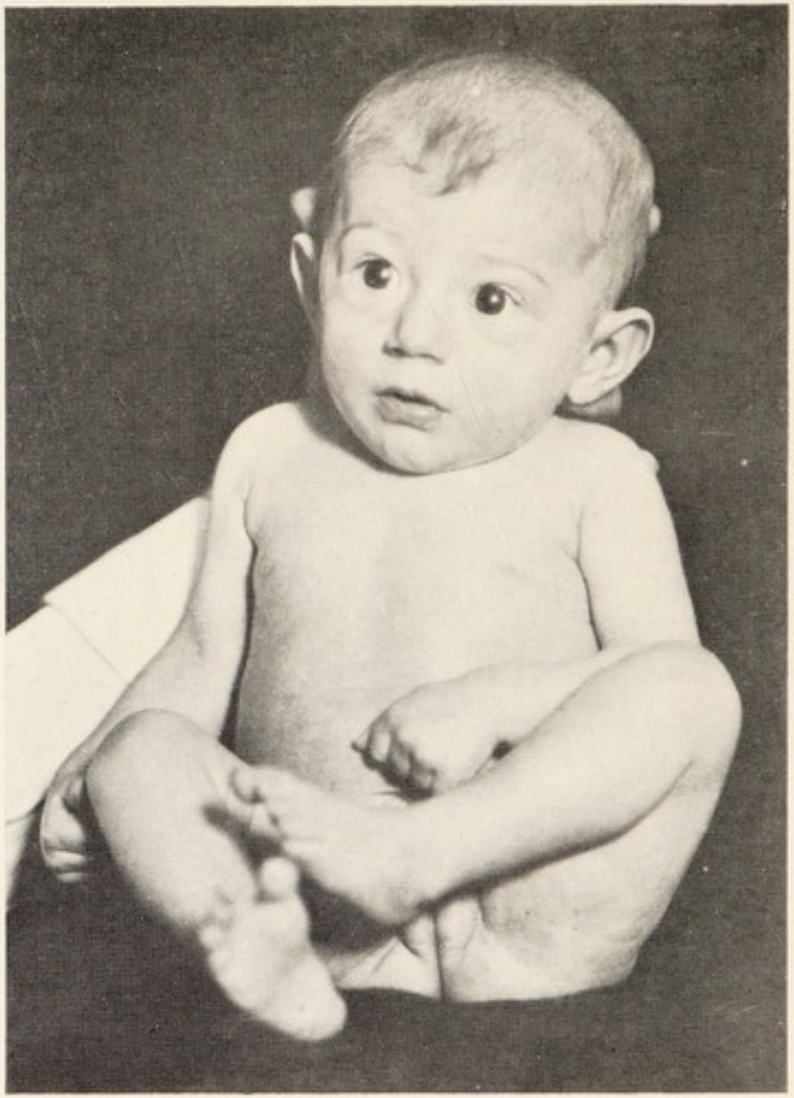


Fig. 17.—Patient B. S. on discharge. Age six and one-half months. Weight 10¼ pounds. Compare Fig. 16.

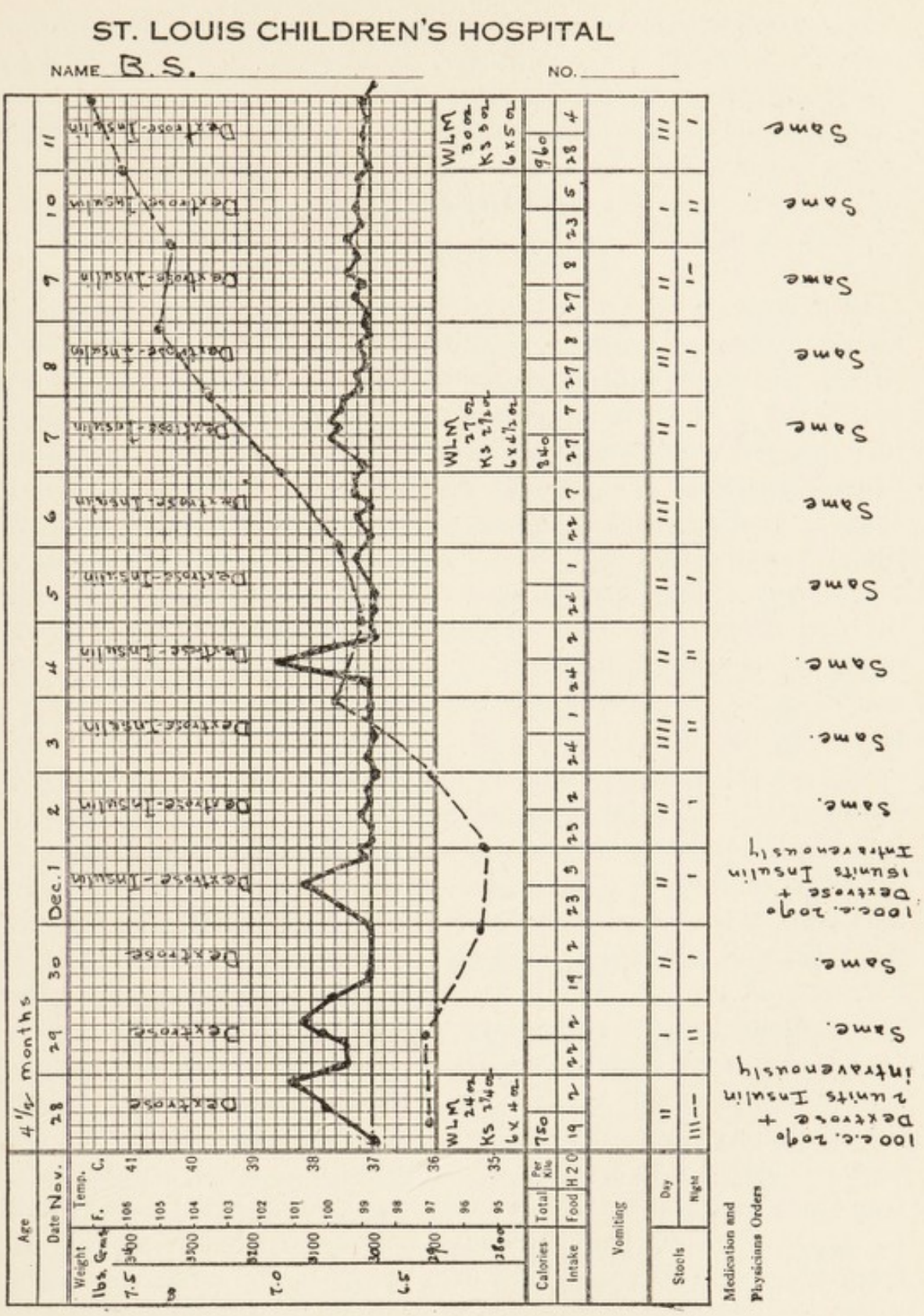


Fig. 18.—Chart of patient B. S. Case of athrepsia treated by injection of dextrose and insulin.

CHAPTER XX

DIARRHEA

General Considerations

In past years, the diarrheal diseases have accounted for almost as many deaths among infants as all other causes combined. There has been a progressive decrease in mortality from this cause, but even at the present time, diarrhea stands in first place in published mortality figures for infants who have survived the neonatal period. The actual number of deaths attributed to diarrhea is probably not so great as vital statistics would indicate because of the fact that infants seriously ill from any cause are likely to develop diarrhea as a terminal symptom and when the original disease has been undiagnosed, diarrhea is given as the cause of death. Even with due allowance for this fact, the mortality rate from infantile diarrhea is high, and unnecessarily so, as the diarrheal diseases are, for the most part, preventable.

Infants are much more likely to develop diarrhea than are older individuals. This is due to a number of factors. The gastrointestinal secretions of the infant differ in composition from those of older individuals. The gastric juice contains less pepsin-rennin and very much less hydrochloric acid. The gastric juice of the infant is well adapted to initiate the processes of digestion when human milk is fed. Small curds are formed in the stomach and the contents become sufficiently acid to inhibit bacterial growth. On passage of the stomach contents into the duodenum, an acid condition is normally maintained throughout the upper portion of the intestinal tract. Probably as the result of this acidity, the stomach and upper portion of the intestinal tract are normally free from bacteria capable of causing gastrointestinal disturbance. In consequence the baby exclusively breast-fed but rarely suffers from severe diarrhea. There are, however, circumstances under which the gastric and intestinal secretions may be diminished.

Any infection accompanied by fever is likely to result in a decreased secretion of gastric acid and of the pancreatic and gastric enzymes. A similar result is brought about by exposure to high

temperatures. Some types of parenteral infection are more likely to lead to diarrhea than others, even though the elevation of temperature may be the same. For example, infants suffering from rhinopharyngeal infections and otitis media are in general more prone to develop diarrhea than those suffering from pyelitis. The nature of the infecting organism also appears to be a factor. Infection of the middle ear with certain strains of toxin producing streptococci are usually accompanied by more severe diarrhea than when the infecting organism is a staphylococcus. Some types of infection appear to lead to a disturbance of the autonomic nervous system which results in increased peristalsis, irrespective of the character of the intestinal contents.

Infections outside the gastrointestinal tract are the most frequent underlying causes of diarrhea occurring in infants who are breast-fed or who are receiving well constructed cow's milk formulas, and this is especially the case during the winter months. (For further discussion of the effects of infection on nutrition, see Chapter XXVIII.)

An infant who from any cause has become badly undernourished, is likely to have lessened gastric secretion. In such cases, the contents of the stomach and of the upper intestinal tract may not be sufficiently acid even when breast milk is fed to bring about bacterial inhibition and as a result organisms such as those of the colon group, which are not ordinarily inhabitants of the duodenum and stomach may be present. When the breast-fed baby receives, in addition, articles of food which are bacterially contaminated, some potentially harmful microorganisms may survive in the intestinal tract.

The infant is incapable of digesting many foods which may be completely digested by the adult with stronger gastrointestinal secretions, and in consequence the diet of the artificially fed infant must be limited largely to milk with added sugar or starch, or possibly egg yolk, during at least the early months of life. Other foods, being less readily digestible, may mechanically irritate the intestinal tract or serve as the means of introduction of bacteria which may cause harm. Both milk and sugar solutions are excellent bacterial culture media, and milk, unless obtained under clean conditions and sterilized before use, contains numerous bacteria, some of which may be of a harmful type. Futhermore, cow's milk, having a higher buffer capacity than human milk,

neutralizes to a greater extent the small amount of gastric acid present. The large tough curds formed from cow's milk tend to act as mechanical irritants and enmesh and protect any bacteria present from the bactericidal action of the gastric juice. These facts serve to explain the greater susceptibility of artificially fed infants to diarrhea. A still further factor in explaining the increased susceptibility to diarrhea of artificially fed infants is that many of these are undernourished and consequently have lessened gastrointestinal secretions. Furthermore, undernourished infants are more susceptible to parenteral infections which when present still further decrease digestive capacity.

Available evidence would appear to support the idea that most of the diarrheas of infancy are due to abnormal bacterial growth, especially in the upper portions of the gastrointestinal tract. The chief exponents of this viewpoint have been Moro, Plantenga, and Arnold. Strong confirmatory evidence has been obtained by a large number of other investigators. According to this viewpoint, we may explain the effects of overfeeding or the feeding of indigestible material as due to the accumulation of unabsorbed bacterial culture media in the intestinal tract rather than to any specific harm caused by the foods themselves. It must, nevertheless, be admitted that certain types of food may have an irritant effect, for example, highly concentrated sugar solutions, large protein curds, or certain lower fatty acids, such as butyric acid. Irritant foods also cause an increased flow of alkaline mucus which not only neutralizes acid but also tends to enmesh food particles and bacteria.

Bacteriologic examinations of the stomach and duodenal contents of infants suffering from severe diarrhea have revealed the fact that organisms of the colon group are usually present in large numbers. These are not found in the case of normal infants, although certain other organisms, especially harmless cocci, may be present. The significance of this rather uniform finding is not entirely clear. It is, of course, possible that the colon bacilli reach the upper portions of the intestinal tract *because* of the fact that a disturbance is present. It seems, however, more likely that their presence is an important factor in bringing about the gastrointestinal disturbance.

Colon bacilli which may exert no harmful effect when growing in the lower bowel may lead to serious disturbances if pres-

ent in the upper portion of the intestinal tract. The effects may be produced in two ways: by actual invasion of the body or by the production of toxic products in the intestine which when absorbed are capable of producing serious symptoms. It has been shown that colon bacilli present in the duodenum may pass through the intestinal mucosa and appear in the thoracic duct lymph in the case of animals in which the duodenal contents have been kept alkaline. The colon bacillus is not infrequently found in blood cultures taken from young infants or those with severe gastrointestinal disturbances associated with the presence of colon bacilli in the stomach and duodenum.

Plantenga has shown that the administration by mouth of the filtrates from broth cultures of B. coli is followed by vomiting and diarrhea, but that the same symptoms are not produced when the filtrates are introduced rectally. The exact nature of the products responsible for the production of the symptoms is not known. Peptones and the ptomaines, cadaverin and putresin, do not produce the symptoms. The symptoms do not appear to be due to endotoxins, for when killed cultures of B. coli are injected, the symptoms do not appear. It has been suggested that histamine and other proteogenous amines may be the toxic agents. Some strains of B. coli are capable of producing large amounts of histamine, or a histamine-like substance; other strains produce none. Histamine, when injected even in minute amounts, causes vomiting, diarrhea, hydrolability and a condition resembling shock, but when introduced into the intestine does not usually cause toxic manifestations. This is explained by the fact that detoxification occurs on passage through the liver and possibly to some extent on passage through the intestinal mucosa, but whether such detoxification occurs completely when the intestinal tract is damaged, or when large amounts of histamine are produced is a question.

It has very generally been believed that acids produced by the bacterial decomposition of carbohydrates are responsible for the production of diarrhea. The fact that diarrheal stools are often fermenting and acid has been the basis of this belief. In any condition in which unabsorbed fermentable carbohydrate reaches the lower bowel, fermentation by the abundant flora invariably occurs; therefore, the fact that the stools are acid is in itself not necessarily indicative of the presence of much acid in the upper

bowel. Some acids are irritating, but it is doubtful if acid production in itself is often the cause of diarrhea. A reasonable amount of acid in the upper intestine is advantageous rather than otherwise, and only a great excess appears to be harmful. Actual observations on infants have usually shown a lesser rather than a greater degree of acidity in the upper intestinal tract in cases of diarrhea. Furthermore, it has been demonstrated experimentally by Lloyd Arnold that alkalinization of the upper intestinal tract favors bacterial growth. When this condition is reproduced in animals by feeding an alkaline buffered solution, the introduction of colon bacilli and other organisms results in severe diarrhea. Acid in the lower bowel may have more effect in delaying absorption and increasing peristalsis than it does in the upper intestine.

Although it appears likely that invasion of the upper intestinal tract by bacterial flora of types found ordinarily only in the lower bowel is a common cause of the serious forms of infantile diarrhea, it should not be inferred that such is the only cause. There are other types of bacteria which may cause diarrhea when introduced by way of the mouth and which in this way gain a foothold in the intestinal tract, as for example, members of the paratyphoid-enteritides group. These organisms, which are to be considered as definitely pathogenic, cause a general body reaction, and at times local lesions in the ileum and colon. Some strains of streptococci also lead to an enteritis. More important than any of these is the dysentery group of organisms, including the "true" dysentery bacillus of Shiga and the para-dysentery group. These organisms are the causative factor in bacillary dysentery which is an acute infectious disease characterized by lesions in the lower ileum and colon consisting of superficial necrosis or ulceration. Diarrhea is one of the symptoms of dysentery. Bacillary dysentery is discussed in detail in Chapter XXI.

Besides the definitely pathogenic organisms there are a group of saprophytic bacteria which may decompose food with the production of irritating substances. When food so decomposed is eaten or if the bacteria in question are introduced into the intestinal tract, where they may decompose food remnants present, diarrhea may be the result. Undoubtedly there are a number of organisms which may fall in this classification, many of which are not clearly defined. Some are definitely proteolytic and it is believed that the products of protein decomposition are respon-

sible for the symptoms. Others appear to produce irritant substances from carbohydrates. Among saprophytes suspected of being the cause of diarrhea are the members of the proteose group, several types of organisms described by Flügge, possibly the gas bacillus and a large group of nondescript organisms which may be found in partially spoiled food. Diarrheas produced by organisms of this type are usually of short duration and are not frequently accompanied by severe manifestations.

There are factors other than the food and bacterial conditions in the gastrointestinal tract which may lead to diarrhea. In some infants hypertonicity and hypermotility of the entire gastrointestinal tract is a constitutional characteristic. This special condition of "gastroenterospasm" is discussed in Chapter XXV. Infections at times appear to disturb the balance of the autonomic nervous system with resulting increased peristalsis irrespective of the character of the food.

That underfeeding may lead to diarrhea would appear, at first sight, unlikely, yet this is a fairly frequent cause. This starvation or hunger diarrhea is more likely to be seen in infants who have been quantitatively and qualitatively underfed for considerable periods of time until they have become undernourished than in the case of healthy infants subject to short periods of starvation or decreased food intake. Too low a total caloric intake or a deficiency in protein, carbohydrate, mineral salts and certain of the vitamins may bring about the condition. In the presence of hunger, hypermotility of the gastrointestinal tract is observed and experimental evidence seems to indicate that such hypermotility is a direct result of lowering of the blood-sugar content. Furthermore, starvation or underfeeding, if prolonged, results in a decrease in the gastrointestinal secretions and also in a decrease in the blood volume and in the volume flow, so that absorption from the intestinal tract is impaired. Hunger diarrhea differs from the other types in that the stools, though numerous, contain relatively little food material. Gastrointestinal colic or cramps are frequent accompaniments.

An inadequate water intake should be included in the category of underfeeding. When the water intake is insufficient to cover the water output by way of the urine, stools, respiration and perspiration desiccation of the body, or anhydremia, occurs. One

of the results of anhydremia is diarrhea, which is often accompanied by symptoms of intoxication.

Diarrhea should not be considered as a disease *sui generis,* but as a symptom resulting from a variety of causes. Many infants with diarrhea also suffer from other manifestations as, for example, vomiting. Indeed, vomiting and diarrhea are often part and parcel of the same condition.

Symptoms of Diarrhea

Diarrhea occurring in well-nourished infants as the result of temporary overfeeding, contamination of the food with non-pathogenic saprophytes, high external temperatures, or the presence of parenteral infection is usually mild in character, of short duration, and unaccompanied by severe constitutional symptoms. The stools are increased in number, and soft in consistency. The color usually varies from yellowish green to bright green. Soft, white soap curds are present if any fat is contained in the diet. Diarrheal stools are likely to be acid in reaction if sugar is being fed. Some glairy mucus is usually present and is an indication of intestinal irritation. Blood and pus are not present in mild cases of diarrhea.

Vomiting frequently accompanies diarrhea. It is seen especially in those cases in which the diarrhea is due to parenteral infection, in which instance vomiting often precedes the first evidences of diarrhea. Only slight temperature elevation is seen in the uncomplicated diarrhea of older infants. Any considerable rise of temperature should arouse the suspicion of the presence of a parenteral infection, or of bacillary dysentery. Some loss of weight is inevitable in all cases of diarrhea, but in the milder types but little weight loss occurs. A marked weight loss is indicative of a more severe or toxic type of diarrhea. There is usually some loss of appetite. Infants with diarrhea are fretful, irritable and appear to suffer from abdominal pain, but severe toxic manifestations are absent in the milder forms.

The severer forms of diarrhea are seen especially in undernourished infants, young infants and those suffering from certain types of acute parenteral infections. To the severe forms of diarrhea, the term "alimentary intoxication" is sometimes applied, because of the toxic manifestations accompanying the gastrointestinal disturbance. The stools are numerous and may

be as many as 15 or 20 in the course of twenty-four hours. At the onset they do not differ a great deal in character from those seen in the milder cases. They are green, acid in reaction and contain food remnants and mucus. Later they may consist of little else than brown watery fluid, often alkaline in reaction. The total volume of the stools may be very great and seemingly out of all proportion to the food and fluid intake. As the stools may be composed largely of fluid, which is absorbed by the diaper, the total volume may not be appreciated. When the stools are collected in a basin and measured, it may be found that the daily volume actually exceeds the fluid intake. Because of the character of the stools, severe cases of diarrhea are sometimes designated by the term "cholera infantum."

Small amounts of blood are occasionally present in the stools, but any considerable amount of blood should arouse the suspicion of bacillary dysentery infection. Gross pus is not seen, but leucocytes may be fairly numerous on microscopic examination. There is sudden and marked loss in weight which is greater than that observed in almost any other condition. A small infant may lose as much as a half pound or a pound in a single day, the loss being chiefly water. Coincident with the loss in weight the appearance of the patient changes greatly. The features become sharpened, the eyes sunken and often fixed in a far-away stare. Later, the conjunctivae lose their luster. The skin becomes dry, gray and inelastic. Fever is always present, and the temperature may be as high as 105° or 106° F. The temperature is lower in the cases in which abundant amounts of fluid are given.

In the early stages, the mental condition is one of restlessness and excitement. Later, if the condition is untreated, the patient lapses into a state of coma. Convulsions are not infrequent and may close the scene. Symptoms of collapse may occur at any time. The pulse is small, sometimes almost imperceptible, often rapid and irregular.

The urine is very scanty, highly concentrated, contains numerous granular casts and some albumin. Occasionally it reduces Fehling's solution. The scanty urine is the result of desiccation of the body.

Remarkable alterations in the character of the respirations may occur. The respirations, instead of being chiefly abdominal, as in the normal infant, become both costal and abdominal. The whole

thorax rises with each inspiration and the accessory muscles are brought into play. The breathing is deep, pauseless, but not especially rapid. It is the same "air hunger" type of breathing as is seen in diabetic coma, and is indicative of acidosis.

The blood is thick, viscid, and does not flow easily when the skin is pricked. A leucocytosis up to 15,000 or 20,000 is regularly observed. The serum protein is increased and may be as much as doubled in concentration. The non-protein nitrogen and urea contents of the blood may be enormously increased. The blood chlorides are usually high and the bicarbonate low.

The symptoms of severe diarrhea are to be explained as the result of the combined action of a number of factors, chief among which are bacterial toxemia, anhydremia and acidosis. (See Chapter XXXII.)

Bacillary dysentery differs in a number of important respects from the other types of diarrhea and is therefore discussed separately. (See Chapter XXI.) It should, however, be pointed out that at the onset the symptoms of bacillary dysentery may be entirely indistinguishable from other forms of diarrhea.

The Effects of Diarrhea on the Body

Diarrhea, if severe or prolonged, may result in serious damage to the body or in death. The chief effects are

(1) Diminished absorption of food resulting in partial starvation
(2) Loss of water
(3) Loss of mineral salts, especially fixed bases
(4) Toxemia from intestinal bacteria

In the presence of diarrhea, the food intake is usually decreased as a therapeutic measure and such food as is taken is only partially digested and absorbed, so that the actual amount of food reaching the tissues may be insufficient to meet the fuel needs of the body. Under such circumstances, the body tissues themselves are necessarily consumed for fuel. The using up of some stored body fat and carbohydrate leads only to a moderate loss of weight and no serious consequences. If, however, partial starvation is long continued, serious malnutrition or athrepsia is certain to result. (See Chapter XIX.) In the presence of diarrhea, fats are especially likely to escape absorption, as much as 25 or 50 per cent

being lost by way of the bowel in the form of fatty acids neutral fats and soaps. A considerable portion of ingested carbohydrate may be destroyed as the result of bacterial action with the production of acids, carbon dioxide, and other substances. The loss in this way may amount to over 50 per cent of the intake. Proteins are usually fairly well absorbed, even in the presence of diarrhea, a loss of more than 15 per cent of ingested protein being unusual. The fat soluble vitamins, like fats, are likely to escape absorption.

In the presence of diarrhea, water absorption is poor and this accounts for the looseness of the stools. The amount of water lost in this way may be very large and is at times as great or greater than the total fluid intake. As a result of this water loss, desiccation of the body tissues and fluids occurs and this in itself may lead to serious symptoms or death. (For further discussion, see Anhydremia, Chapter XXXII.)

The fluid present in diarrheal stools represents in part unabsorbed gastrointestinal secretions. These secretions contain mineral matter. In the gastric juice, acid ions predominate; in the duodenal and intestinal secretions, basic ions; in the combined total secretion of the gastrointestinal tract, bases predominate over acids in about the proportion of 3:2. Failure to reabsorb the secretions from the gastrointestinal tract results therefore in a continuous depletion of the base of the body. One of the results of this is the development of acidosis. (See Chapter XXXII.) Destruction of body tissues because of starvation and desiccation is another factor leading to the loss of mineral matter.

In diarrhea of the infectious type, or bacillary dysentery, the toxemia of the infection may in itself be sufficient to bring about a fatal outcome. Some infants succumb to toxemia even before very severe diarrhea has developed.

The Differential Diagnosis of Diarrhea

Inasmuch as there are a variety of causes of diarrhea and as the treatment to be adopted depends, to a considerable extent, upon the character of the diarrhea, an effort should be made, in each case, to determine if possible the underlying etiologic factor.

A careful case history is essential in arriving at a diagnosis as to the type of diarrhea. It is necessary to know the character of the previous feeding, whether the food mixture has been of suitable composition or whether any constituents have been deficient

or present in excess; whether the feedings have been given at proper intervals and, in the case of artificially fed babies, whether the formula has been prepared from raw or heat treated milk; whether articles of diet other than milk have been given, and whether or not dysentery has been epidemic in the neighborhood. It is also important to know whether the infant has shown any symptoms of infection outside of the gastrointestinal tract, such as, for example, a cold in the head or running ears.

If the food has been one of suitable composition, fed at proper intervals, and has previously agreed with the infant, it may safely be assumed that the diarrhea is due to some extraneous factor, most likely a parenteral infection. Such an infection should particularly be suspected if fever and vomiting have preceded the onset of diarrhea. In order to confirm the diagnosis, a careful search should be made for the presence of infection. The nose, throat and ears, especially, should be carefully inspected. The urine should be examined microscopically for the presence of pus. Ordinarily, however, pyelitis is less likely to lead to diarrhea than either rhinopharyngitis or otitis media. If high temperature is coincident with the development of diarrhea and if no parenteral infection is discovered, one may suspect the presence of bacillary dysentery, but an absolute diagnosis cannot always be made early in the course of this disease. Later, the appearance of blood and pus in the stools or the finding of dysentery bacillus on stool culture may make the diagnosis clear. (See Chapter XXI.)

Diarrhea due to underfeeding is, of course, seen only in infants who have been grossly underfed, especially those who have reached a state of undernutrition. The stools of hunger diarrhea are usually not large, although they may be numerous. They are likely to be dark colored and do not contain much in the way of food remnants.

The diarrhea occurring in infants suffering from gastroenterospasm is chronic except in those cases due to acute infection and is accompanied by other evidences of autonomic imbalance, such as colic, vomiting, and a general tendency to hypertonicity.

Diarrhea due to overfeeding can best be diagnosed from the history of the giving of an unsuitable milk mixture in which some one food element has been present in large excess. Examination of the stools gives but little information as to the original cause of the diarrhea. An infant who has been receiving any

fat in the food is likely to show some neutral fat, soaps, or fatty acids in the stools; and likewise, when diarrhea develops in an infant receiving any carbohydrate, the stools are likely to be acid in reaction, irrespective of the original cause of the diarrhea. The appearance of starch in the stools indicates merely that starch has been fed and that some has escaped digestion. Since this is a normal condition in the case of young infants fed on starch, no significance can be placed upon this finding.

Bacteriologic examination of the stools is of value chiefly in diagnosing the presence of bacillary dysentery. It is of but little value otherwise, as the bacteriology of diarrheal stools ordinarily does not differ essentially from that of normal stools. It is questionable whether the presence of the gas bacillus is of significance. This organism is often found in normal stools and is sometimes present in large numbers in diarrheal stools. It is possible that it may, in some instances, produce sufficient butyric acid from the decomposition of sugars to cause intestinal irritation. Some strains of the gas bacillus also produce histamine. The finding of bacteria of the paratyphoid-enteritides group or of unusual strains of colon bacillus is probably of significance. Some importance has been attributed to the finding of predominantly proteolytic or predominantly fermentative types of bacteria in the stools, and efforts have been made to use these findings as a basis for treatment which consists in reducing either carbohydrates or proteins in the diet. Unfortunately, this procedure has not proved to be of great practical value because of the fact that the findings are not clear cut. Both normal and diarrheal stools contain both types of bacteria and either may predominate. Alterations in the diet only occasionally change the predominating type of flora and such change is by no means regularly associated with clinical improvement.

Prophylaxis of Diarrhea

Infantile diarrhea is largely preventable. Short periods of diarrhea may occur in the case of almost any infant, even with the best of care, but serious or fatal diarrhea need not occur if an infant is fed properly and lives under good hygienic conditions. Breast feeding is one of the best means of preventing severe diarrhea because there is but little chance of introduction of harmful microorganisms into the intestinal tract; and because human milk

is readily digestible and when fed favors the maintenance of conditions in the gastrointestinal tract which are unfavorable to the growth of bacteria capable of producing gastrointestinal disturbance. Furthermore, breast-fed infants are more likely to receive sufficient food than those who are artificially fed on dilute milk mixtures, and, as the result of the better nutrition, are less susceptible to infections.

In the case of the artificially fed infant, the chief essential in the prevention of diarrhea is that the milk should be free from harmful bacteria. The general improvement in the market milk supply and the almost universal custom of heating milk before feeding have been most important factors in the reduction of mortality from the diarrheal diseases. Heat treatment of milk results not only in reducing the bacterial content, but also in rendering the milk more digestible. In these respects, boiling is more effective than pasteurization; dried milk is more digestible than boiled milk, and evaporated milk even more readily digestible, as well as being absolutely sterile.

The chief methods of modification of milk which are important in preventing diarrhea are those designed to reduce the size of the curds and to neutralize the buffer substance. The various methods for reducing the size of the curds have previously been discussed. The most effective of these are heat treatment and addition of acid. Dilution also reduces the size of the curds but is undesirable because it is likely to result in underfeeding. Acidification reduces the size of the curds and also neutralizes the buffer substance. Acidification is not essential in the case of normal infants, but is a safeguard against diarrhea because harmful bacteria are not likely to multiply in acidified milk and abnormal conditions in the stomach and duodenum are much less likely to occur even in the presence of infection or during hot weather when acidified milk is fed. Heat treated acidified milk is, for these reasons, an excellent food for the prophylaxis of diarrhea.

Further modification of milk should consist in the addition of carbohydrate which is not too readily fermentable nor added in too large an amount in proportion to the protein present. A carbohydrate addition greater than one of carbohydrate to ten of milk should, in general, be avoided, but on the other hand, an amount less than one of carbohydrate to twenty of milk is hardly

enough to meet the nutritional needs. When milk is suitably modified as indicated there is but little danger that overfeeding with the mixture will cause diarrhea.

A sufficient intake of water, especially during the warmer months of the year, is an important factor in the prophylaxis of diarrhea.

Overheating of the body results in decreased digestive capacity, which is likely to result in diarrhea. Infants should not be overclothed or kept in hot, humid rooms, but they can stand considerable summer heat if kept out of doors or in well-ventilated quarters, given plenty of water and not overclothed.

Maintenance of the nutrition is one of the important means of preventing diarrhea. The undernourished infant is especially likely to suffer from diarrhea and from the infections which lead to diarrhea. Many more infants have died from diarrhea which, in the ultimate analysis, has been the result of underfeeding than have died from overfeeding.

Diarrhea occurring in the case of infants who have been fed properly is in most instances the result of infection somewhere in the body. This frequent type of diarrhea may be prevented by prompt recognition and suitable treatment of infections as they occur.

Treatment of Diarrhea

It is not always possible to tell at the outset of diarrhea whether the case will be of a mild or severe type, hence it is safer to consider all cases of diarrhea as potentially severe and to treat them accordingly. A day or two of proper treatment early in the course of diarrhea may be far more effective than a week or two of treatment later in a neglected case.

General Principles

There are certain general principles of treatment which apply to all forms of diarrhea. These are:

(1) Recognition and suitable treatment of parenteral infections.
(2) Rest of the gastrointestinal tract.
(3) The giving of food adapted to the limited digestive capacity.
(4) Restoration and maintenance of the fluid balance.
(5) Restoration and maintenance of the mineral balance.
(6) Blood transfusion in severe cases.

In addition to the above general measures, the use of specific sera may be considered in certain types of cases, as, for example, bacillary dysentery and certain B. coli infections.

In the treatment of any case of diarrhea, the first step should be to determine, if possible, the underlying cause. A careful search for any form of parenteral infection should be made. More information is often obtained from examining the ears of an infant suffering from diarrhea than from examining the stools. In those cases of diarrhea which are caused by parenteral infection, the results from treatment are unsatisfactory so long as the infection is present.

In any form of diarrhea, the digestive function is necessarily impaired and the intestinal tract is either irritated or is the seat of abnormal bacterial infection. In any event, food is imperfectly digested and absorbed and any unabsorbed excess may be decomposed and cause further irritation. An excess of food not only serves no good purpose, but is a source of potential harm. In all cases of diarrhea, it is essential that the intestinal tract should be given a rest for at least a period of time. The only fully satisfactory method of accomplishing this is the withholding of all food for a period of time, during which only water is given. The duration of the period of starvation will depend upon the age and nutritional condition of the infant, the severity of the diarrhea, and the reaction to starvation. A single starvation period at the onset of diarrhea is preferable to repeated starvation intervals or to prolonged periods of underfeeding. In bacillary dysentery, and in many of the diarrheas due to parenteral infection, starvation, even if prolonged, is likely to have but little effect in causing a cessation of the diarrhea.

Following the starvation period, the food given a patient with diarrhea should be nonirritating, readily digestible, a poor culture medium for bacteria, and of a type which tends to bring about normal chemical conditions in the gastrointestinal tract. A relatively low fat content is desirable inasmuch as fat tends to delay the emptying time of the stomach and predisposes to vomiting, hence skimmed or partially skimmed milk is preferable to whole milk. Such milk as is used should be so treated that only very fine curds are produced during the process of gastric digestion. Boiled, evaporated, dried or acidified milk meets this indication. The milk itself should, of course, be sterile and should in addition

be a poor culture medium for bacteria and should be of such character that when fed conditions are produced in the gastrointestinal tract which are unfavorable for bacterial action. Acidified milk meets these indications. Inasmuch as carbohydrates and especially the simple sugars are good culture media for bacteria, only moderate amounts of carbohydrate should be given in the form of the more readily absorbable and less easily fermented sugars. Dextrin, or dextrin-malt mixtures, are preferable to milk sugar or cane sugar.

Foods which meet the above requirements and which have proved especially serviceable in the feeding of infants with diarrhea, are acidified, whole or partially skimmed boiled, evaporated or dried milk, and protein milk. To these various forms of milk, moderate amounts of dextrin, dextrimaltose or corn syrup are added. The parenteral administration of dextrose solution may be resorted to as a means of introducing extra food.

In all forms of diarrhea, it is essential that a sufficient amount of fluid be given to maintain a normal water balance of the body. Unless this can be done other methods of treatment are likely to fail. The amount of water which may be taken by mouth is often limited because of persistent vomiting. A larger total amount of water can often be taken when given in very small amounts at frequent intervals than when larger amounts are given less frequently. When it is impossible to administer a sufficient amount of fluid by way of the gastrointestinal tract to prevent the symptoms of dehydration, recourse must be had to parenteral means of fluid administration. Fluid may be given subcutaneously, intravenously, or intraperitoneally. Intravenous administration restores more quickly a depleted blood volume and is indicated in all cases of acute anhydremia accompanying diarrhea. By the use of the subcutaneous and intraperitoneal routes, much larger quantities of fluid may be introduced than intravenously and the effects are more lasting. For intravenous use, dextrose solutions of a strength of 10 or 15 per cent are preferable to saline solutions. For subcutaneous or intraperitoneal injection, saline solutions are usually used. Ordinary physiologic sodium chloride and Ringer's solution are suitable for the treatment of mild degrees of dehydration accompanying diarrhea. In the more severe cases, however, the usual sodium chloride solutions possess certain dis-

advantages. A preferable solution is that described by A. F. Hartmann (see page 323). The methods of maintaining the fluid balance are discussed in further detail in Chapter XXXII.

Inasmuch as in all severe cases of diarrhea there results a loss of mineral matter in which the fixed bases predominate, restoration of the mineral content of the body is essential. This may to some extent be accomplished by means of the food, but is more quickly accomplished especially in severe types of diarrhea, by the parenteral administration of salt solutions designed to restore the depleted elements. (See Chapter XXXII.)

In the severer types of diarrhea, repeated transfusions with citrated whole blood are of the greatest benefit. Transfusions improve the circulation and lead to improvement in the digestion and absorption of food. The injected blood also supplies materials for the reconstruction of damaged body cells and may have some effect in increasing the infant's resistance to infection.

Treatment of Diarrhea in the Breast-Fed Infant

In infants fed exclusively at the breast, diarrhea is but rarely severe or of long duration. A moderate increase in the number of stools and a slight change in their character if unaccompanied by fever or constitutional symptoms is of but slight significance. The only treatment indicated, in such instances, is temporary reduction in the food intake, which may be accomplished by lengthening the feeding intervals (if less than four hours) and shortening the time of each nursing.

When the diarrhea is more severe and especially if fever is present, a search should be made for parenteral infections, since these are the most common causes of diarrhea in breast-fed infants. Diarrhea is not an indication for weaning, although the food intake should be diminished for a period. At the start, several feedings may be omitted and the infant given instead water or barley water. This may be sweetened with saccharin (¼ gr. to the bottle). If the diarrhea is severe, starvation should be continued for as long as 24 hours. It is not usually necessary to continue it longer than this in the case of previously healthy breast-fed infants. During the period of starvation, the mother's breasts should be emptied by manual or mechanical expression (see Chapter XI). When the feedings are resumed, the intervals should not be shorter than every four hours, and the

infant should nurse for only a few minutes. It is advisable to offer several ounces of water previous to putting the infant to the breast. If water is offered from a bottle just preceding the nursing, the infant is not likely to nurse as vigorously or to take as much milk and such milk as is taken will have a low fat content if the breast is incompletely emptied. Supplemental foods, especially fruits and vegetables, should be omitted during the period of acute diarrhea. In most instances, diarrhea of breast-fed infants responds to the simple treatment of temporary withdrawal of food followed by a diminished food intake. In some instances, recovery is hastened by the administration of a buffered lactic acid solution (see page 211) preceding each nursing. From 1 to 3 ounces of this solution may be given. Small feedings of skimmed lactic acid milk or protein milk just preceding the nursing sometimes have a beneficial effect. Some infants, however, who are accustomed only to feedings at the breast, may refuse any acidified foods because of the sour taste.

If the diarrhea fails to respond within a reasonable time to these simple methods of treatment, the presence of unrecognized parenteral infection should certainly be suspected. In rare instances a continuation of the symptoms is due to bacillary dysentery infection.

The Treatment of Mild Cases of Diarrhea in Artificially Fed Infants

In well nourished artificially fed infants, and especially in those over four or five months of age, diarrhea is usually mild in type and responds promptly to suitable treatment. In very young infants, in those who have been fed on unbalanced food mixtures, high in carbohydrates (such as sweetened condensed milk) and in infants who have suffered from chronic or repeated acute infections, diarrhea is likely to be much more severe.

The first step in the treatment should be to ascertain, if possible, the cause of the diarrhea. A search should be made for any infections and, if found, these should be treated appropriately. If such an acute infection as otitis media is discovered and the eardrum opened, there may be but little need for further treatment. If the food has been well balanced and especially if some form of acid milk has been the basis of the diet, the regular feedings may be continued but in smaller amounts for a few days. In those

cases in which the infant has been receiving a sweet milk formula, it is often advantageous to resort, at least temporarily, to a formula prepared from acidified milk. It is advantageous to reduce the sugar content somewhat and such sugar as is used should be of the maltose-dextrin type.

If no source of infection can be found, or if such infection as is found is not amenable to immediate treatment, a stricter regulation of the diet is necessary. A period of starvation should be instituted, during which the infant receives an abundant amount of water. The length of the starvation period will depend upon the age and nutritional condition of the infant as well as upon the severity of the diarrhea. The omission of one or two feedings often suffices. During the starvation period, a buffered lactic acid solution may be given. (See page 211.)

In the presence of diarrhea, the intestinal tract usually empties itself well without the aid of cathartics, but if the infant is known to have taken an unsuitable article of diet, or if the diarrhea has barely started, a cathartic may be administered in order to remove the offending material before damage has occurred. Usually, however, the infant is seen after the diarrhea has continued for some time and no good is to be accomplished through the use of a cathartic. Certainly not more than one dose should be administered in any event. If a cathartic is used at all, castor oil is as satisfactory as any. A suitable dose is from one-half to one tablespoonful, depending upon the size of the infant.

After a short period of starvation, the feedings may be resumed. In the case of older infants unaccustomed to the taste of acid milk, as suitable a feeding as any is boiled skimmed milk without added sugar. The amount of this given at first should not be more than about one-half or two-thirds the usual volume taken at a feeding by the infant. After a few feedings, the amount of milk given may be increased and if the diarrhea shows signs of cessation, a moderate amount of carbohydrate is added. The proportions should not be greater than one of sugar to twenty of milk. If all goes well, whole milk may gradually be substituted for the skimmed milk in the formula. Additional carbohydrate may then be added until the infant is receiving the usual formula for the age.

In infants who have been taking acid milk mixtures, the only change in the diet indicated in the presence of mild diarrhea is a

reduction in the sugar content and a moderate reduction in the total volume of food taken, the difference being made up with water. If the diarrhea does not respond promptly to this form of treatment, or if there are constitutional symptoms present, skimmed lactic acid milk or protein milk should be used as in the treatment of the severe types of diarrhea discussed below. The decision as to how rapidly the food intake may be increased will depend more upon the general constitutional symptoms than upon the character of the stools. The infant should be carefully watched for the appearance of any of the symptoms of intoxication, especially fever, desiccation, grayness of the skin and the nervous manifestations. Any sudden or marked loss of weight is of great significance. When any of these symptoms are present, the diet should be increased only with the greatest caution and in addition other methods of treatment, such as the administration of fluids and transfusion are indicated.

Treatment of the Severe Forms of Diarrhea

(Alimentary Intoxication)

The severer forms of diarrhea are seen almost entirely in artificially fed infants who have received unsuitable food mixtures, in very young infants and in those who have suffered from chronic or repeated acute infections. Under these conditions a diarrhea which, at the outset, may apparently be mild, may be the forerunner of a severe form with toxic manifestations.

In the treatment of the severe toxic diarrheas the first step always should be to ascertain, if possible, the underlying cause. Of the various types of parenteral infection, otitis media is perhaps the most frequent contributory cause, although the ordinary middle ear infections are not likely to result in severe diarrhea unless the infant is already in poor nutritional condition. In athreptic infants, the onset of otitis media is often insidious and gastrointestinal symptoms may appear before any local manifestations of ear infection are detected. The first changes in the eardrum consist only of a lack of luster and transparency and it may be a number of days before definite redness and bulging are observed. Early paracentesis is often followed by improvement in the gastrointestinal symptoms, but such a favorable result does not follow in all cases. The symptoms may persist, even though the ears are freely opened. In a certain number of instances the infec-

tion spreads to the mastoid antrum and surrounding cells and becomes walled off. There may be but slight local evidences of such infection (see Chapter XXVIII), but it may be necessary to resort to antrotomy or postauricular drainage in order to clear up the infection.

There is a variety of otitis media due to a virulent toxin producing strain of hemolytic streptococcus which occurs occasionally in epidemic form, and which may lead to severe toxic diarrhea even in the case of infants who are well nourished and have previously been well fed. This phase of the subject is discussed in further detail in Chapter XXVIII.

Otitis media is, of course, only one of the infections which may be followed by severe gastrointestinal disturbances. A careful search should be made for any other possible infection and, if found, appropriate treatment should be instituted. Whatever the cause of the diarrhea, infectious or otherwise, conditions in the gastrointestinal tract are essentially the same, and the same principles of treatment should be followed.

The most effective methods of treatment of the severe toxic diarrheas are those based upon the original observations of Finkelstein. The technic of treatment has been improved from time to time until at present fairly satisfactory procedures are available. The modern treatment of the severe toxic diarrheas has been summarized in an admirable manner by Powers,* and the outline of treatment which follows is based, for the greater part, upon his comprehensive plan.

Inasmuch as in all severe cases of diarrhea, dehydration occurs, and may in itself lead to serious consequences, active measures should be taken to restore the fluid content of the body. Fluid should be administered by mouth, intravenously, subcutaneously or intraperitoneally. Water should be given by mouth in as large amounts as can be retained. The administration of small amounts of water at frequent intervals is usually a more effective means than the giving of larger volumes at a time, which may be vomited. The use of a buffered lactic acid solution possesses certain distinct advantages over plain water, as such a solution tends to render the contents of the stomach and of the upper portions of the small intestine sufficiently acid to inhibit bacterial growth. The solution

*Powers, Grover F.: Am. J. Dis. Child., xxxii, 232, 1926.

which we have found most serviceable for this purpose is one devised by A. F. Hartmann. This has the following composition:

BUFFERED LACTIC ACID SOLUTION

Lactic Acid, U.S.P.	15 c.c.
Sodium hydroxide, 10%	20 c.c.
Water to	1000 c.c.

We have found it convenient to prepare a concentrated stock solution of ten times this strength and to dilute 1:10 with water before use. The solution may be flavored with a small amount of saccharin. In many instances as much as 5 per cent of Karo Syrup may be added to the acid solution fed without resulting in any appreciable harm. Such a mixture has a taste which is not unpleasant, resembling that of lemonade. Some infants, however, refuse it.

In severe diarrhea with dehydration, it is difficult or impossible to introduce a sufficient amount of fluid by mouth so that recourse must be had to parenteral means. Physiologic sodium chloride solution or Ringer's solution has been chiefly used for parenteral administration. Such solutions, although effective in many cases, possess certain distinct disadvantages. The sodium chloride content is considerably higher than that of the blood and tissues so that the excess must be eliminated. In the presence of anhydremia, renal function is impaired so that there is likely to be an accumulation of chlorides in the blood. Any further increase in the chloride content as the result of salt administration is usually accompanied by a corresponding decrease in bicarbonate. In cases of this type a dextrose solution is preferable to one of sodium chloride as the dextrose may be utilized, leaving behind only water. Dextrose in a concentration of 8 to 10 per cent may safely be given intravenously or subcutaneously.

In cases of dehydration accompanied by acidosis, the administration of alkali is indicated. (See Chapter XXXII.) A fluid for parenteral injection which meets all of the requirements is the "combined solution" of Hartmann (see page 323). This solution of mixed salts containing sodium lactate is designed to replenish the depleted mineral content of the body, yet contains a concentration of sodium chloride no greater than that of normal blood, so that accumulation is not to be feared. The solution is neutral in reaction, yet supplies potential alkali

in the form of sodium lactate convertible into sodium bicarbonate in the body. The solution possesses the special advantages of Ringer's solution, of dextrose and of alkali without their disadvantages. We have found this solution most satisfactory for routine use. Dextrose solution should, however, be used when it is desired to give an extra amount of food, and sodium bicarbonate in cases of very marked acidosis.

In cases with cyanosis oxygen administration is indicated and is of value. The most satisfactory method of administering oxygen is to place the infant in an oxygen tent.

In beginning the treatment of a toxic case of diarrhea, the intravenous injection of fluid should be the first step as this immediately increases the blood volume and volume flow and probably improves intestinal absorption. This should be followed by subcutaneous or intraperitoneal injections. More fluid can usually be introduced intraperitoneally and with less pain than by the subcutaneous route. The amount given intraperitoneally is limited only by the size of the abdominal cavity and the injections may be repeated as frequently as every six to twelve hours, provided the fluid is absorbed. (The technic of intraperitoneal injection is discussed in Chapter XXXIV.) It is surprising what enormous quantities of fluid are sometimes required to restore the body fluids in cases of dehydration with diarrhea. There is little or no danger in giving too much fluid, a very real danger in not giving enough. Fluid administration should be pushed to the point of relieving the symptoms of anhydremia and of restoring and maintaining the weight. In some instances it is necessary to continue daily parenteral administration of fluid for a period of weeks. One small infant under our care was given 72 intraperitoneal injections over the course of several weeks, the total volume of injected fluid being over ten gallons! (Ultimate recovery followed.)

The good effects of fluid administration are likely to be more lasting if combined with blood transfusion. It is, however, better not to give a transfusion until the fluid content of the body has been restored partially. The optimum time for the first transfusion is about twelve hours after the initial fluid injection. Repeated small transfusions are likely to be more effective than a single large one. The amount given at each transfusion should be from 20 to 40 c.c. per kilogram of body weight. Citrated blood

is used and a sufficient amount obtained from the donor at one time for three or four transfusions. The blood may safely be kept in an ice box for as long as three or four days before being used. (For the technic of blood matching and transfusion, see Chapter XXXIV.) Three or four transfusions at intervals of eighteen to twenty-four hours early in the course of the diarrhea are usually sufficient, although subsequent developments may be such that additional transfusions are required later.

In the treatment of all cases of severe diarrhea with symptoms of intoxication and dehydration, a period of starvation should be instituted. This should, in no case, be less than twelve hours and may be as long as three or four days. During the starvation period, the infant receives only water, barley water, or buffered lactic acid solution with a little added sugar. Markedly undernourished infants do not stand starvation well, but even in these starvation is less dangerous than feeding, and furthermore such food as is given during the acute stages of the condition is not well utilized. The dangers of starvation in athreptic infants may be minimized, to some extent, by repeated intravenous or subcutaneous injections of 10 per cent dextrose solution.

In determining the length of the period of starvation, the general symptoms of the infant, rather than the character of the stools should be the guide. So long as the temperature is high, the infant's appearance toxic, the skin dry and gray and the initial weight loss but slightly regained, the administration of food by mouth is dangerous. As these manifestations of toxemia disappear, feeding may cautiously be begun. The initial feeding should always be some form of acidified milk. Half skimmed acidified milk or protein milk, with or without the addition of 3 to 5 per cent of difficultly fermentable sugar are suitable foods. Even more effective than these is a combination of dried protein milk with buffered lactic acid solution. The formula has the following composition:

Dried protein milk	1 part by weight
Buffered lactic acid solution	10 parts

The lactic acid solution is the same as that previously described. (See page 211.) The acidity or hydrogen-ion concentration of this solution is sufficient to inhibit bacterial growth, but not so great as to interfere with intestinal digestion or

to cause irritation. The presence of the sodium lactate as a buffer permits the administration of even larger amounts of acid than would otherwise be possible and tends to stabilize the reaction. We have noted the prompt disappearance of colon bacilli from the stomach following the feeding of this mixture and coincident with this there has usually occurred an improvement in the general condition of the patient and a decrease in the number of stools.

Whether lactic acid milk or protein milk is used, the feeding intervals should not be more frequent than every four hours and the total volumes given at individual feedings should at first not be more than 40 per cent of those ordinarily taken by the infant when well. If the giving of this amount of food is followed by no untoward symptoms, at the end of 48 hours a moderate increase in the volume of the feedings may be made. The daily increase should, however, usually not be greater than ½ ounce at each feeding. Too rapid increases in the amounts of food may result in a loss of all the ground gained. If there are no marked toxic symptoms, gradual increases in the food may be made from day to day, even though the stools may not have become entirely normal. So long as the stools are loose and watery, however, only minimal amounts of carbohydrate should be added and no increases in the amount of carbohydrate should be made until the character of the stools has shown some improvement. In the meantime the carbohydrate needs of the infant may be covered partially by the parenteral administration of dextrose.

By the end of four or five days it is usually possible to add some carbohydrate, especially in the form of a relatively non-fermentable sugar, such as dextrin, dextrimaltose or Karo syrup. The amount added at first should not be more than 3 to 5 per cent or one of sugar to 20 or 30 of the milk mixture. During all this period the food intake will not be sufficient to cover the caloric requirements and no gain in weight other than that due to restoration of the water and mineral contents of the body is to be expected. From this point on the food intake may be increased by the substitution of whole lactic acid milk for partially skimmed, a concentration of the protein milk mixture from one to ten of diluted fluid to one to eight and by the addition of increasing amounts of difficultly fermentable carbohydrate, up to the proportion of one of sugar to 12 or 15 of milk mixture.

There is a real danger in keeping the food intake of these infants at too low a level for a long period of time. If one pays too much attention to the character of the stools, the feedings may never be increased up to anywhere near the maintenance requirement, and in consequence the infant may develop a starvation diarrhea and succumb to inanition. If the initial period of starvation has been sufficient, a prolonged period of underfeeding is not usually necessary.

In the treatment of all of these cases, one should always bear in mind the possibility of an intercurrent parenteral infection, even though none may have been discovered at the outset. Not infrequently in infants recovering from severe diarrhea, an exacerbation of all of the symptoms occurs as the result of such an infection as otitis media or pyelitis. The otitis in these cases is likely to be of a different character from that usually observed in infants not suffering from diarrhea. The infecting organism is very often a colon bacillus or other intestinal type. There may be extensive necrosis of the mastoid with practically no local evidences. In cases with this complication, the prognosis is bad, even though the ears are opened and drained. Mastoid operation is indicated, but the chances of recovery are much less than in cases of primary mastoid involvement.

There is one type of severe diarrhea which fails to respond to the treatment outlined above. This particular variety of diarrhea appears to be due to infestation of the intestinal tract with organisms capable of decomposing protein with the production of toxic products. This is a relatively rare type of diarrhea, but is sometimes seen in epidemic form. This particular type of diarrhea must have been more frequent in the past than it is at the present day. It is discussed at length in many of the older textbooks, but is referred to only occasionally in the more recent literature. We have only once observed such an epidemic. It has not been determined just what organism is responsible, but it is not one of the ordinary forms of dysentery bacilli. At the outset, it is difficult or impossible to distinguish this from any other form of diarrhea. Fever, dehydration and toxic manifestations are marked. Convulsions are of more frequent occurrence than in other types of diarrhea. The stools are more likely to be foul in odor rather than sour. Blood and pus are not observed in the stools. The giving of milk in any form to these patients,

even though it be acid milk or protein milk in small quantities results in an exacerbation of all the symptoms, and if the usual methods of treatment are followed, the mortality is high. These patients tolerate carbohydrate fairly well, and indeed seem to be benefited by reasonable amounts of carbohydrate.

The treatment of diarrhea of this type should be the same as that of the other severe forms of diarrhea except for the character of the feedings. After the initial period of starvation, feedings are begun with an 8 to 10 per cent solution of Karo syrup or dextrimaltose. Later arrowroot or corn starch gruels are given. No milk of any sort is allowed until the temperature has fallen and the symptoms of toxemia have disappeared. It may be a week or ten days before it is safe to add milk, or other protein-containing food. Some form of acid milk may then be added to the diet in small amounts. If well tolerated, the quantity may be increased until finally the infant is returned to the usual diet for the age. An initial cathartic appears to be of more value in these cases than in the ordinary cases of diarrhea.

Medicinal Treatment

Drugs have relatively little place in the treatment of diarrhea. Cathartics, as previously stated, should not be used except in the very early stages of diarrhea, and not always then. If a cathartic is used at all, only a single dose should be given. Repeated doses of cathartics serve only to keep up irritation of the intestinal tract. If a cathartic is to be used, castor oil is usually the most satisfactory, although a simple saline or milk of magnesia may be used. Calomel had best be dispensed with; it possesses no advantages over other cathartics and may cause severe irritation of the gastrointestinal tract.

Opium in the form of paregoric may be used when the diarrhea is severe, prolonged and accompanied by marked water loss. Opium also serves the purpose of relieving pain and of allowing the infant to obtain some rest. Marked abdominal distention is a contraindication to the use of opium. Paregoric may be given in small doses at frequent intervals, up to the point of the physiologic effect of contraction of the pupils. Standing orders for the administration of paregoric or of any other opiate should not be given; a certain number of doses should be ordered, and the order then repeated only to meet definite indications. The initial dose of

paregoric in the case of a young infant is usually 5 minims; for a larger infant 10 to 15 minims. The dosage may be repeated every two to three hours for six or seven doses, or until the physiologic effect is obtained. After this the dosage should be decreased.

Bismuth preparations have been much used in the treatment of diarrhea. They are supposed to act as demulcents, coating over the irritated intestinal mucosa and also to have a slight antiseptic effect. Bismuth subnitrate should not be used as the nitrate is likely to be reduced to nitrite by bacterial action and to give rise to toxic manifestations. If bismuth is used at all, the subcarbonate is to be preferred. Small doses could hardly be expected to have any effect in coating the very large intestinal area. We have never been impressed with the value of bismuth in the treatment of diarrhea.

It has been claimed that purified kaolin is capable of adsorbing bacterial toxins and that its administration in cases of diarrhea is of value. The usual dose recommended is from 2 to 3 teaspoonfuls in a little water following the feedings.

Various antiseptics have been used with the idea of inhibiting bacterial growth in the intestinal tract. Most of the so-called intestinal antiseptics are entirely ineffective, for when given in sufficient amounts to exert any bacterial inhibiting action they are likely to prove toxic for the infant. The water soluble antiseptics, such as the dyes, are quickly absorbed from the upper intestine, and can therefore exert an effect for only a very short time. Colloidal silver preparations of the argyrol type (mild silver protein U.S.P.) are not readily absorbed from the intestinal tract. We have found that these preparations have but little inhibiting effect upon bacterial growth in milk unless present in a concentration of at least 1:500. We have used these preparations as additions to the milk formulas fed, making the concentration 1:300 so as to allow for subsequent dilution by the gastric and intestinal juices. In preparing the feeding the silver is added just before the feeding is given. In order to prepare a 1:300 dilution, 3 c.c. of a 10 per cent solution of the silver preparation is added to 100 c.c. of the formula (1 teaspoonful of 10 per cent mild silver protein solution to each four ounces of formula). Following the administration of silver compounds the stools become black in color. They are, however, by no means sterile. Although we have never seen argyria occur as the result

of the absorption of silver when used as indicated, this possibility must always be borne in mind, and it is inadvisable to continue the administration of silver for more than a day or two. Its use should be restricted to extremely severe cases of diarrhea or dysentery.

Atropine is of value in those cases of diarrhea which are accompanied by marked gastrointestinal spasm and vomiting. The atropine appears to relieve the colicky pains and to decrease the amount of vomiting. The methods for the administration of atropine are discussed in Chapter XXV. It should be noted in this connection that the administration of atropine sometimes leads to elevation of temperature.

Anti-coli serum has been recommended by Plantenga for the treatment of those cases of diarrhea which are associated with colon bacillus infection in the upper intestinal tract. A polyvalent serum is used and the dosage is from 20 to 30 c.c. It is stated that the injection of serum is often followed by a disappearance of the colon bacilli from the stomach and duodenum, but that the general symptoms are not improved until after a number of days. This method of treatment would appear to be worthy of further trial.

CHAPTER XXI

BACILLARY DYSENTERY

(Ileo-Colitis, Infectious Diarrhea)

Bacillary dysentery is an acute, specific infectious disease characterized by lesions in the intestinal tract, chiefly in the lower ileum and colon. The causative organism is the dysentery bacillus or one of the members of the dysentery group. This group includes the "true dysentery" bacillus of Shiga and the closely related strains of paradysentery bacilli. Among the latter are included the "Flexner," "Hiss-Russell" and "Strong" types. A number of subdivisions of these strains have been described which differ from each other in fermentation reactions and immunologic properties. The Flexner and Hiss-Russell types are more frequently the cause of dysentery throughout the United States than is the Shiga type.

Dysentery bacilli gain access to the body by way of the gastrointestinal tract. Contaminated milk and water are the most frequent sources of infection. Such organisms as escape the bactericidal action of the gastric juice and intestinal secretions find lodgment in the mucosa of the lower ileum and colon.

Pathology

The lesions of dysentery are found chiefly in the colon and to a lesser extent in the lower quarter of the ileum. In mild cases there may be only a superficial hyperemia with outpouring of mucus. In more advanced cases there is considerable round cell infiltration of the mucosa and submucosa. In all severe cases some superficial necrosis occurs. This may be extensive so that almost the entire mucosa of the colon is covered with a necrotic pseudomembrane, resembling that seen on the throat in cases of diphtheria. In the majority of cases, however, the necrosis is confined to the area overlying the lymph nodules where it usually results in localized ulcerations. The ulcers are fairly deep with overhanging edges. Hemorrhages occur due to erosion of superficial blood vessels, but the ulcers rarely perforate. Secondary infection of the ulcerated areas with pyogenic organisms is com-

mon. The mesenteric lymph nodes draining the affected areas are usually swollen. In most cases of dysentery the intestinal lesions are of such nature that complete repair is possible, but when extensive ulceration is present, repair is likely to be slow, and in some instances a chronic colitis may persist for months or years.

The liver is enlarged and fatty.

Dysentery bacilli are present in the lesions throughout the course of the disease, but except in rarest instances do not invade the blood stream. In most cases of dysentery the causative organism may be isolated from the stools through the use of suitable cultural methods. (See Chapter XXXIV.) The organisms are not likely to be found during the first day or so of the disease or after the subsidence of the temperature.

Specific agglutinins for the various strains of dysentery bacilli may be demonstrated in the blood serum toward the end of the first week of the disease. The presence of such agglutinins is of some diagnostic value, especially in those cases in which the clinical symptoms are not clear cut and in which it has not been possible to isolate the organisms from the stools. The agglutination test is performed in the same manner as the Widal reaction, using strains of the several dysentery bacilli.

Bacillary dysentery is a self-limited disease in which recovery depends upon the development of immunity on the part of the body. The immunity in question consists in the production of antitoxins and agglutinins and until such immunity has developed, the disease will continue irrespective of the character of the feeding.

Symptoms

In typical cases of dysentery in infants the onset is sudden with severe prostration and high temperature. The infant may vomit a few times, but vomiting is neither a constant nor a persistent symptom. Convulsions may occur, especially in the case of young infants and those suffering from infection with the Shiga type of dysentery bacillus. The initial toxemia may be so severe that the infant succumbs within less than twelve hours, even before any marked degree of diarrhea has developed. The general condition of the patient is one of extreme prostration and apathy closely resembling that of typhoid fever. The severe abdominal pain may result in periods of restlessness and irritability. Refusal of

food and even of water is commonly observed, so that it may become necessary to feed by gavage. The abdomen is usually distended and often tender.

At about the time of the onset of fever or shortly thereafter the stools become loose. They are at first thin, watery, and contain large amounts of mucus, but do not differ greatly from those seen in other forms of diarrhea. In typical cases, by the second or third day, or at times even on the first day, blood appears in the stools, either as small flakes or in sufficient amounts to color the whole stool. Microscopic pus is present in the stools early in the course of dysentery. Later it is present in macroscopic quantities. In some instances shreds of necrotic membrane may be seen. The characteristic stools of well-developed cases of dysentery are small, brownish-green and may consist of little but blood, mucus and pus. They have a peculiar musty odor, resembling that of wet hay. The stools are very frequent and may be passed as often as every half hour throughout the day. Accompanying the passage of the stools there is marked abdominal pain and tenesmus. In those cases of dysentery in which the course has been prolonged and extensive ulceration has occurred, the stools may not resume their normal character until weeks or months after the temperature has fallen to normal and other symptoms of the disease have disappeared. The stools may continue to be numerous, eight to ten a day, and contain undigested food elements and some mucus, although pus and blood may no longer be present.

The urine is scanty, due to the diminished fluid intake and the loss of fluid by way of the bowel. During the height of the disease traces of albumin and a few granular casts are usually present in the urine. Fairly numerous pus cells appear in the urine in most severe and prolonged cases of dysentery. Colon bacilli are often present, dysentery bacilli but rarely. Acetone and diacetic and oxybutyric acids are often present in traces and sometimes in large amounts, even early in the course of the disease. Acetonuria is more frequent in dysentery than in other forms of diarrhea and is probably the result of liver damage by the toxins of the dysentery bacillus.

The blood shows a polymorphonuclear leucocytosis, the total white count being between 18,000 and 25,000. In some instances

there is a marked leucopenia at the onset of the disease. This initial leucopenia may lead one to suspect the presence of typhoid fever.

The course of dysentery is variable, the average duration being from two to three weeks. During this time the temperature is likely to be of a continuous type with only occasional remissions. As convalescence begins, the temperature falls by slow and irregular lysis. The temperature may fall to practically normal for a day or two and then rise again and remain elevated for a number of days. At any time during the course of the disease the temperature may be influenced by complicating infections, especially pyelitis, otitis media or pneumonia. The course of dysentery may be greatly prolonged, especially in undernourished infants who have survived the acute period; in these the temperature and all of the other symptoms may persist for as long as one or two months. In other cases, death may occur within the first twenty-four hours as the result of an overwhelming toxemia. Abortive attacks are also seen, especially in older infants and during epidemics. There may be only slight fever for two or three days, the stools may be loose but not of the characteristic dysenteric type. One might suppose that these were not true cases of dysentery were it not for the fact that dysentery bacilli are isolated from the stools.

In all severe cases of dysentery marked impairment of the nutrition occurs, but with suitable dietetic management the degree of malnutrition may be controlled to a considerable extent.

Complications

Infants suffering from dysentery very frequently develop pyelitis. In fact, a certain degree of pyelitis may be considered almost as a symptom of dysentery. The pyelitis is usually due to colon bacillus infection. Another frequent complication is otitis media, which often develops insidiously and may therefore be overlooked. There may be no symptoms referable to the ears even in those cases in which otoscopic examination reveals a red, bulging drum. Only too often the first intimation of the presence of otitis media is a discharge of pus from one or both ears. Pneumonia occurs as a complication, especially in young and undernourished infants. It may be of either the lobar or the lobular type. Prolapse of the rectum occurs in most severe cases.

Prognosis

The mortality from dysentery varies in different epidemics, depending apparently upon the virulence of the infecting organism. Shiga infections are especially virulent. Young infants and those who are undernourished usually have less resistance to the infection than older individuals so that in these the disease is more likely to prove fatal. In the presence of epidemics of dysentery, one may observe in a single family a rapidly fatal course in infants, a prolonged course with ultimate recovery in older children, and merely slight indisposition and mild diarrhea in the case of adult members of the family. The average mortality rate of dysentery in infancy, considering all cases, is in the neighborhood of 35 per cent.

Treatment

The underlying principle in the treatment of dysentery is to maintain the nutrition until the body has developed sufficient resistance to overcome the infection. The diet should be one which is adequate to meet the nutritional requirements, but which is, at the same time, adapted to the limited digestive capacity. It should be clearly understood that the diarrhea of dysentery cannot be entirely checked by dietary means alone, for the diarrhea will continue so long as the infection is active and until the intestinal lesions have healed, irrespective of whether the infant is fed or starved. Any type of food, however, which is indigestible and irritating may keep up the diarrhea.

Because of the limited digestive capacity of infants suffering from dysentery, acid milk in some form is preferable to sweet milk as a basis of the diet. From the theoretical standpoint, milk soured with cultures of B. acidophilus should be preferable to milk soured by the addition of lactic acid; but practically little difference is noted. Fat should not be present in the diet in any large amount because of the fact that fatty acids and soaps appear to be especially irritating to the ulcerated areas in the intestine. The milk fed should, therefore, be partially or completely skimmed. An excess of readily fermentable carbohydrate should be avoided because of the irritating action on the lower bowel.

A suitable feeding for patients with dysentery is one which has for its basis acidified skimmed milk to which has been added dextrin or a dextrin-maltose sugar; dextrin itself is preferable.

A suitable proportion of carbohydrate to add is one ounce to each fifteen ounces of skimmed lactic acid milk. Such a formula should be fed at reasonable intervals and the infant allowed to take all he desires. Anorexia may be so extreme that but little food is taken, and in such cases the food should be gavaged. The total amount given at a feeding should be but slightly less than that ordinarily taken by an infant of the age under normal conditions. Starvation or prolonged underfeeding must be avoided. A continuation of the diarrhea is no contraindication to feeding; but if the feeding appears definitely to increase the diarrhea, the character of the feeding should be somewhat modified, preferably by decreasing the amount of sugar present. In the case of infants over six months of age, the milk and sugar mixture may be supplemented by the addition of well-cooked cereal gruels prepared from barley flour, cornstarch, or arrowroot. The rougher whole grain cereals, such as oatmeal should not be used. Many older infants refuse to take any form of acid milk, and for these boiled skimmed sweet milk may be substituted. Vegetables, fruit, meat and other supplements to the diet should be omitted until convalescence is established. In prolonged cases, however, it is advisable to add strained orange juice and small amounts of cod liver oil to the daily feeding. As the temperature falls and the general symptoms of dysentery disappear, a normal diet for the age may gradually be resumed.

A diet which has had considerable vogue in the treatment of dysentery has been one containing very little protein and considerable lactose. The theoretical basis of this diet is the observed fact that dysentery bacilli do not readily ferment lactose, and produce but little toxin when grown in media low in protein. As a matter of fact, lactose, when administered in the food, is split largely into the fermentable dextrose and galactose before reaching the lower portion of the intestinal tract, and furthermore the dysentery bacilli have invaded the mucosa and are capable of producing toxins there at the expense of body protein. In actual practice the lactose diet has not proved to be an effective one.

As in all cases of diarrhea the maintenance of the water balance of the body is essential, therefore, abundant water should be given. Many dysentery patients persistently refuse to take water voluntarily, so that it becomes necessary to administer fluid by gavage, by nasal drip or parenterally in the form of dextrose or

saline solution. The technic of administration is the same as in other forms of diarrhea. Blood transfusions are of great value and are indicated in all cases in which the course of the disease is prolonged beyond a week or ten days.

Opium is of value in relieving pain and tenesmus and in slowing down intestinal peristalsis. The disadvantage in the use of opium is that it tends to increase abdominal distention. Opium may be given in the form of paregoric, beginning with an initial dose of 5 to 10 minims. Colloidal silver salts, as recommended under the treatment of diarrhea, sometimes appear to be of value. Bismuth is practically useless. Irrigation of the colon with dilute silver nitrate solution has been used with the idea of promoting healing of the ulcers. These irrigations are extremely painful, accomplish but little good, and are not to be recommended. Irrigation of the colon with plain water or normal saline serves to remove mucus and at times appears to relieve tenesmus. It is well to follow the irrigation with a retention enema of starch paste containing from 3 to 5 minims of laudanum. The use of cathartics is contra-indicated in all cases of dysentery.

The administration of antidysenteric sera would appear to be a logical method of treatment. If any effect is to be obtained, however, the serum should be one which is specific for the particular infecting organism. Polyvalent sera have been prepared by immunization of horses with cultures of the various types of dysentery bacilli. The results from the use of such sera have, in general, been disappointing; although in some reported epidemics of Shiga bacillus infection, good results have been obtained when the specific serum was administered very early in the course of the disease. If serum is to be used at all, it should be given early in the disease and in doses of from 10 to 20 c.c. subcutaneously once or twice daily. It has been claimed that serum given by rectum is sometimes effective. We have had no experience with this method of administration.

The value of vaccines in the treatment of dysentery has not been demonstrated.

CHAPTER XXII

CELIAC DISEASE

(Chronic Intestinal Indigestion, Intestinal Infantilism)

Quite different from the usual types of diarrhea which have been described is a condition of chronic gastrointestinal disturbance known as celiac disease. This condition was first clearly differentiated by Gee, of St. Bartholomew's Hospital in 1888. His original description was as follows:

"There is a kind of chronic indigestion which is met with in persons of all ages, yet is especially apt to affect children between one and five years old. Signs of the disease are yielded by the feces; being loose, not formed, but not watery; more bulky than the food taken would seem to account for; pale in color, as if devoid of bile; yeasty, frothy, an appearance probably due to fermentation; stinking, stench often very great, the food having undergone putrefaction rather than concoction. The pale loose stool looks very much like oatmeal porridge or gruel. The hue is somewhile more yellow, otherwhile more drab.

"The patient wastes more in the limbs than in the face, which often remains plump until death is nigh. In the limbs, emaciation is at first more apparent to hand than to eye, the flesh feeling soft and flabby.

"To *diarrhea alba* add emaciation and cachexia, and we have a complete picture of the disease."

This description covers the essential features of the disease. Typical cases of celiac disease are not common. They appear to be somewhat more numerous in the southern states than elsewhere in the United States.

Etiology and Pathology

Celiac disease is not seen in breast-fed infants and is rarely seen during the first year of life. In some cases there is a history of improper feeding, especially with large amounts of fat or with raw milk. Occasionally the disease follows an attack of diarrhea

resembling dysentery. Herter believed the condition to be due to a persistence of an infantile type of flora in the intestinal tract, but the evidence in support of this view is not convincing. Because of the fact that the stools are usually light colored, deficiency in bile secretion has by some been considered the chief etiologic factor. The liver, it is true, is usually smaller than normal, but microscopically shows no changes. In one case under our observation, the gall bladder failed to throw a shadow on cholecystography. Pancreatic insufficiency has been suspected, and in a few reported instances interstitial changes have been found in the pancreas. A condition closely resembling celiac disease has been described by Bramwell, in which there is definite pancreatic insufficiency; this condition differs from celiac disease, however, in that neutral fats are not split to soaps in the intestinal tract.

A very constant finding in celiac disease is gastric achlorhydria, and this may in part explain the symptomatology.

Symptomatology

Celiac disease develops some time after the infant has been weaned, usually during the second year of life. The onset may be insidious. The infant ceases to gain in weight, becomes irritable, the abdomen distends, and the stools gradually assume the features characteristic of the disease. In other instances the onset is dated from an attack of diarrhea or dysentery from which the infant never completely recovers. When the condition is fully developed, the stools are very bulky, light colored, greasy in appearance, frothy, and extremely foul smelling. They consist chiefly of fatty acids and soaps with small amounts of neutral fat. Undigested food particles may be seen. Blood or pus is not present. The light color of the stools is not due to absence of bile pigment, as this may be detected chemically.

The number of stools usually varies from four to eight daily, but during acute exacerbations of the condition, the stools may become much more numerous. Alternating with the severe diarrhea are periods in which the stools have a fairly firm, putty-like consistency. They are, however, still very bulky, foul smelling and always contain an excess of lime soaps.

Unless the condition is brought under control, the infant wastes to a marked degree. Subcutaneous fat disappears throughout the

body. The wasted extremities are in striking contrast to the greatly distended abdomen. This gives the infant a peculiar spider-like appearance.

Marked edema is frequently present. This is not due to renal or cardiac disease, but appears to be dependent upon a low concentration of protein in the blood plasma.

There occurs progressive muscular weakness. Infants and young children who may have been walking often become bedridden.

A moderate degree of secondary anemia is usually present. The calcium of the blood is often lower than normal.

Growth of the long bones becomes retarded so that a patient with celiac disease may at the age of five or six years be no taller than a normal child of two. The changes in the bones, however, are not those of rickets.

Tetany is of frequent occurrence. Scurvy is sometimes seen as a result of dietary restrictions. Patients with celiac disease are likely to be irritable and fretful and difficult to handle, but there is no actual mental deficiency.

The condition is a chronic one, lasting for years.

Treatment

The essential factor in the treatment of celiac disease is dietary regulation. These patients have an intolerance to fat of all types. The fat which is fed is saponified but the soaps are not readily absorbed. In some instances, as much as 70 per cent of the total fat intake is lost by way of the stools. The feeding of fat is likely to cause an exacerbation of all of the symptoms. Carbohydrates are more readily utilized than fats, but the administration of cane sugar or milk sugar is likely to result in marked intestinal fermentation and increase in the diarrhea. Dextrin, maltose and dextrose can be taken in reasonable amounts. The tolerance for dextrose is higher than that for other sugars. Starch may be taken in moderate amounts by older infants without increasing the gastrointestinal disturbance. Protein is the one form of food which appears to be well digested and absorbed. Patients with celiac disease are incapable of digesting very much sweet milk, probably because of its high buffer value and the fact that there is usually a marked deficiency in the hydrochloric acid of the gastric juice. Because of the irritable condition of the intestinal tract,

any type of rough food is not well tolerated. These patients cannot take whole grain cereals or fibrous vegetables without suffering from gastrointestinal irritation.

It is thus seen that the diet must consist largely of protein supplemented with moderate amounts of dextrin, maltose, dextrose or starch and that its buffer value should be low. Skimmed lactic acid milk to which has been added casein or the curds of skimmed milk forms a suitable basis for the diet. In beginning treatment the child may receive one to two pints of skimmed lactic acid milk to which have been added one to three ounces of dried casein or the curds from one or two quarts of skimmed milk. Such a diet contains almost no fat, very little lactose and considerable protein. It has a low buffer value and is usually well digested. It supplies necessary mineral salts and some of the vitamins. The diet, however, is not a complete one, nor one upon which an infant may be expected to thrive. It is suitable for a period of a week or two at the beginning of treatment. As the character of the stools improves, additions should be made to the diet. Cod liver oil in a total amount of one to two teaspoonfuls a day should be given in divided doses or incorporated in the milk mixture. This may be accomplished by grinding the oil with the casein or curds before mixing with the milk. Strained orange juice in an amount of at least two ounces is given daily; this may be mixed with the milk.

The next addition to the diet should consist of dextrose, which may be added to the milk mixture, beginning with one-half ounce in the total day's feeding and increasing gradually up to two ounces. If this is well tolerated, additional dextrose may be given in the form of 10 or 15 per cent solution flavored with orange juice. This is given between feedings. It has been shown by Nelson* that dextrose given in this way is well absorbed and rarely leads to an exacerbation of the diarrhea. Dextrose solution may be given at hourly intervals and as much as 6 or 8 ounces of dextrose administered daily in this manner. No further additions to the diet should be made for a number of weeks. If the child is then doing well, scraped rare beef in an amount of one to two tablespoonfuls or the white of one or two eggs is given daily. The diet may also to advantage be supplemented with well ripened bananas. For some unexplained reason, bananas are well tolerated by patients with celiac disease. It has even been claimed that

*Nelson, M. V.: Am. J. Dis. Child., xxxix: 76, 1930.

bananas exert an actual beneficial effect. The bananas fed should be well ripened to such a degree that the pulp is a light brown color. From one to four bananas a day may often be taken even by young children with celiac disease and without untoward effect and with apparent benefit.

The next addition to the diet should be thoroughly cooked, sieved green vegetables: spinach, peas, string beans, tomatoes and carrots. From four to six ounces of the vegetable purées may be given. No further change should be made in the diet for a number of months, when fats may be cautiously added; first by substituting whole egg for the white of egg and then by a gradual substitution of whole milk for skimmed milk. No butter, however, should be allowed.

If all goes well, the next addition to the diet should be starches in the form of small amounts of well-cooked farina, cornstarch, or barley gruel and a little dry toast and subsequently well baked potato. The diet with these additions is sufficient to meet all of the nutritional requirements, and this type of diet should be continued for a period of years.

In the treatment of celiac disease it is essential that the child's parents should realize that quick results cannot be expected and that any attempt to advance too rapidly in increasing the variety of foods may result in serious relapse and a loss of all the ground gained during a period of weeks or months. It may be years before the child can take an ordinary diet, and even then any excess of fat or sweets must be avoided.

The general plan outlined above for the dietetic treatment of celiac disease is one which has proved satisfactory in practice. Certain variations in the details of the plan may be made so long as the general underlying principles are followed. An excellent comprehensive plan for the treatment of celiac disease has been outlined by L. W. Sauer.* According to Sauer's plan, dried protein milk constitutes the basis of the diet, the character of the feeding being divided into three phases, as follows:

Phase 1: Dried protein milk is the exclusive diet. One packed level tablespoonful for each 1.5 pounds of body weight. This is mixed with from 1.5 to 2 pints of warm water and sweetened with saccharin (¼ to 1 grain). It is given in four feedings a day with a long interval at night. The total amount of protein milk powder

*Sauer, L. W.: Am. J. Dis. Child., xxix: 155, 1925.

in the day's feedings is increased by about two tablespoonfuls every five to seven days until the number of tablespoonfuls approximates the child's best previous weight in pounds. The first phase is adhered to until the stools, distention and appetite show distinct improvement. It should never be less than two weeks and is usually three or four weeks, occasionally more.

Phase 2: The protein milk feeding is continued but is supplemented with the curds from buttermilk, scraped rare beef, tongue and egg. Cod liver oil and orange juice are added to the diet. Toward the end of this phase, powdered skimmed milk may be substituted for a part of the powdered protein milk. This phase continues until the weight approximates the theoretical weight for the height.

Phase 3: During the third phase, carbohydrates, at first dextrinized flour, arrowroot crackers and toasted white bread are added. Dextrose and Karo syrup are added and well-cooked vegetables in slowly increasing amounts. If any exacerbation of the diarrhea occurs, a temporary return to Phase 1 is made.

The three-phase régime of Sauer does not include the giving of bananas. These may, however, to advantage be added during the second and third phases.

Although the amount of fat in protein milk is usually tolerated by patients with celiac disease without increasing the diarrhea, much of the fat is lost in the stools in the form of soaps, so that it serves but little purpose in the nutrition. Other patients fail to do well so long as even this amount of fat is present.

In the treatment of any case of celiac disease, it is essential that proper attention be paid to the general hygienic surroundings of the child. Fresh air, sunlight, proper clothing and sufficient rest all tend to promote recovery. These children are difficult to handle, fretful and querulous. Whoever cares for them should be even-tempered, considerate and possessed of infinite patience. Harsh treatment may cause such a patient to lose the appetite for days. On the other hand, these children must not be spoiled by excessive indulgence. The diet must be persisted in and no variations made merely for the purpose of satisfying the child.

With proper treatment patients with celiac disease may reach approximately normal physical and mental development.

CHAPTER XXIII

VOMITING IN INFANCY

The most frequent causes of vomiting in infancy are:

(1) Overdistention of the stomach by swallowed air
(2) Too frequent feeding
(3) Too large volumes of food
(4) Unsuitable composition of the food
(5) Parenteral infections
(6) Habit or "nervous" vomiting (rumination)
(7) Gastroenterospasm
(8) Pyloric stenosis
(9) Miscellaneous abdominal conditions (appendicitis, intestinal obstruction, etc.)
(10) Anhydremia
(11) Intracranial conditions
(12) Toxic states

Vomiting Due to Swallowing of Air

All young infants swallow a certain amount of air during and between feedings. Roentgenograms almost invariably reveal an air bubble in the stomach. Infants who are underfed and are consequently always hungry are especially likely to swallow large amounts of air. More air is swallowed when an infant takes a feeding from a bottle while lying on the back than when he nurses the breast or is fed in an upright position. When an infant having a large bubble of air in the stomach is fed, he is likely to take a volume of food which, together with the air already present, is in excess of the gastric capacity so that something must escape. If the infant is lying on the back the bubble of air will accumulate anteriorly in the stomach above the level of the cardiac orifice so that milk will be expelled until the distention of the stomach is relieved. If, on the other hand, the infant is held in an upright position, the air bubble will rise to the cardiac end of the stomach, and be belched up. In order to prevent the vomiting occurring as the result of swallowed air, the infant should be held over the shoulder just before and just after each feeding and patted on the back until belching occurs. It may be necessary to interrupt the

feeding in order to get rid of the air. In the case of some infants who swallow large amounts of air between feedings, it is necessary to keep the infant constantly propped up in bed in a semi-upright position. This is readily accomplished by resting the infant's back against pillows and holding him in position by means of a small harness made from tapes fastened to his body and to the sides of the crib. This does not usually interfere with the infant's sleep.

Some infants develop the habit of finger sucking, and in this way, swallow a great amount of air. This may be prevented by encasing the elbows in lightweight cardboard splints, by pinning the sleeves to the bed clothes or diaper or by covering the hands with aluminum ball mits.

Overdistention of the Stomach by Too Frequent Feedings or Too Large Volumes

There is considerable variation in the emptying times of infants' stomachs. This depends upon constitutional factors as well as upon the character of the food. When feedings are given at such frequent intervals that one feeding has not passed out of the stomach before the next is given, overdistention is likely to occur with consequent spitting up or vomiting. Vomiting is much more frequent in the case of infants fed at two- or three-hour intervals than in those fed at four-hour intervals.

The giving of too large volumes at a feeding results in overdistention of the stomach, especially when the feedings are taken very rapidly. Vomiting from this cause is seen especially in artificially fed infants receiving very dilute milk formulas, for in such instances the intake of large volumes is necessary in order to meet the nutritional demands and to satisfy the infant's hunger. Vomiting from this cause may be remedied by the administration of a more concentrated food. One of the most frequent mistakes in infant feeding is to increase the dilution of the food in the case of infants who are vomiting when they are already taking too large volumes of a food which is too dilute.

Vomiting Due to Unsuitable Composition of the Food

Feedings containing a large proportion of fat leave the stomach slowly, so that complete emptying may not occur before the next feeding is given. This predisposes to vomiting. Vomiting from this cause is not usually seen except when top milk or cream mix-

tures are fed, as the amounts of fat in whole milk are not sufficient to delay greatly the emptying of the stomach.

It has often been stated that carbohydrates of the dextrin maltose type are more likely to cause vomiting than other sugars. We have been unable to confirm this.

Some infants with more than the average amount of gastric acid secretion vomit when given acid milk but are able to retain sweet milk. The feeding of raw milk or milk which has been heated to only a moderate degree results in the formation of very large curds in the stomach, which pass the pylorus with difficulty. Excessive curd formation is one of the causes of vomiting. This is readily remedied by using boiled, evaporated, dried or acid milk. Spoiled food or unusual articles of diet, such as fruits, candies and pickles, may of course cause vomiting.

Vomiting Due to Parenteral Infections

In the presence of any acute infection, vomiting may occur. Infants suffering from otitis media and pyelitis are especially likely to vomit. In the case of a beginning otitis media, vomiting may be noted before any rise of temperature has occurred and before any local evidences of infection in the ear are detected. Infants with chronic pyelitis may vomit at frequent intervals for months at a time. Almost any other infection in the body may at times lead to vomiting.

If an infant has been taking reasonable amounts of a well-balanced formula at proper intervals and then suddenly begins to vomit, the first step should be to look for evidences of infection rather than to assume that the feeding is at fault. Certain measures should, however, be taken to control the vomiting while the infection is being treated. Whatever formula is being given may be thickened by boiling with a sufficient amount of barley or rice flour. (See page 235.) The volumes of food given should be small; hence concentrated formulas are preferable to dilute ones. The administration of atropine is also at times of service except when much fever is present. (See Chapter XXV.)

Habit or "Nervous" Vomiting, Rumination

Some infants vomit readily on the slightest provocation. This is especially likely to occur in infants of the nervous, fretful type and in those who are continually handled and played with. Other

infants develop the habit of bringing up food voluntarily. The food may be merely brought up into the mouth, held for a while and swallowed, or may be completely expelled. This habit is known as "rumination." There appears to be no organic basis for the condition. Some infants appear to ruminate just to amuse themselves, others to attract attention. The infant usually goes through a series of grimaces and contortions just preceding the vomiting; he frowns, smiles, works his jaws backward and forward, stiffens the body, arches the neck and expels the food. Occasionally vomiting is initiated by putting the hand in the mouth. Rumination is a habit which may continue throughout infancy and well into childhood and one which may result in a severe degree of undernutrition. It is important that rumination should not be confused with other forms of vomiting.

There are various means of treating rumination, and no one means is effective for every infant, and with some all methods of treatment may fail. One of the most effective means of treatment consists in thickening all feedings given by boiling with cereals. The milk formulas may be the same as in the case of a normal infant except that 10 to 15 per cent of barley flour is added and the whole heated for an hour or longer in a double boiler. Such a mixture should be so thick that it will not fall from an inverted spoon. Any vegetables or other articles of diet are incorporated in the thick feeding. These thick mixtures cannot, of course, be fed from an ordinary nipple or from a bottle, but must be given with a spoon or, in the case of young infants, from a nipple of the type used on wide-mouthed feeding bottles. (Hygeia nipples.) The tip of the nipple is cut off so as to leave a hole about the diameter of a lead pencil. The body of the nipple is filled with the thickened mixture, and as the baby sucks the food is forced down into the tip with a glass or wooden rod. These thick mixtures are vomited with difficulty, and some infants, finding themselves unable to vomit the food, after a period of time give up the attempt. The thick mixtures may, however, have to be continued for a year or more. The infant may be so accomplished in the art of vomiting that even these thick mixtures are promptly returned. It then becomes necessary to use other means of treatment.

The mouth may be kept tightly closed after feeding by the use of a chin strap (a small square of muslin attached by four

tapes to a larger square covering the top of the head to which the tapes are tied tightly). There is a certain danger in the use of this contrivance because if the infant should vomit, he may aspirate a portion of the food.

In the case of infants who initiate the vomiting by placing the hands in the mouth, the arms may be restrained by loose splints or the hands encased in ball-shaped aluminum mitts. Placing the infant on the stomach after feeding is sometimes effective, but unless retained in this position by mechanical means, older infants are likely to roll over on their backs when they are ready to ruminate.

Gastroenterospasm

Vomiting may be one of the symptoms of gastroenterospasm. It is in this particular type of case that the use of atropine is of especial value. (See Chapter XXV.)

Pyloric Stenosis

This condition is discussed in Chapter XXIV.

Intestinal Obstruction and Atresias

Any condition leading to organic or functional intestinal obstruction may result in vomiting. Among such conditions are congenital obstruction or atresia of any part of the intestinal tract, peritonitis, appendicitis, and acute intestinal obstruction.

Congenital atresia of the intestinal tract may occur at any one of a number of places. There may be an atresia of the esophagus just opposite the point of bifurcation of the trachea. Such atresia may be complete or incomplete. When the atresia is complete, the upper portion of the esophagus ends in a blind pouch, and the lower portion often communicates with a bronchus. In cases of congenital atresia of the esophagus, true vomiting does not occur; that is, there is no expulsive effort on the part of the stomach. There is simply difficulty in swallowing, unchanged food pouring out of the mouth. Diagnosis of this type of atresia may best be made by fluoroscopy after administration of barium. The treatment is surgical. Occasionally good results are obtained in the case of partial atresia, but when complete atresia is present the prognosis is hopeless.

Atresia of the duodenum, although of less frequent occurrence than pyloric stenosis, is not altogether rare. The symptoms so

closely resemble those of pyloric stenosis that a fair proportion of cases are mistakenly diagnosed as pyloric stenosis. The lumen of the duodenum may be narrowed for a short distance, or there may be a complete obstruction, several inches of the duodenum being represented merely by a fibrous cord. Obstruction is most likely to occur in the third part of the duodenum. The symptoms of duodenal atresia are usually present immediately after birth, the vomiting being of the same character as that observed in pyloric stenosis. There also occurs distention or hypertrophy of the stomach. Visible peristaltic waves are seen, but no pyloric tumor is palpable, although in some instances the distended duodenum may be palpated following a peristaltic wave and may be mistaken for a pyloric tumor. The vomitus is usually bile-stained. Infants with atresia of the duodenum do not respond to atropine or to thick cereal feeding. A correct diagnosis may often be made by fluoroscopy. The barium meal leaves the stomach, enters the duodenum but is held back partly or completely at some point in the duodenum. At times the diagnosis is not made until the infant reaches the operating table for pyloroplasty, when the pylorus is found to be normal. Often the duodenal atresia is not evident even then, as the obstruction may be behind the stomach and pancreas. A careful search for possible atresia should be made in any case presenting the symptoms of pyloric stenosis in which no pyloric tumor is found at the time of operation. The treatment of duodenal atresia is surgical and consists of anterior or posterior gastrojejunostomy. When this operation is performed early, the results are usually excellent.

Atresia may occur at almost any other point in the intestinal tract and may be due to failure of development, to the presence of bands or to a persistent Meckel's diverticulum. The treatment of all cases of this type, as well as of acute abdominal infections is, of course, surgical.

Intussusception

The most common form of acute intestinal obstruction during infancy is ileocecal intussusception. In this condition, the symptoms are acute and consist of sudden paroxysmal pain, persistent and projectile vomiting of bile-stained material and the passage of bright red blood by rectum. This is followed by complete constipation. The invaginated ileum may usually be felt in the left

iliac fossa as a sausage-shaped tumor. The invagination may be so great that the entire lower bowel is filled and anal protrusion of the small intestine may occur. There is no fever at the onset, but in untreated cases there is always a rising temperature after the second or third day. Infants with this condition are greatly prostrated and show all the symptoms of profound shock. Anhydremia complicates the picture. In some instances the intussusception is spontaneously relieved, and there are reported instances in which it has been relieved by pressure enemas. It is, however, unsafe to adopt expectant treatment in these cases. Operation should be performed without delay, as the mortality is extremely high unless the condition is promptly relieved. Even after successful operation, many of these patients suffer from diarrhea of the alimentary intoxication type. This is associated with invasion of the stomach and duodenum with colon bacilli. The treatment is that outlined in Chapter XX. Buffered acid protein milk feedings are indicated.

Anhydremia

Vomiting is a frequent symptom in cases of severe desiccation of the body, or anhydremia, however brought about. The treatment of vomiting of this type consists of methods designed to restore a normal fluid balance. (See Chapter XXXII.)

Intracranial Conditions

In infants, as well as in older individuals, vomiting is an accompaniment of intracranial conditions, such as meningitis or brain tumor. Because the sutures are not ossified, however, they are easily separated, and consequently the intracranial pressure is never so great as in adults, and projectile vomiting is less frequent and less characteristic.

Toxic States

In any severe toxemia, whether due to infection or to such metabolic disturbances as are seen in the presence of uremia or diabetic coma, vomiting may occur.

Acidosis is usually considered as an important cause of vomiting. As a matter of fact, acidosis in itself rarely causes vomiting. In cases of cyclic vomiting (which are usually seen after the age of infancy), vomiting is to be attributed to a general

metabolic disorder and not to the acetone body acidosis which is occasionally present, for infants with a much more severe degree of acidosis than that usually seen accompanying cyclic vomiting may not vomit at all. The faulty assumption that vomiting is a frequent result of acidosis has led to the indiscriminate administration of sodium bicarbonate to many infants showing the symptom of vomiting. This type of therapy is capable of producing definite harm, inasmuch as vomiting is likely to be complicated by alkalosis because of the loss of acid in the vomited material. The administration of alkali serves only to aggravate the condition and may even be an important contributing factor in bringing about a fatal termination.

CHAPTER XXIV

PYLORIC STENOSIS

(Pylorospasm, Congenital Hypertrophic Stenosis of the Pylorus)

Pyloric stenosis is one of the rarer causes of vomiting in infants when all cases are considered. It is, however, sufficiently frequent to warrant its consideration as a possible cause in any case of vomiting beginning during the early weeks of life, especially when the feeding has been suitable and no evidences of infection are present.

Pyloric stenosis may be of all degrees. There may be merely a tendency to spasm of the pyloric sphincter or definite hypertrophy of the circular muscle fibers of the pylorus and fibrosis with constant and practically complete occlusion of the pyloric orifice. In some cases the pylorospasm is merely part of a general gastroenterospasm.

Symptoms

Although at times referred to as congenital hypertrophic stenosis, the symptoms are not usually marked until after the second or third week of life. Persistent vomiting from the time of birth is more likely to be due to other causes, such as duodenal atresia. Infants with pyloric stenosis vomit even though the feedings are of good composition and given at proper intervals. Pyloric stenosis is as frequent in breast-fed babies as in those artificially fed. The first symptom noted is vomiting, which does not differ in character from that due to any other cause. Vomiting usually occurs shortly after feedings or the taking of water. The vomiting may at first consist merely of spitting up a small amount after feedings. Very soon, however, the vomiting becomes more forcible, the hypertrophied and dilated stomach becoming capable of expelling food in a projectile manner, often to a distance of several feet. The vomitus may be forced through the nose as well as the mouth. Even water is vomited.

As the result of obstruction at the pylorus, the gastric musculature hypertrophies. The normal gastric peristalsis becomes

greatly accentuated until the peristaltic waves may become clearly visible. These waves always pass from the left to right and should not be confused with peristaltic waves in the transverse colon, which pass in the opposite direction. In those cases in which the stomach has become much dilated and ptosed, the waves may be seen passing downward and to the right and may cease in the neighborhood of the umbilicus or below it. The waves may be present constantly, or may be seen only after the infant has taken food or water. They may at times be stimulated by applying cold to the abdomen and pinching the skin of the abdominal wall. Visible peristaltic waves of the stomach are observed in almost all cases of pyloric stenosis of any considerable duration; they are, however, not absolutely pathognomonic of this condition, inasmuch as some gastric peristalsis may also be seen in infants who have been vomiting excessively from other causes.

In a fair proportion of cases the thickened pylorus may be felt. The size of the tumor varies from that of the tip of the little finger to that of a large olive. The position of the pylorus may vary so that the tumor is not always felt in the same place. It is more frequently found just below the edge of the liver in the nipple line, but may be considerably lower, in the neighborhood of the umbilicus. Some idea as to the location of the pylorus may be obtained by noting the point at which the peristaltic waves disappear.

In severe cases of pyloric stenosis in which a large portion of the food taken is vomited, very little absorption of water takes place. Consequently the urine is scanty and highly colored, and the infant becomes dehydrated. When much of the food intake is lost by vomiting, progressive malnutrition occurs until finally the infant becomes markedly athreptic. On the other hand, even though a portion of each feeding may be vomited, enough food and water may pass the pylorus so that the nutrition is not impaired. Infants with pyloric stenosis are usually constipated, but may suffer from a starvation type of diarrhea. When the pylorospasm is a part of general gastroenterospasm, diarrhea is common.

The persistent vomiting results in a great loss of chlorides from the body in the form of hydrochloric acid and to a lesser extent sodium chloride. This depletion of chlorides may result in a severe degree of alkalosis with accompanying symptoms. (For further discussion, see Chapter XXXII.)

In pyloric stenosis there is a tendency to ultimate recovery, provided the infant's nutrition can be maintained. In most cases the usual course of events, as judged from clinical symptoms and roentgenographic evidence, appears to be a progressive narrowing of the pyloric opening during the first month or two after the onset of symptoms; this is followed by a stationary period in which little change occurs. After the third or fourth month the tendency is for the pyloric aperture to become larger so that food passes more readily. The tumor, it is true, may become larger during this time, but with the growth of the stomach the pyloric opening also becomes larger, so that finally food may pass through readily.

Diagnosis

The diagnosis of pyloric stenosis is made on the basis of the symptoms and signs described above. It is not essential for the diagnosis that a tumor should be palpated. Fluoroscopic examination after a barium meal is often of aid in the diagnosis of questionable cases, but is rarely necessary in typical, well-defined cases. Fluoroscopic examination is of especial aid in differentiating between pyloric stenosis and duodenal atresia.

The character of the vomitus may give information of value in the differential diagnosis of pyloric stenosis. In pyloric stenosis the amount of food vomited is large and when the stomach has become dilated may be considerably greater than the amount of food taken at a single feeding, the residues of previous feedings being included. The vomitus is not bile-stained because the constriction of the pylorus prevents the regurgitation of bile into the stomach, whereas in other forms of vomiting, especially those due to obstruction lower in the intestinal tract, bile is often present in the vomited material.

Congenital duodenal atresia may give rise to symptoms which are very similar to those of pyloric stenosis, but there is no palpable pyloric tumor. The vomitus is usually bile-stained, and fluoroscopy after a barium meal reveals passage through the pylorus with blocking at some point in the duodenum.

Treatment

The treatment of pyloric stenosis may be divided into medical and surgical methods. The choice of the method to be used will depend upon the age and nutritional condition of the infant,

whether or not he is breast-fed, and the degree of obstruction present. The choice of treatment will also be influenced by such extraneous factors as financial conditions, and the availability of suitable hospital facilities and surgical and nursing care.

In any case of suspected pyloric stenosis, medical and dietetic means of treatment should first be tried, but should not be continued unless some gain in weight is attained within a reasonable time. It is dangerous to allow an infant to remain at stationary weight or to lose weight while waiting for natural processes of recovery, because once the nutrition is impaired, complicating infections are likely to occur, and the infant may finally reach such a condition that neither medical nor surgical means are effective. Very young infants, especially those who are still breast-fed and who are not retaining sufficient food on which to gain, should not be weaned in order to apply any method of treatment, but should preferably be operated upon promptly. Infants who have been treated with more or less success for three or four months, whether breast-fed or artificially fed, usually do not require operation, as spontaneous improvement of the condition is the rule at this age. The application of medical means of treatment may necessitate hospital care and at times special nursing for long periods of time. Surgical operation requires but a short period of hospitalization and when skillfully performed is accompanied by negligible mortality in the case of infants in reasonably good nutritional condition.

The chief medical methods of treatment consist in the administration of atropine, the feeding of thickened formulas, refeeding and gastric lavage.

Atropine administration is successful in a fair proportion of cases; in others it somewhat diminishes the vomiting but not to a degree sufficient to allow for retention of enough food for adequate nutrition. In still other cases, atropine appears to be entirely ineffective. It has been assumed that in this latter group an hypertrophied inelastic pyloric musculature is present. It is not usually possible, in an individual case to predict whether or not atropine will be successful; it may act well even when palpation reveals a definitely enlarged pylorus. Certain precautions are necessary in the administration of atropine. The solution used should be freshly prepared, as atropine solutions deteriorate on standing. Furthermore, no two atropine solutions, even when

fresh, seem to have exactly the same potency. It is, therefore, necessary to use a fresh solution about every ten days and to determine the proper dosage of each lot by actual trial. A suitable strength of solution is a 1:1000 dilution of atropine sulphate (¼ grain to one-half ounce of water). The initial dose of this solution is one minim, equivalent to approximately 1/1000 grain. The dose is then increased in minim at a time until the physiologic effect is observed, which consists of a diffuse blush of the skin occurring within ten or fifteen minutes following the administration. This dosage should be continued and given before each feeding. The average amount of atropine solution required to produce the physiologic effect in an infant one month of age is two to three minims, but infants vary greatly in their susceptibility to atropine. Some infants flush on one minim and others require as much as seven or eight minims; this latter dosage should not be exceeded. Often the infant seems very drowsy and sleepy for some hours following the first few doses, but this should not prevent further administration. The atropine solution is measured with a minim dropper, added to a teaspoonful of water, and given through an empty nipple fifteen or twenty minutes preceding each nursing or bottle feeding. It may be necessary to keep up the administration of atropine for several months. The administration of atropine at times leads to an increase in body temperature, which may be as high as 103° or 104° F. and which cannot be accounted for by the presence of infection. It is important to bear this fact in mind. The occurrence of "atropine fever" is not necessarily a contraindication to the use of atropine, although it is usually well to diminish the dosage somewhat.

The second method of treatment consists in the administration of food thickened by boiling with cereal. (See p. 235.) It is usually advisable to apply this method only in the case of bottle-fed babies, although it is possible to express breast milk and thicken it. The formula may be of the same type as that given to normal infants of the same age, except that it is thickened. As in the treatment of any other form of vomiting, the volume of each feeding should be small and the intervals long, preferably every four hours. This necessitates the use of fairly concentrated formulas, such as undiluted whole lactic acid milk with 9 or 10 per cent of Karo syrup or one-half diluted evaporated milk with the same amount of carbohydrate; or one may use a dried milk

formula made up to have approximately the concentration of whole milk. All of these formulas, especially that made with evaporated milk, lead to the formation of very small curds in the stomach, which may readily pass the pylorus. Most babies retain properly prepared thick formulas better than the ordinary liquid types of feeding, but occasionally an infant seems to vomit the thickened feedings more readily.

In the use of such concentrated formulas thickened with starch, there is some danger that the infant will receive an inadequate amount of fluid. Water should not be given by mouth very soon after the feedings, but a moderate amount of water may be given one-half or one hour before a feeding is due. If the water is vomited within a short time, more water may be given and is then likely to be retained. Often water will be retained if given in moderate amounts at night, when no feeding is given. The administration of water, even though vomited, has a certain advantage in that it is a simple method of gastric lavage and accomplishes the purpose of removing mucus and irritating food residues. Should any considerable degree of desiccation of the body occur, parenteral administration of fluid is required. The injection of a normal salt solution provides not only water but also chlorides to compensate for the loss by way of the vomited gastric juice.

Refeeding, immediately after vomiting has occurred, is a valuable means of introducing sufficient food to provide for the infant's nutrition. Refeeding should be attempted only when the vomiting occurs within one hour after the time of feeding. The volume of food vomited is roughly estimated, and this amount of the same formula given immediately after the vomiting. In such cases the feeding is often retained. In this way it may be possible, even though the infant vomits after each feeding, to introduce and have retained in the course of a day a normal amount of food. In breast-fed infants the refeeding may consist of a formula, or if the mother has an abundant supply of milk, expressed breast milk. In general, however, refeeding is not satisfactory in the case of breast-fed infants.

Gastric lavage with a one per cent solution of sodium bicarbonate just previous to feeding serves to remove accumulated mucus and to allay gastric irritability. Following the lavage, the

feeding may be given through the same tube. In some infants, tube feeding appears to stimulate peristalsis less than feeding by mouth.

A combination of all of the methods mentioned may be used. An infant may be given atropine before each feeding, the feedings may be concentrated and thickened with starch, and refeeding and lavage may be done.

The methods of treatment outlined above are more likely to be successful when instituted early than they are after the infant's nutrition has suffered and the stomach has become dilated and hypertrophied. It is claimed that as many as 80 per cent of cases of pyloric stenosis may be treated by medical and dietetic means without recourse to surgical operation, but in a fair proportion of cases so treated, the nutrition of the infant suffers even though ultimate recovery may occur. It is not wise to delay operation in the case of any infant who fails to show a fair gain in weight within a reasonable time.

Surgical Treatment

A number of surgical procedures have been used for the treatment of pyloric stenosis. The most satisfactory is the simple pyloroplasty originally used by Fredet and later developed by Rammstedt. In the case of infants in good nutritional condition, the operative mortality is practically nil and prompt disappearance of symptoms follows. The results of operation, however, are disappointing unless proper attention is given to the preoperative and postoperative care of the patient. In patients who have been badly fed or in whom the condition has lasted for a long time until the nutrition is poor, the stomach greatly distended and the water balance disturbed, operation cannot be expected to accomplish the impossible. The procedures which have been adopted in the St. Louis Children's Hospital in the case of infants operated upon for pyloric stenosis have been outlined by Clopton and Hartmann,* as follows:

"Most of these cases are brought to the hospital markedly dehydrated and at times showing alkalosis. To overcome these conditions abundant subcutaneous injections of Ringer's solution are necessary. If athrepsia is present, glucose solution intravenously or whole blood transfusion is indicated. We regard the

*Clopton, M. B., and Hartmann, A. F.: Surg. Gynec. & Obstet., xlvii: 527, 1928.

preparation of the patient for operation as one of the most important factors in the handling of these cases. Frequently 18 to 24 hours are given to the replacing of fluid loss and to the counteracting of starvation symptoms before we dare operate. Hence, the big part of the battle is fought before we enter the operating room.

"In general, the factors which tend to increase the operative risk are: (1) a disturbance of the acid-base equilibrium of the body, (2) anhydremia, (3) marked asthenia due principally to malnutrition and anemia, and (4) the presence of infection.

"Marked vomiting, when due to pyloric stenosis, causes loss in the vomitus of hydrochloric acid, usually in large amount, and of base chloride in smaller amount. The loss from the body of the chloride ion is almost invariably compensated for in a large part by retention of the bicarbonate ion in the blood and tissue fluids, leading to alkalosis of varying severity. Such a shift toward the alkaline side is just as serious as a commensurate shift to the acid side (acidosis), if not more so. Death may occur promptly, as a result of collapse, cessation of respiration, or generalized convulsions with laryngeal spasm. Aside from loss of acid by vomiting, alkalosis may be increased by any measure which would tend to cause exaggerated breathing. Such instances are commonly seen as a result of crying because of hunger, pain, or manipulation. It is important then since all of these factors may be present before, during, or immediately after operation, to restore if possible the acid-base balance of the body to its normal equilibrium, before operation.

"The diagnosis of alkalosis can be made both clinically and chemically. In the first place, alkalosis of some degree almost invariably accompanies marked vomiting due to some type of obstruction of the gastro-intestinal tract. It is, therefore, always expected in cases of pyloric stenosis; especially if breathing is shallow, depressed, and irregular with frequent long apneic pauses. Further evidence of alkalosis might be noted in the appearance of general hypertonicity and such evidences of tetany as carpopedal spasm, positive Chvostek sign, or generalized convulsions. The urine characteristically is free from chloride (when acidified gives little or no white precipitate after the addition of silver nitrate) but also contains so little base bicarbonate ($BHCO_3$) that it is distinctly acid (pH 5-6) in reaction. This latter point is of importance. Ordinarily, alkaline urine will rule out the pres-

ence of acidosis of any type except that associated with nephritis, but acid urine not only does not rule out alkalosis, when of the type associated with vomiting, but its presence actually lends support to this diagnosis. Certain diagnosis of alkalosis, however, can be made by chemical examination of the blood. Increased P_H (alkalinity) and CO_2 content (base bicarbonate) will be found associated with diminished base chloride.

"Not only does this disturbance of the acid-base balance tend to produce tetany and its consequences, but it also contributes to anhydremia. In addition to the fluid loss of food intake, there occurs the loss of water bound to HCl and BCl. These two factors, i.e., disturbance of the acid-base balance and anhydremia are, therefore, closely associated and the treatment for both is carried out at the same time. The treatment in general consists of the administration of water and salt in the form of Ringer's solution. As a rule Ringer's solution is administered subcutaneously two to three times daily until the chloride content of the blood remains at approximately the normal level. When this occurs the bicarbonate content of the blood will have dropped to approximately the normal level because of the excretion of bicarbonate into the urine, which now renders the urine highly alkaline. At the same time anhydremia disappears. In a few severe cases, showing very marked chloride reduction, it may be necessary to give a more concentrated salt solution, such as 3 per cent sodium chloride, subcutaneously, but this rarely has to be done.

"If at any time acute manifestations of alkalosis are seen, such as tetany or marked depression of the respirations, immediate measures directed toward relief of these acute symptoms will have to be instituted. They are: (1) the breathing of 30 per cent CO_2 in oxygen. This is a quick and effective means of stimulating respiration, of providing sufficient oxygen to saturate the hemoglobin, and to increase the free carbonic acid of the blood so that the ratio $NaHCO_3$:H_2CO_3 becomes more nearly normal due to the increase in the denominator. When this happens the acute signs of tetany are relieved. (2) In addition a 5 per cent calcium chloride solution in an amount equivalent to 0.5 cubic centimeter per kilogram body weight is given intravenously. Calcium given in this way is also immediately effective in relieving tetany. A drug which acts in a similar manner and which is sometimes used is a 10 per cent anhydrous magnesium sulphate solution given in

2 doses each of 1 cubic centimeter per kilogram of body weight, 15 or 20 minutes apart. Magnesium is just as effective as calcium in relieving tetany, but has one distinct disadvantage in that it tends to depress the respirations which, because of the presence of alkalosis, are frequently already depressed. If, however, depression of respirations follows the administration of magnesium sulphate, calcium chloride may then be given as an antidote to magnesium sulphate for this depressant effect.

"Occasionally an infant is so weak because of starvation and anemia that he would be a very poor operative risk. In such instances frequent intravenous injections of from 10 to 20 per cent glucose are given very slowly so as to provoke as little diuresis as possible. Fluid and some immediate food in this way are given. Blood transfusions are also resorted to and in addition an attempt is made to give the infant the usual thick cereal feedings preceded by atropine in physiologic dosage. (See Chapter XXIII.)

"Very frequently otitis media is seen associated with pyloric stenosis. Presumably the high incidence of otitis media in these cases results from the vomiting and the forcible introduction of vomitus into the eustachian tubes. The operative risk in such cases will be lessened if the otitis media is recognized and treated as it would be in other cases. (See Chapter XXVIII.)

"After operation all the measures described as being of importance before operation may have to be continued, if the factors already enumerated still exist. In addition it is important to institute the proper regulation of feeding; i.e., atropine and thickened feedings are discontinued and breast milk or the usual artificial formula (whole lactic acid milk with 3 ounces of Karo syrup to the quart) are used instead. Two hours after the operation ½ ounce of water is given by mouth and 2 hours later ½ ounce of pumped breast milk or formula. Water and milk are then given alternately every 4 hours, increasing by ½ ounce every other feeding. In this way, by the end of the second day the infant will, as a rule, be able to take his full formula or, if breast-fed, may then be put back to the breast.

"The operation is done on an improvised hot-water table and the baby's arms, chest and legs are wrapped in cotton wool or flannels to protect against chilling. The skin of the abdomen is prepared with alcohol.

"The choice of anesthetics is 0.25 per cent novocain in normal salt solution. We use about 60 cubic centimeters over the upper right rectus muscle and in the muscle sheath. The injection is so done as to raise a large tense lump under the skin. (Fig. 19.) An interval of from 10 to 15 minutes after injection permits considerable absorption of the fluid injected. The incision is then made through the edematous tissues, the peritoneum being anesthetized by the forceful injection of fluid beneath the rectus sheath.

"No general anesthetic is used except that in about 1 out of 10 cases a few whiffs of ether are needed when the wound is being closed. The incision, about 2 inches long over the outer border of the rectus, exposes the lower edge of the liver, which is held aside to expose the greater curvature of the stomach. This edge of the stomach is drawn into the wound with rubber-covered forceps and is followed until the pylorus presents. When the olive-shaped, hard, hypertrophied mass is brought into the wound, the index finger of the left hand is hooked over its upper surface and the pylorus drawn well out and held in place by the crooked finger. On the upper anterior face of pylorus, there is a practically avascular area which we select for the incision. The incision is made solely with a blunt dissector. The peritoneum and the hypertrophied muscle are as easily divided with this blunt instrument as normal tissue is with a knife. (Fig. 20.) The blunt instrument is advantageous because it makes injury to the submucosa, which is exposed over the whole area beneath the muscle incision, practically impossible. The divided ends of the muscle are spread apart, the submucosa that has been confined by the constricting bundle pouts out, and the relief of the stenosis is plainly shown. Another advantage of the blunt dissection is that it is almost impossible in this way to divide the muscle of the normal duodenum and stomach. If by any chance this unhypertrophied muscle is cut, bleeding results and a ligature may be required. However, if no damage is done to normal muscle, there is almost never any bleeding.

"The pylorus is now dropped into the peritoneal cavity and the peritoneum of the abdominal wall is closed with fine catgut. A small flat spoon is used as a spatula beneath the line of suture. (Fig. 21.) As the last stitch is about to be tied, we insert a tapered glass tube and through this introduce as much warm salt solution as the peritoneal cavity will hold. When about 100 to 150 cubic

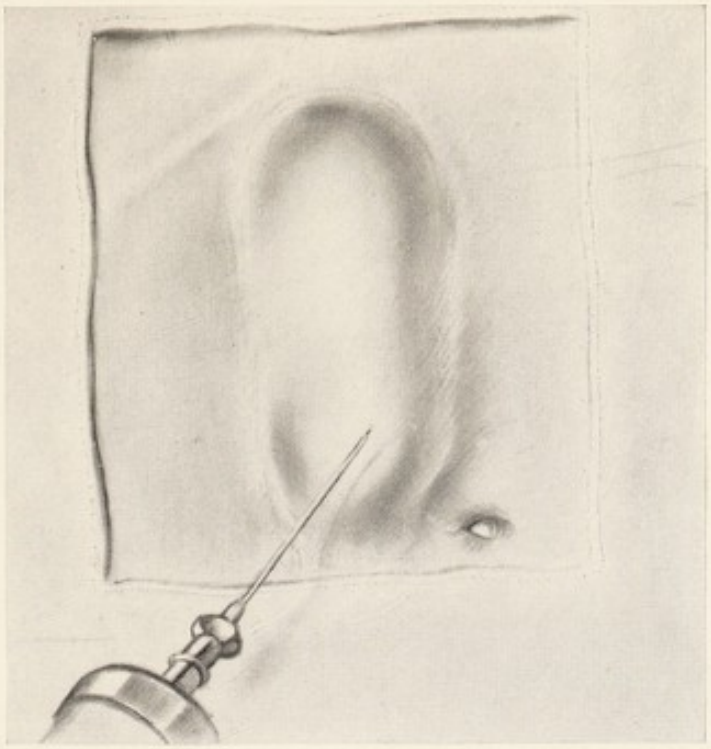


Fig. 19.—Pyloric stenosis operation, first step. Injection of novocaine solution. (Clopton and Hartmann.)

Fig. 20.—Pyloric stenosis operation. Division of the hypertrophied pylorus with the blunt dissector. (Clopton and Hartmann.)

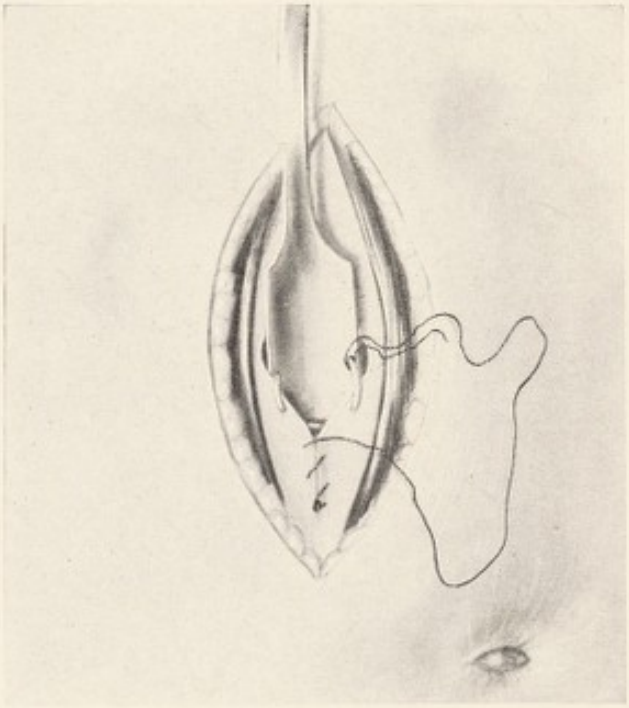


Fig. 21.—Pyloric stenosis operation. Closure of the peritoneum. (Clopton and Hartmann.)

centimeters have been introduced, the solution begins to flow back around the sutures. The tube is then removed and the last stitch tied. The wound is sutured with catgut for muscle and fascia, but we bury 3 to 5 fine silk sutures that include the muscle and fascia. We use these non-absorbable sutures because under any circumstances healing is poor in undernourished infants and we have seen wounds that were sutured with catgut alone break down throughout. The skin is closed with a silk stitch that includes only the epidermal tissues which in about 9 or 10 days is entirely loose and we are spared the task of taking interrupted skin stitches out of a squirming youngster. To complete the operation a small pad of dry gauze is placed over the wound and held in place by means of small adhesive plaster strips and a narrow binder.

"During the operation the baby has a holeless nipple in its mouth as a pacifier and in over half of the cases does not cry at all. In most instances the operation takes only a few minutes and the child leaves the table looking as well if not better than before operation.

"The infant is returned to the ward in its cotton wrappings, and in two hours is given a few sips of water. Two hours later it receives small feedings which are gradually increased as already indicated. In a few days the full-sized feedings are given, and the infant begins to gain weight. In those cases with but little stomach irritability, vomiting ceases after operation, provided one does not increase the size of the feedings too rapidly.

"In long-standing cases in which the stomach has been considerably dilated, there may be a little spitting up for a few days until the stomach regains its proper tone. The baby is kept in the hospital only as long as it is necessary to get the picture of improvement. The infants in better condition go home before the end of a week, but the poorly nourished ones may have to remain two weeks."

Prognosis in Pyloric Stenosis

In cases of pyloric stenosis seen at the onset of the condition there should be practically no mortality. Medical means of treatment are at first instituted, and in favorable cases the results may be excellent. The medical treatment should, however, not be continued if only indifferent success is met with, as this results

in poor nutrition, dilatation of the stomach and often complicating infections. Prompt recourse to surgical treatment in such cases is almost invariably successful, and failures are due usually to some accident, such as puncture of the thin walls of the stomach or duodenum adjacent to the pyloric tumor. Actually the mortality from pyloric stenosis, when all cases are considered, is probably 20 per cent or more. This high mortality, which is preventable, is due chiefly to failure to recognize the condition early, with the result that the infant is weaned and one feeding after another tried with the idea of finding something which the infant can digest. The infant's nutrition suffers in consequence, and he becomes a poor risk for either medical or surgical treatment.

CHAPTER XXV

COLIC, FLATULENCE AND GASTROENTEROSPASM

In its widest application, the term "colic" is used to describe any form of paroxysmal abdominal pain. The pain of infantile colic is due either to overdistention of the stomach and intestines or to forcible peristaltic contractions or to a combination of the two factors.

Overdistention of the intestinal tract may be the result of swallowing of air or excessive fermentation of undigested food, especially carbohydrates. Painful peristaltic contractions may be the result of intestinal distention, of irritation, hunger, or of an instability of the autonomic nervous system.

Intestinal distention is due more frequently to swallowed air than to the gases resulting from fermentation, since the latter, consisting largely of carbon dioxide, are absorbed much more quickly than is air. Air swallowing, in the majority of instances, is the result of underfeeding. The infant who is underfed is always hungry and consequently sucks on everything he can get into his mouth. In this way he swallows much air which distends the stomach and passes through the pylorus into the intestines. Hunger, furthermore, results in a general hypertonicity of the gastrointestinal tract. Forcible hunger contractions of the stomach occur which are transmitted to the intestines. These "hunger pains" are a frequent cause of colic. Various forms of irritation may set up painful peristaltic contractions. The taking of cold food or exposure of the body to cold are frequent factors. Irritating products may be produced in the intestinal tract when diets containing excessive amounts of fat or easily fermentable carbohydrates are fed.

There are certain infants who suffer chronically from flatulence and colic even when given good diets. In these infants there appears to be a constitutional imbalance of the autonomic or vegetative nervous system. The condition has been described in the French and German literature under the name of "neuropathic diathesis." Haas has adopted the term "hypertonic infant" to describe these patients. White has designated the same condition

by the symptomatic term "gastro-enterospasm." Infants of this type are likely to be seen in families in which there is a distinct neurotic taint. The infants are nervous, wakeful and tense; they become pale or flushed on the slightest provocation. Spitting up of food is common, and definite waves of gastric and intestinal peristalsis are frequently seen. The abdomen is always more or less distended. There may be constipation due to spasticity of the anal sphincters or diarrhea due to increased peristalsis. Fluoroscopic examination may show the intestinal contents hurrying rapidly downward to the lower bowel where stasis occurs. Infants with gastroenterospasm are always "colicky" and "gassy." The colic may be accentuated by underfeeding or by the presence of infection.

Colic is more frequently seen during the first three or four months of life than later. It is about as frequent in breast-fed babies as in those who are artificially fed. One of the chief reasons for the frequency of colic during the early months of life is that during this period the diet is being adjusted to the needs of the child. In breast-fed infants the secretion of the mother's milk does not always increase as rapidly as do the demands of the infant. In artificially fed infants the customary use of very dilute formulas during the early months of life is a frequent cause of hunger colic.

There is a widespread belief that overfeeding is an important cause of colic, and there has been much discussion as to whether it is the fat, carbohydrate or protein of the diet which is chiefly responsible. It is possible that an unbalanced diet containing an excess of fat or easily fermentable carbohydrate may lead to occasional abdominal pain. The feeding of too much cream or top milk or of large amounts of readily fermentable carbohydrate may result in irritation of the intestinal tract. It is also possible that large casein curds formed in the stomach when raw cow's milk is fed may bring about some irritation and spasm, but this is probably not a frequent cause of colic.

In the presence of pain anywhere in the body, infants are likely to show symptoms referable to the abdomen; for example, an infant with an acute otitis media may show every evidence of abdominal pain.

The infant with colic screams incessantly. The face becomes suffused, the arms and legs are drawn up and often tremble. The

abdomen is distended and rigid during the paroxysms but relaxes in the intermissions. In simple cases of colic there are no evidences of infection, such as fever or leucocytosis, and no localized findings in the abdomen.

Treatment

In the presence of an acute attack of colic, symptomatic treatment is indicated, but this should never replace treatment directed to the removal of the underlying cause.

During an attack of colic the baby's position should be changed so as to allow accumulated air to escape. The infant should be held upright over the shoulder and patted on the back until belching occurs. If this is ineffectual, a stomach tube may be inserted. Distention of the lower bowel is best relieved through the use of a rectal tube or a high soapsuds enema. A change of position of the baby often brings about the shifting of a large air bubble to a point where fewer symptoms are produced. The infant may be turned from side to side, held upside down, or placed on the abdomen. The application of external heat tends to relieve intestinal spasm. An effective method of treatment of the attack is to place the baby on his stomach over a protected hot water bottle. Carminatives, such as elixir of catnip and fennel (5 to 15 minims in a little warm water), or a soda-mint tablet dissolved in a tablespoonful of warm water are of value. The symptoms of colic may be so severe that the use of sedatives is necessary in order to permit the infant to obtain some rest. Paregoric and chloral are the sedatives most frequently used. Paregoric is especially bad, as its use is often followed by increased abdominal distention. Paregoric does, it is true, often relieve the pain and quiet the infant, but this is likely to lead one into the erroneous belief that the condition has been cured and that further means of treatment directed toward the removal of the underlying cause are not necessary.

In searching for the cause of colic, the character of the feeding should receive first consideration, inasmuch as underfeeding is the most frequent cause of colic. Colic due to underfeeding promptly disappears when a sufficient amount of food is given at proper intervals. It is bad practice to wean an infant who suffers from colic when the condition may be relieved by the giving of complemental or supplemental feedings or by regulation of the

feeding régime. One should not make frequent changes in the formula of artificially fed infants with the idea of decreasing the amount of some supposed offending element. Such a procedure usually results in decreasing the infant's total food intake, and this only aggravates the condition. The treatment of colic due to underfeeding consists in giving more food, not less. Some infants with hypermotility of the gastrointestinal tract suffer from hunger and colic when the feedings are given at four-hour intervals. In these the symptoms may be relieved by feeding more frequently. The intervals should, however, not be shorter than every three hours.

When there are evidences of excessive intestinal fermentation, it is sometimes desirable to substitute an acid milk mixture for a sweet milk one, to reduce the amount of added sugar and to substitute a less fermentable type of sugar. Grulee recommends the administration of extra casein to replace carbohydrate. There are some infants with an excessive secretion of acid in the gastric juice who suffer from colic when acid milk mixtures are fed, and these are benefited by the substitution of sweet milk. In general the feeding of a well-balanced food mixture up to the point of satisfying fully the infant's appetite is a safe procedure and one which is extremely unlikely to produce the symptoms of colic.

The use of atropine is of great value in cases of colic associated with gastroenterospasm. The method of atropine administration is the same as that described in the chapter on Pyloric Stenosis (see page 243). In most cases of gastroenterospasm the administration of a formula thickened with starch is followed by some relief of the symptoms. The method of preparing and feeding the thickened formulas is the same as that described under the treatment of rumination (see page 235).

CHAPTER XXVI

CONSTIPATION

Constipation results when there is an insufficient stimulus to peristalsis or when the food residue is of such a nature as to form firm hard masses which are expelled with difficulty. Constitutional and anatomic factors are of importance. Some infants have intestinal tracts which are active and easily stimulated to peristalsis; others have intestinal tracts which are sluggish. Of two infants of the same age and on exactly the same feedings, one may have regularly four or five loose or semisolid stools a day and the other a single hard constipated movement. Anatomic conditions such as an excessively long sigmoid or variations in the mesenteric attachments with resultant sagging or kinking of the bowel predispose to stagnation.

Since the normal stimulus to peristalsis is food, and especially easily fermentable carbohydrate, an insufficient total amount of food or a relative insufficiency of fermentable carbohydrate may result in constipation. When the food taken is so completely digested and absorbed that little or no residue remains, the bowel movements are of course infrequent, but on the other hand, if the food leaves a firm nonirritating residue, this may accumulate in the lower bowel and be passed with difficulty. A food leaving a reasonable amount of soft residue is likely to lead to normal bowel evacuations. Dextrins and sugars are completely digestible and absorbable and leave no residue; starch, on the other hand, may not be completely digested by a young infant and may leave a soft residue which is readily passed. When excessive fermentation of sugars occurs in the intestinal tract, the products of such fermentation stimulate peristaltic activity. The cellulose residue of cereals consisting of pericarp or husks stimulates peristalsis by mechanical irritation. Although, in general, an excess of carbohydrate in the diet tends to cause diarrhea, one not infrequently sees obstinately constipated infants who are receiving large amounts of such readily absorbable carbohydrates as dextrin or maltose.

Protein in the diet, especially casein, tends to neutralize any laxative effect of sugars due to fermentation. Infants receiving considerable protein and little carbohydrate are, therefore, likely to be constipated.

An excess of fat in the diet may result in constipation, especially when much casein is also present and relatively little sugar. Under such circumstances a residue of lime soaps remains in the intestine. These lime soaps have very little effect in stimulating peristalsis and may form hard, firm masses. Constipated stools often consist largely of lime soaps together with insoluble calcium phosphate which represents the unabsorbed residue of casein. Constipated stools are usually alkaline in reaction because of the presence of soaps and the absence of acid products of carbohydrate fermentation.

For the reasons set forth, constipation is seen most frequently in artificially fed infants, especially in those whose diet is composed largely of milk with relatively little added carbohydrate. Constipation is less frequent in breast-fed infants because of the high proportion of easily fermentable lactose present in human milk, the low proportion of casein and the more complete digestibility of the fat. When constipation occurs in breast-fed infants, it is likely to be due either to an insufficiency of milk or to constitutional factors on the part of the infant. Some breast-fed infants, however, receiving adequate food and gaining steadily are chronically constipated until supplementary solid foods are included in the diet.

Constipation may be the result of local conditions such as fissures in the anal region which lead to painful evacuations. Under such circumstances, the infant may voluntarily suppress the passage of stools.

Habit is an important factor in leading to constipation, especially in older infants. Unless means are taken to encourage regular movements by placing the infant on a vessel at certain times of the day, chronic constipation may result. The retention of fecal material in the lower intestine leads to dilatation and atony of the bowel so that the condition is likely to become progressively worse.

Treatment

Many cases of so-called constipation require no treatment. If an infant is passing only one firm stool a day or even occasionally

misses a day, but is thriving in every respect, the character of the stools may be disregarded. If, however, the movements are passed with difficulty and accompanied by pain, if there are flecks of blood on the stool, or if there are general symptoms, treatment of the constipation becomes necessary. The most important element in the treatment is dietetic regulation. Constipation due to underfeeding is merely a symptom. In such cases an adequate diet should be given not so much to relieve the constipation as to provide for normal nutrition.

In constipation of artificially fed infants, who have been taking relatively large amounts of milk with little carbohydrate, the treatment is to increase the proportion of carbohydrate. An increase in difficultly fermentable carbohydrate does not always result in relief of the constipation; in fact, the extra carbohydrate may be absorbed quickly, taking with it considerable water, so that the stools may become even firmer. When this occurs, a suitable method of treatment is to substitute for a portion of the carbohydrate a more fermentable preparation. The liquid malt extracts are especially suitable for this purpose. From one-half to one and one-half ounces may be included in the total day's feeding and may replace an equivalent amount of other carbohydrates. Instead of adding malt extract, the feeding formula may be allowed to remain the same and the infant be given several teaspoonfuls of liquid malt extract in one to two ounces of water once or twice a day just preceding a regular feeding. The amount given should be regulated by the effects produced.

A special type of formula has been recommended for the treatment of infants who have become constipated and are not thriving on diets containing much cow's milk and little sugar. This is the "malt soup" formula of Keller, which is a one-third dilution of whole milk with added malt extract and flour. In the preparation of Keller's Malt Soup, one and one-half ounces of wheat flour are stirred into one pint of whole milk. This is mixed with a solution containing 3 ounces of liquid malt extract (Malt Soup Extract with potassium carbonate) dissolved in 20 ounces of water. The whole mixture is then boiled for two minutes or heated in a double boiler for twenty minutes. The proportions of milk and of malt extract may, of course, be altered in individual cases. Malt soup prepared in the manner indicated usually has a marked laxative effect. It is contraindicated in the case of infants younger than three

months. It is usually unsafe to feed this mixture during the summer months as it may lead to severe diarrhea. Keller's Malt Soup has no especial advantage over the mixtures with malt extract which have been mentioned above and has the disadvantage of such a low content of milk that the infant's requirements may not be met fully when this mixture is fed. Malt soup is not suitable as a regular diet to be continued for any length of time. There is, however, no serious objection to its use for as long as a week or two.

The constipation of artificially fed infants of the type just discussed may at times be further relieved by removal of a portion of the fat of the milk in order that there may remain a smaller residue of lime soaps in the bowel. It is not usually necessary to resort to this form of diet modification.

One should always bear in mind that the condition of the infant and not that of the stools is the primary consideration. It is all right to alter the diet in order to relieve constipation provided the alteration is not of such type as to make the feeding inadequate for the nutritional requirements.

In the case of breast-fed infants, little or nothing can be done to alter the composition of the breast milk, nor would one know just what alterations would be desirable, even if possible. A simple and effective means, however, of relieving constipation is the giving of liquid malt extract in a little water just preceding one of the nursings.

Fruit juices and cooked fruits have some laxative effect. Orange juice, unless given in large amounts, has relatively little influence in relieving constipation; prune juice is somewhat more effective, probably due in part to its high sugar content. Well-cooked apple sauce may be given in small amounts to older infants with safety, but should not be given to young infants because of the danger of causing too much intestinal irritation. Coarse cereals, such as oatmeal and other whole grain preparations are more laxative than Cream of Wheat, Farina, or barley, and may be given to infants after the age of six months. These should be given unstrained, and small amounts of bran or cellulose rice flakes may to advantage be added.

In all cases of constipation the institution of regular habits is important. By the fourth or fifth month, many infants may be made to pass the movements at regular times of the day. This

is encouraged by gentle massage of the abdomen, following along the direction of the colon, or a baseball may be rolled around the abdomen. This type of treatment is of especial value in infants with lax abdominal walls.

Mechanical means of emptying the bowel only occasionally have to be resorted to. A soap stick, greased paper cone, or suppository may be used to encourage regular habits and to relieve, temporarily, constipation. Glycerin suppositories are irritating and should not be used regularly, although there is no serious objection to occasional use. Enemas should also be used only as an occasional and temporary measure. The development of the enema habit is almost as bad as the cathartic habit.

When the simple measures indicated above are used, there will rarely be need for recourse to drug treatment. The habitual use of cathartics is to be condemned. If other means of treatment fail, the most harmless laxative is probably mineral oil. This may be given as the pure oil or in the form of one of the agar emulsions. No set rule as to dosage can be given as each infant's response is different. It is best to divide the oil into several small doses a day rather than to give one large dose. Mineral oil is not irritating and serves merely to keep the fecal masses soft so that they may be passed readily. When blood-streaked stools indicate the presence of anal fissures, the stools should be kept soft with mineral oil for at least ten to fourteen days until the fissures have healed. Touching the fissures with 5 per cent silver nitrate promotes healing.

Milk of magnesia is partly converted into magnesium chloride in the stomach. Its action is the same as that of magnesium sulphate. It is a hydragogue cathartic, more irritating than mineral oil. Magnesia has the further disadvantage in that it is partly combined with fats to increase the insoluble soap residue of the stools. Milk of magnesia has, however, been widely used for the relief of constipation in infants, and serious ill effects are not common. Neither castor oil nor calomel should be used for the treatment of constipation. In cases of spastic constipation associated with gastroenterospasm the administration of atropine is often an effective means of treatment (see page 243).

CHAPTER XXVII

PREMATURITY

Infants born before the thirty-sixth week of gestation and those weighing less than 5½ pounds (2500 grams) usually differ anatomically and physiologically from normal full-term infants. Infants having a low birth weight are not necessarily premature as the small size may be an inherited characteristic; nor are all infants who have been delivered prematurely necessarily below the average weight of newly born infants. In general, however, those infants who are born prematurely and those who are very small at birth require special care and feeding. In those cases in which prematurity is the result of disease, such as syphilis, further special therapeutic procedures are necessary.

Premature infants lead an essentially vegetative existence. There is very little muscular activity, the reflexes are poorly developed, even the sucking and swallowing reflexes being at times almost absent.

The respiratory center often fails to react to normal stimuli so that the respirations of premature infants may be very irregular and punctuated with long periods of apnea. These periods may be so long at times as to lead to death from suffocation. The apnea of premature infants, however, is sometimes due to intracranial hemorrhage involving the respiratory center.

Premature infants show a marked tendency to thermo-lability, the heat-regulating mechanism not being well developed. The body temperature tends to fall below normal on slight exposure to cold and likewise to rise above normal even in the absence of infection when the temperature of the surroundings is high. Daily variations of body temperature of as much as 5° F. may be observed in the case of premature infants whose surroundings have not been subject to careful regulation.

The digestive capacity of premature infants is low because the normal digestive enzymes are present only in minimal amounts. Intestinal motility is impaired and absorption of food is poor. The gastric capacity of premature infants is likely to be disproportionately small.

The nutritional requirements of the premature infant cannot be estimated on the same basis as those of the average-sized young infant. During the first two or three weeks of life the total caloric requirement is relatively low in proportion to the body weight, probably because of the very slight muscular activity. The caloric requirement during this period varies from 35 to 45 calories per pound (65 to 100 calories per kilo). After the first few weeks the caloric requirement rapidly increases as the infant becomes more active and it then usually exceeds that of the normal infant, and may be as high as 70 or 80 calories per pound (155 to 180 calories per kilo). The premature infant requires relatively large amounts of protein and mineral salts because of the rapid rate of growth. The vitamin requirements of premature infants are also high, especially the requirement for the antirachitic or "D" vitamin. Premature infants are very likely to suffer from rickets, even when given fair amounts of vitamin "D" containing foods, such as cod liver oil. Premature infants have but little immunity, and infections when contracted are likely to lead to serious results.

In the management of premature infants, there are a number of factors which must be given particular consideration. The body temperature must be maintained at a constant level, infections must at all costs be avoided, and respiratory activity must be maintained. The diet must be sufficient to cover the special requirements and yet must be small in volume and easily digestible. Premature infants cannot be raised successfully merely by prescribing a suitable feeding formula. Careful and intelligent nursing is essential.

In order to maintain the body temperature, the premature infant must be clothed properly, kept in a warm environment and not exposed to the chilling effects of injudicious bathing. The clothing of the premature infant should be so designed as to provide against loss of body temperature, but at the same time arranged for ready changing without subjecting the infant to undue chilling. Suitable clothing for the premature infant consists of the customary shirt and diaper and a quilted cotton jacket such as shown in Fig. 22. This jacket is made from cotton batting covered with gauze and is provided with a hood to cover all the baby's head except the face. The jacket should be sufficiently long to extend at least six inches below the infant's feet and wide enough to completely envelop the infant and lap over several

inches. (Fig. 23.) The premature jacket is opened but not removed when the diaper is changed. The diaper should be easily removable and not fastened with too many pins. The premature should not only be protected by suitable clothes, but should also be kept in a warm environment. This may be accomplished either by putting the infant in a small warm room or in one of the forms of heated infant beds or incubators.

The temperature of the warm room should be 85-90° F. for very small premature infants, and 80-85° F. for those weighing over 4 pounds. Fresh air is essential, but drafts are to be avoided. The air of the room should not be excessively dry, a humidity of 55 per cent being about optimum. The proper humidity may be obtained by evaporating water from an open basin on an electric stove or by placing pans of water or moist cloths on a radiator. The degree of humidity may be determined by the use of a humidity indicator such as is sold for use in cigar cases or by the wet and dry bulb thermometer. It is not always possible to provide a suitable warm room which may be maintained at a constant temperature and be properly ventilated nor is such a warm room absolutely essential. The baby may be placed in a heated bed improvised from a clothes basket lined with quilting and kept warm by the use of hot water bottles or well protected electric pads. Considerable skill is required in keeping the temperature just right by these means and there is always the danger of overheating or burning the infant. With careful supervision, however, satisfactory results may be obtained.

Various incubator beds with automatic heat control have been devised. Of these, a very satisfactory and widely used form is the Hess incubator bed (Fig. 24). The temperature of the incubator bed is usually kept at about 85° F.

Premature infants should not be removed from the warm room or from the incubator bed for changing or bathing. In bathing the premature infant, only a portion of the body should be exposed at one time and cotton seed or olive oil instead of water should be used for cleansing purposes. Desirable though it may be to know the weight and progress of gain of a premature infant, it is inadvisable to make daily weighings as this involves considerable exposure. Weighing every three or four days is sufficient, and this should be conducted in a warm room. In weighing the infant a warmed blanket should first be laid on the scales and balanced,

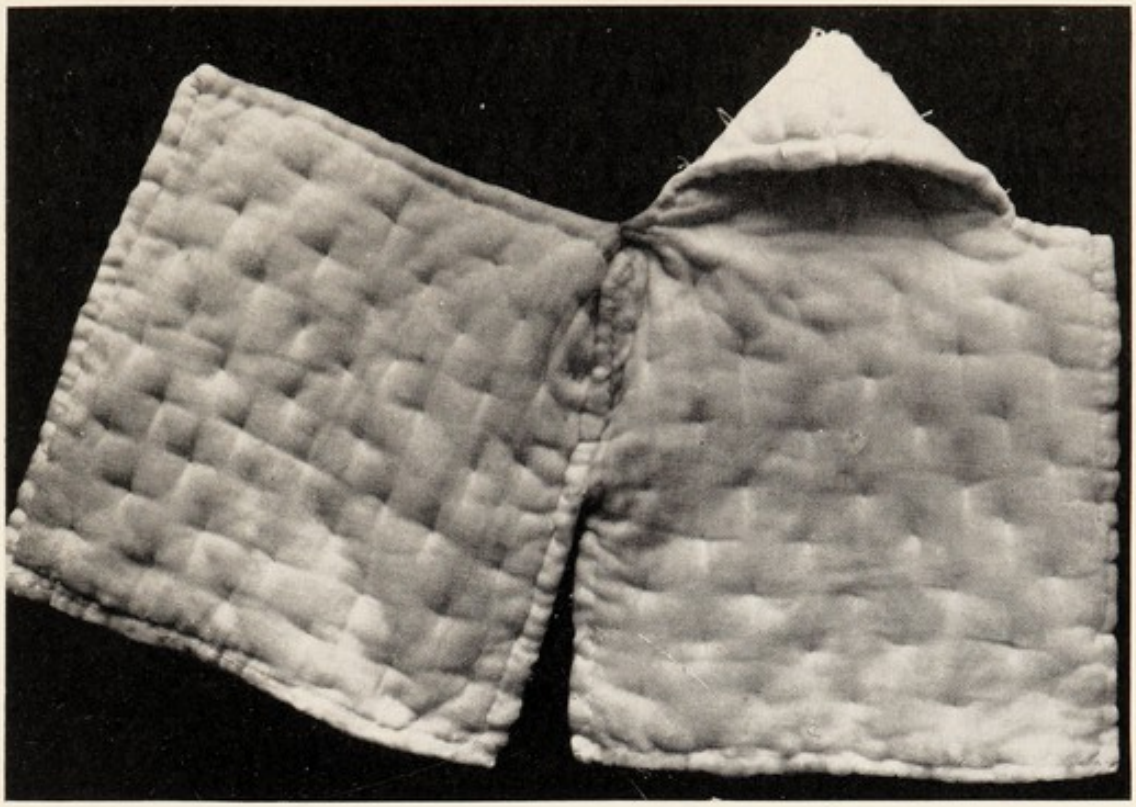


Fig. 22.—Premature jacket of quilted cotton and gauze.

Fig. 23.—Premature jacket applied to infant.

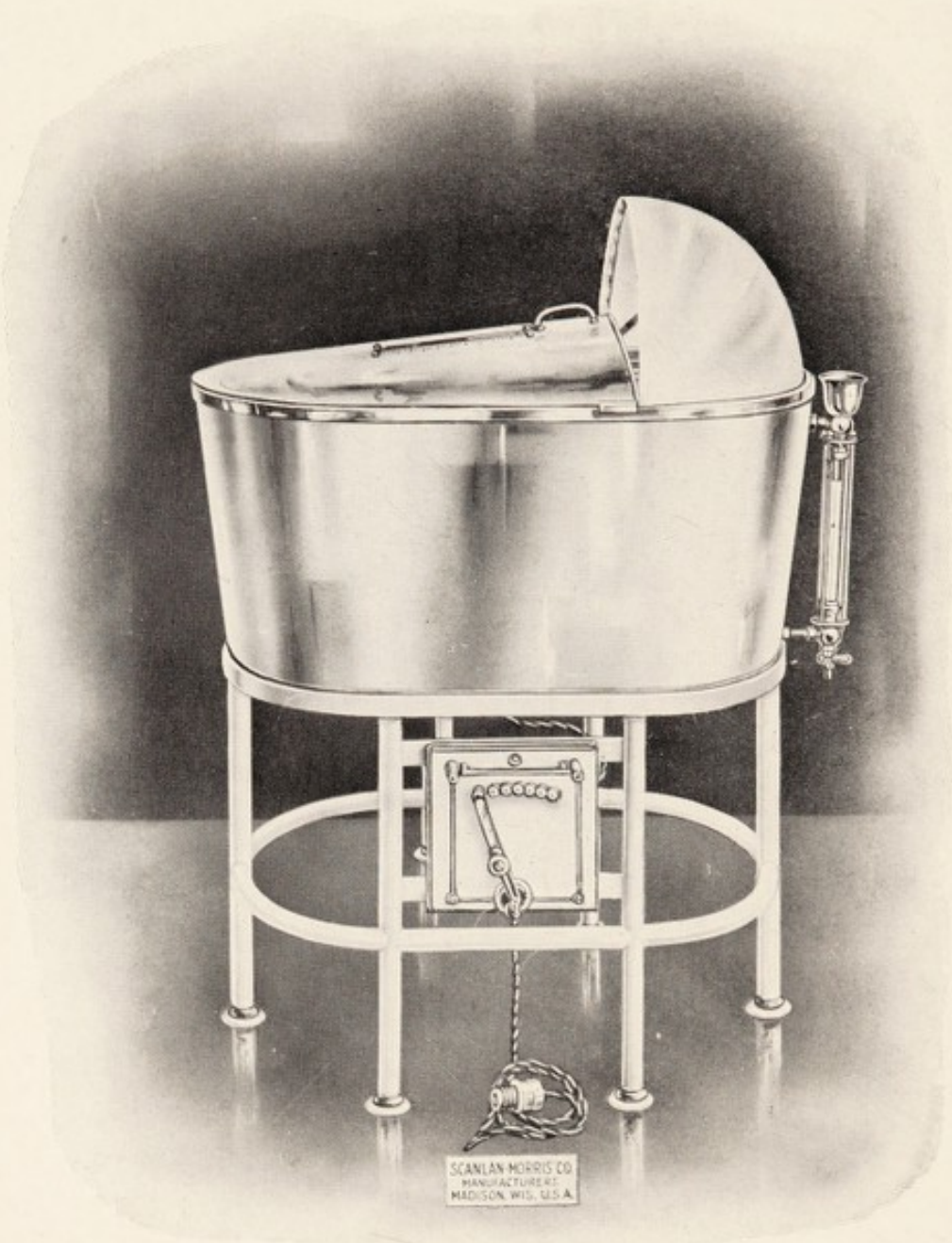


Fig. 24.—Incubator bed of Julius H. Hess. (Scanlon-Morris Co., Madison, Wis., manufacturers.)

the infant should then be quickly undressed, wrapped in the blanket and kept covered while being weighed.

Premature infants are extremely susceptible to infections and even such a simple infection as a cold in the head may have fatal consequences. Whenever possible only one nurse should be in charge of the infant with a single individual to relieve her. As an extra precaution against infection the nurse should wear a face mask when handling the infant. Should the nurse contract a cold in the head or any infection, she should be promptly relieved from duty. No visitors should enter the infant's room.

The respiratory effort of the premature infant is at best poor, and is often insufficient to expand the lungs completely. The respirations are not only shallow, but often irregular. At times it appears that the infant simply "forgets to breathe." During these apneic attacks cyanosis may become extreme and death may occur. The maintenance of normal respiratory activity is difficult as the respiratory center is relatively insensitive. Sensory stimulation of the skin is impractical as these infants must be kept warmly clothed and not subjected to chilling. The most efficient method of bringing about expansion of the lungs is inhalation of a mixture of oxygen and carbon dioxide. These mixtures may be obtained in small cylinders. Either a mixture containing 5 per cent CO_2 or one containing 30 per cent may be used. The 30 per cent mixture has a strong stimulating effect on the respiratory center and should be used only for short periods when the respiratory action has practically ceased. The 5 per cent mixture is suitable for prolonged inhalation after the respirations have been started. The inhalation of these mixtures brings about an increased carbonic acid content of the blood without at the same time diminishing the oxygen content. Inhalations of oxygen alone are also of value in relieving cyanosis. In giving the inhalations a bottle of water with short tube reaching to the bottom should be connected between the tank and the funnel or catheter to be used. The tank value should be so regulated as to allow from 60 to 80 bubbles a minute to pass through the water. (Fig. 25.) The oxygen may be administered to the baby by means of a funnel or small face mask or by a small-sized nasal catheter. The latter method is more effective and economical of oxygen, but is open to the objection that any sudden change in pressure of the gas may damage the infant. The catheter method should not be used un-

less the gas tank is supplied with a high grade valve capable of fine adjustment.

The sprinkling of a little cold water on the infant's face sometimes serves to stimulate respirations, as does the administration of drop doses of aromatic spirits of ammonia. Alpha-lobelin has recently become popular as a respiratory stimulant, but it is a dangerous drug, capable of producing respiratory depression, vomiting and collapse in overdosage. The initial dose (given subcutaneously) should not exceed 1/40 to 1/20 gr. (0.0015 to 0.003 grams) and this should not be repeated within four to six hours.

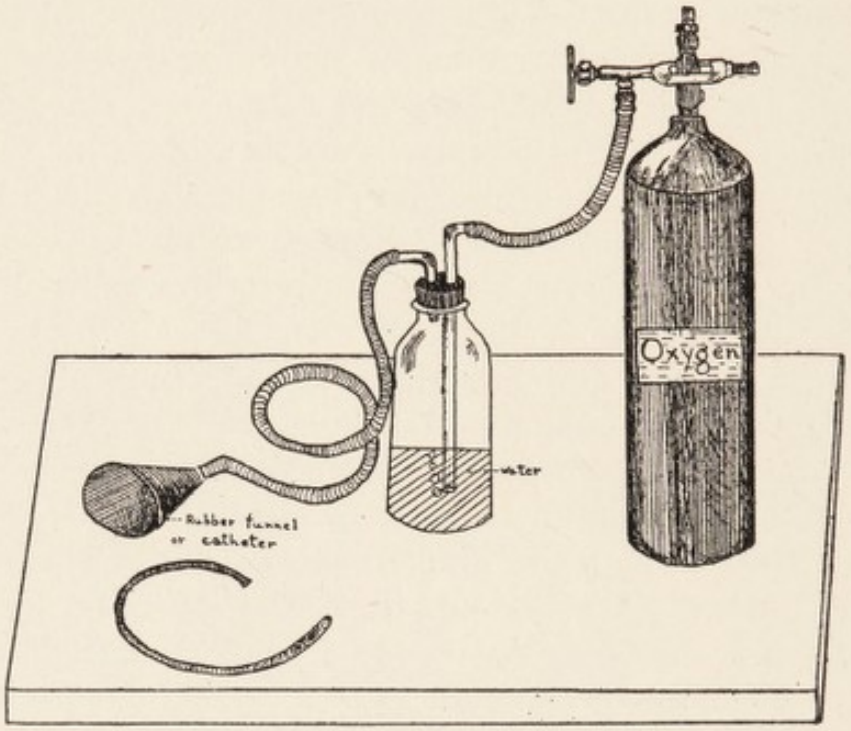


Fig. 25.—Oxygen inhalation apparatus.

The feeding of premature infants presents special difficulties. The digestive capacity is low, the total food requirement fairly high, yet but small volumes can be given at a time because of the small gastric capacity; furthermore the feeding intervals must usually be fairly long because of delayed gastric motility. The food of choice for the premature infant is human milk, but inasmuch as the mother delivered prematurely is likely to have but little secretion and the infant too weak to nurse vigorously, it is generally necessary to express the mother's milk manually or by the use of a breast pump (see Chapter XI). If it is not possible to obtain sufficient milk in this way, the milk of another woman may be used.

Breast milk is readily digested and if the infant is able to take a sufficient volume in the course of a day, his caloric requirements may be met, but this is not always possible, as some premature infants will take no more than a half ounce or an ounce at a feeding and vomit when fed more often than every three or four hours. In these cases the total food intake may be deficient in calories. It may also be deficient in protein because human milk contains very little protein and the needs of the premature infant for protein are greater in proportion to the weight than those of the normal infant. Better results are likely to be obtained if the breast milk is "fortified" by the addition of a digestible preparation of cow's milk and carbohydrate. The caloric value and the protein content of the milk may be increased by the addition of prepared casein or dried partially skimmed milk (Dryco) or powdered lactic acid milk. The caloric value may be increased still further by the addition of difficultly fermentable carbohydrate, such as Karo syrup or dextrimaltose. One may add safely as much as 2 per cent by weight of casein or 5 per cent of dried milk and in addition 5 to 8 per cent of Karo syrup. With these maximum amounts added, the caloric value of the breast milk may be almost doubled without appreciable increase in volume. Such mixtures are usually well tolerated.

If breast milk is not available, a concentrated formula of whole lactic acid milk may be used. Undiluted whole lactic acid milk with the addition of 7 to 10 per cent by volume of Karo syrup is a suitable formula for most premature infants. This formula appears to be more readily tolerated when prepared from evaporated milk. (See Chapter XVI.) The formula described has a caloric value of 25 to 30 calories per ounce, therefore relatively small volumes are needed in order to meet the nutritional requirements. Another type of formula with which we have obtained excellent results is prepared from dried protein milk made up with acid buffered solution. This is the same formula which is used in the treatment of diarrhea. A formula of this type is of especial value because of the fact that premature infants are prone to develop diarrhea as the result of the low gastric acidity which predisposes to colon bacillus infestation of the stomach and duodenum. In preparing the formula a buffer solution is first made up as follows:

BUFFERED BACTERIA ACID SOLUTION

Lactic acid U. S. P.	15 c.c.
Sodium Hydroxide 10%	20 c.c.
Water to	1000 c.c.

One part by weight of dried protein milk is incorporated with 10 parts of buffer solution. To this may be added Karo syrup up to as much as the amount of dried protein milk used. For example, a premature baby taking six feedings of two ounces each would receive the following formula:

Dried protein milk	1 oz. (30 grams)
Buffer solution	10 oz. (300 c.c.)
Karo syrup	1 oz. (30 grams)

The amount of sugar added should not exceed that mentioned and should be diminished in the presence of loose stools. A better nutritional result is, however, to be expected when the full amount of sugar indicated is fed.

Egg yolk should be added to the formulas after the first two weeks, starting with one-half a yolk and increasing to one yolk daily. The raw yolk is shaken into the milk mixture.

Mechanical difficulties are likely to be encountered in the administration of any type of feeding. Most premature infants will not suck well on a nipple and furthermore the usual sized nipple is too large for the mouths of very small infants. Special types of feeders have been devised for premature infants, such as the Breck feeder, but these devices are hard to keep clean and possess no advantages over an ordinary medicine dropper, the tip of which is protected by a short length of narrow rubber tubing. In using the medicine dropper, small amounts of milk are allowed to flow into the infant's mouth at a time. If the infant swallows well, this method is satisfactory, but some infants will hold a portion of the milk in the mouth and allow it to flow out, or else partially aspirate it. Should this occur regularly, resort must be had to tube feeding.

In feeding by tube, a small catheter, not larger than No. 12 French, should be used. This is inserted through the mouth or nose and passed down to a point just above the cardiac orifice of the stomach. It is better not to allow the catheter to enter the stomach, where it may cause irritation. The length of the catheter

to be inserted will, of course, vary with the size of the infant. For a premature infant weighing 4 pounds, the length of catheter to be inserted, measured from the margin of the gums, will be 5 to 6 inches. This point may be marked with indelible ink on the catheter. Milk should be allowed to flow into the stomach slowly by gravity from a glass tube receptacle held not more than 6 to 8 inches above the head. After feeding, the catheter is pinched off before withdrawal so as to prevent dripping of milk into the pharynx.

Tube feeding may be accomplished with very little disturbance to the infant. In fact, some infants sleep through the entire procedure. The volume of food taken by a premature infant is necessarily small. Some may not be able to take more than one-half or one ounce at a feeding because of underdevelopment of the stomach. No set rule may be laid down as to the volume of food which the premature infant of a given weight may be expected to take at a feeding. One should begin with a small amount and increase cautiously in amounts of one dram at a time. The regular occurrence of vomiting after feeding is an indication that too large a volume is being given. Larger amounts are usually taken when the feeding intervals are longer, for gastric motility is sluggish in the premature infant and when too short feeding intervals are used, one feeding may not have left the stomach before the next is given. Because of the small volumes which it is possible to feed at a time, frequent feedings have been advised in order to give sufficient food in the course of a day, but in general the more satisfactory method of introducing more food is to give more concentrated feedings at longer intervals. The four-hourly interval may to advantage be used for the majority of premature infants, and it is rarely necessary or advisable to feed more often than every three hours. In any individual case, the feeding intervals and volume of the individual feedings must be regulated according to the response of the infant.

The symptoms of vomiting and diarrhea occurring in premature infants are to be treated along the same general lines as indicated in the case of normal infants. When vomiting occurs the volume of the individual feedings should be decreased and the feeding intervals, if short, should be lengthened. In order to prevent inadequate food intake, the food should at the same time be made more concentrated. Thick cereal feeding is not so effective in

preventing vomiting in premature infants as in normal infants. The thickened formulas may remain in the stomach for fairly long periods, not being forced through the pylorus because of the weak gastric musculature. It is difficult to feed thickened formulas to premature infants, but such feeding may be given by a tube, using a syringe to force the feeding through the catheter.

Diarrhea is always serious in the case of premature infants and a severe diarrhea is usually fatal. The treatment of diarrhea is rendered difficult because of the fact that premature infants cannot be starved for very long. Food must be supplied in some form, even though diarrhea persists. Certain alterations in the type of feeding are indicated. If breast milk is being fed, it may be acidified with lactic acid, using two drops of acid to the ounce. (Breast milk requires less acid than cow's milk, because of the lower buffer value.) If an artificial formula is being used, a portion of the fat may be removed. It is not generally necessary to decrease greatly the sugar content of the feeding when sugars of the dextrin or maltose type are being fed, but if the sugar used is lactose, it should be decreased or a less fermentable sugar substituted. Premature infants receiving sweet cow's milk formulas should be changed to acid milk. Parenteral injections of dextrose solution may to a considerable extent compensate for a diminished food absorption during periods of diarrhea. These injections of dextrose may be repeated at frequent intervals. (For technic see Chapter XXXIV.)

As premature infants are especially likely to develop rickets vitamin D in goodly amounts should be administered from the time of birth. The newer preparations of vitamin D (viosterol) are less likely to upset the digestion than is cod liver oil. The premature infant should be given 2 to 5 drops daily of the standard "100-D" viosterol solution. As soon as the infant is old enough to stand exposure, irradiation with the ultraviolet lamp may to advantage be instituted.

By the time premature infants have attained a weight of 6 pounds, they may be treated in very much the same way as young full-term infants and the treatment thereafter will be essentially the same as that of average normal infants; the food requirements for a number of months, however, are likely to be relatively high in proportion to the body weight. Premature infants who are free from disease may gain sufficiently rapidly so as to be of average

size and weight by the age of six months. The majority, however, are somewhat undersized and underweight throughout the greater part of the first or even the second year. Ultimately the development both physical and mental is usually as good as that of individuals who have been born at full term. Premature infants are especially prone to convulsions, which in the majority of instances appear to be the result of tetany, although in some, intracranial birth hemorrhage is the underlying cause. Certain infants are born prematurely because of such pathologic conditions as syphilis or other diseases on the part of the mother, and these infants are likely to be handicapped.

CHAPTER XXVIII

COMMON INFECTIONS WHICH ARE ASSOCIATED WITH NUTRITIONAL DISTURBANCES

An important reciprocal relationship exists between infections and nutrition in infancy. Infants whose diets have been inadequate in total fuel value or in such specific factors as certain of the vitamins or protein and who, as a result, have become undernourished are especially susceptible to infections. On the other hand, any infection which gives rise to vomiting, diarrhea, cell destruction or increased metabolism, or which results in diminished secretion of the gastrointestinal juices or diminished absorption from the gastrointestinal tract necessarily affects the nutrition. Infants in general are more susceptible to infections of the rhinopharynx, middle ear and urinary tract than are older individuals, and it is these infections which are especially likely to exert a deleterious effect upon the nutrition.

Because of the fact that any of the symptoms produced as the result of infections are similar to those resulting from an unsuitable diet, a frequent error is made in assuming that the food is at fault when the difficulty is really infection. On the mistaken assumption that the symptoms are due to unsuitability of the food, the diet is often changed by reducing various food elements and this leads only to still greater impairment of the nutrition and to further susceptibility to infection. Certain infections in infancy are so common and so frequently associated with nutritional and gastrointestinal disturbances as to deserve special and somewhat detailed consideration.

Rhinopharyngitis

Rhinopharyngitis, or simple "cold in the head" is of relatively frequent occurrence and of little importance in itself except in so far as extension of the infection to the middle ear, bronchi or lungs is concerned. Infants otherwise in good condition who develop an acute rhinopharyngitis have slight temperature, refuse a portion of the feedings and occasionally vomit. The stools may be somewhat more numerous. The symptoms persist only for a day

or two and require no modification of the character of the feeding, although the infant will himself limit the intake. It is doubtful whether the course of an acute rhinopharyngitis can be shortened materially by therapy, although local treatment may make the infant more comfortable and may prevent extension of the infection. Suitable local treatment consists in the instillation in the nose of one of the following solutions:

(1) *P & I Drops*

		c.c.	or gm.
Phenol (crystals)	gr. iss	0	1
Iodine (resublimed)	gr. ¼	0	016
Petrolatum liquidum q. s. ad	oz. i	30	00

In the preparation of this prescription it is essential that the mixture be placed in a dry bottle, as the presence of moisture or water causes a separation of the ingredients.

(2) *Ephedrin and Neosilvol*

		c.c.	or gm.
Ephedrin (alkaloid)	gr. viiss	0	5
Neosilvol	gr. xxii	1	5
Aquae dest. q. s. ad	oz. i	30	0

This makes a 1½ per cent solution of ephedrin alkaloid in 5 per cent neosilvol. Ephedrin sulphate or hydrochloride may be substituted for the alkaloid, but the latter is less irritating.

Otitis Media

Otitis media is one of the frequent complications of rhinopharyngitis, infants being especially susceptible to this infection because of the fact that the eustachian tubes are short and relatively wide. One of the first symptoms of otitis media in addition to restlessness and fretfulness is likely to be vomiting. This may occur even before there is any fever or local evidence of inflammation on otologic examination. The vomiting may be persistent and projectile. Later, diarrhea may develop. The diarrhea is mild or severe, depending to a considerable extent upon the severity of the infection and the type of infecting organism.

In most instances, the diarrhea is not severe and clears up rather promptly on suitable treatment of the ear infection. Occasionally middle ear infections occur in epidemic form which are due to a toxin-producing strain of streptococcus. The diarrhea is then likely to be more severe, especially in undernourished infants and warrants the designation of "cholera infantum" or "in-

toxication." In these cases severe anhydremia (see Chapters XX and XXXIV) often occurs and unless vigorous treatment is instituted a fatal outcome may be the result. Streptococcus infections of the middle ear are very likely to spread to the mastoid antrum and there become walled off.

Some degree of fever is present in any type of otitis media. In mild infections in well-nourished infants, the temperature does not go above 100.5° or 101° F. Higher temperatures are associated with extension of the process beyond the middle ear. The infant's actions may give no clue as to the location of the infection. There may be some rolling of the head and occasional pulling at one ear in the case of older infants. The diagnosis must be made by otologic examination. For this purpose, an electrical otoscope with magnifying glass attachment is almost essential, as it is difficult to make a satisfactory examination of the ears of a young infant merely with the use of a head mirror and speculum. The ear of the infant differs in a number of particulars from that of the adult and the evidences of middle ear infection are quite different. The following description has been given by Dr. L. W. Dean.

"In infants with an acute otitis media that has clinical significance, there is something about the drum-head or the wall of the external canal which indicates the presence of this infection.

"The membrana tympani is characterized by the absence of those things which are so striking in the same condition in the adult. The blister-like bulging of the drum-head, the bright red color, the yellow spot at the apex of the bulge are all unusual findings in the infant.

"The sinking of the posterior superior canal wall adjacent to the drum-head is not the usual finding with acute otitis media in the adult. There is almost always at least a slight sinking at this point in the infant. Usually this sinking is very marked and has more significance in the diagnosis of this condition in infants than the changes in the membrana tympani. This bulge of the posterior superior wall is common in the infant as compared with the adult, because at this point the antrum of the infant is much closer to the canal wall than in the adult.

"In the adult there is a distinct layer of hard bone between the mastoid antrum and the canal. In the infant this bony wall is often absent. When present it is usually a thin, paper-like layer

of bone which may be readily penetrated with the myringotomy knife. At times the mastoid abscess ruptures into the canal at this point.

Shrapnell's membrane in the infant forms the innermost part of the posterior superior wall of the canal. The bulging at this point may be confined either to the portion of the wall adjacent to the mastoid antrum or that part of the wall formed by Shrapnell's membrane; usually both are involved. This bulging in the posterior superior wall is more red than normal. It looks at times so much like a blister that it is often mistaken for a bulging of pars membrana tensa.

"The incidence of the bulging of the posterior superior wall of the canal in infants with otitis media is influenced by the presence of mesenchyme in the attic. It is not all absorbed until several months or even years after birth. The embryonic tissue is easily infected and when infected it swells. The swollen mesenchyme obstructs the floor of the attic. This separates the mastoid antrum and the attic from the tympanic cavity proper.

"With the growth of the child, the mesenchyme is usually absorbed. Large cavities filled with air appear in it. With the complete absorption, the ligaments which support the ossicles and which, together with the ossicles form the floor of the attic, become separated by distinct air spaces, so that in the older child and in the adult this blocking does not so readily take place. The only exit from the mastoid antrum is through the *aditus ad antrum* and when the aditus, the attic, or its floor is blocked by the swollen embryonic tissue, there may be pus under pressure in the mastoid antrum and no pus in the tympanic cavity. The embryonic tissue may be in the aditus ad antrum, in the attic, or in the floor around the ligamentous bands supporting the ossicles, or in all three places.

"It is the blocking of the exit from the mastoid antrum at one or more of these places that results in the surgical mastoid occurring so frequently in otitis media in the infant. Naturally, it explains why incision of the drum-head does not give relief.

"The changes in the pars membrana tensa are definite. The eustachian tube in the infant is much shorter than in the adult. Its diameter is greater. It has no isthmus. In the infant, it is a good drainage tube, consequently in acute otitis media, bulging of the membrana tympani because of retained fluid, is not always

present. Sometimes there is a distinct bulging of the drum-head in the infant and incision of the drum-head does reveal pus which seems to be under pressure.

"More common than the bulging of the drum-head is a condition that is due to an edema with a resulting thickening of the membrane because of infection in the cavum. It is the difference in structure between the infant and the adult drum-head that accounts for the greater thickening in the infant. The fibrous layer of the pars membrana tensa is much thicker in the adult and more dense; the mucous layer is much thicker in the infant and the lymphatic spaces in the subepithelial layer are larger. This serum logged drum-head is characterized by an early loss of lustre; the membrane does not shine in reflected light as does the normal membrane; it is discolored; early, it is drab, later it may be dark red. The landmarks of the membrane are early distorted because the membrane is thicker. The processus brevis appear smaller. The outlines of the long process of the malleus are not distinct. The light reflex is distorted or lost.

"If there is a question as to whether the drum-head is normal or not, it may be incised. In otitis media, the drum-head does not cut like a normal membrane. It is not like cutting through tense thin paper, but rather like incising paper that has been moistened."

The treatment to be instituted will depend upon the severity and degree of extension of the infection and the state of nutrition of the infant. In healthy well-nourished infants middle ear infections often subside without paracentesis being performed, and this may be observed even in cases where the tympanic membrane is red and moderately bulging. Recovery in these instances is presumably due to drainage through the eustachian tubes and overcoming of the infection by natural resistance. Local treatment of the nose as recommended for rhinopharyngitis is of some value. Ephedrin is especially useful in causing a shrinking of the edematous mucosa surrounding the openings of the eustachian tubes. The instillation of drops containing phenol and glycerin in the ear often relieves pain by the local anesthetic action of phenol, allows the child to get some much needed rest, and possibly has some effect in overcoming the infection, although one could hardly expect very much phenol to find its way through the eardrum. A 5 to 8 per cent solution of phenol is glycerin is

recommended, and may be used, slightly warmed, as often as every half hour. In using these drops, one should bear in mind that the phenol tends to blanch the tympanic membrane.

Unless the temperature falls and evidences of infection subside within a reasonable time, paracentesis should be performed. Paracentesis is required in not more than one-half of the cases of otitis media occurring in well-nourished infants, but is required in almost all cases of otitis media in undernourished and athreptic infants. Paracentesis should be done at the point of greatest bulging of the drum. Following paracentesis, the ears should be gently syringed two or three times a day with sterile normal salt solution. Re-incision may be required if the opening closes before subsidence of the infection. After adequate drainage of the tympanic cavity is established, cessation of the general symptoms may be expected, though the ears may drain for a considerable time, especially in undernourished infants. Profuse drainage continuing for longer than a week or two may indicate poor resistance on the part of the infant, reinfection by way of the eustachian tubes, or extension of the infection to the mastoid. A much rarer cause of continued discharge is tuberculosis of the middle ear.

In undernourished infants with poor resistance, the giving of repeated transfusions and the feeding of an adequate diet offers the best means of improving the resistance. Reinfection of the middle ear by way of the eustachian tubes is most often seen when an infected mass of adenoids is present in the rhinopharynx. Chronically infected tonsils may also be a factor in keeping up ear infection. The indication in these cases is removal of the adenoids or tonsils or both. When the infection has extended to the mastoid, there may be definite local evidences of swelling and edema over the mastoid or merely a bulging of the posterior superior canal wall and adjacent drum-membrane. In some cases roentgenographic evidence of mastoid involvement is obtainable; this is, however, not to be relied upon as many mastoid infections in infants fail to show in the roentgenogram. When definite mastoid infection is present, drainage of the mastoid antrum or a more extensive mastoid operation is indicated.

The treatment of such gastrointestinal symptom as may be present is that outlined in Chapters XX and XXIII. The infant should not be forced to take food beyond the desires of his

appetite, and water should be supplied freely especially if any evidences of dehydration have developed.

Mastoiditis

Probably in the majority of instances of otitis media, extension of the process to the attic and mastoid antrum occurs, especially if much pus has accumulated. The mastoid antrum in the infant is present at birth as a single small cell. Later this enlarges and with growth and pneumatization of the mastoid processes communicates with other cells. During early infancy the antrum and middle ear form essentially one cavity, communicating through the *aditus ad antrum.* When the tympanic cavity is drained, the antrum also drains unless obstruction occurs at the aditus. Such obstruction may occur, however, if the aditus is narrow as the mucosa readily swells so as to shut off completely the antrum from the tympanic cavity. In these instances the infection remains in the antrum and may give rise to constitutional manifestations. In infants who are undernourished and whose resistance is lowered, infection in the antrum tends to spread into the adjacent bone, even though blocking off may not be complete. It is in such infants that severe constitutional symptoms are seen and in whom the infectious process in the antrum persists despite free drainage of the middle ear, through a tympanic opening. A variety of organisms may be responsible for mastoid antrum infections, hemolytic streptococci being especially frequent and serious offenders. The general symptoms of mastoid antrum infection do not usually differ greatly from those of middle ear infections with the same organism except that the symptoms are likely to be somewhat more marked and to continue for longer periods of time. In many cases of mastoid infection with such organisms as the staphylococcus occurring in well-nourished infants, no effects other than those of any febrile disturbance may be observed, even though large subperiosteal abscesses occur. There may be little or no vomiting or diarrhea and no tendency to anhydremia. On the other hand, in malnourished infants with hemolytic streptococcus infections, or with mastoid infection with organisms of the intestinal group, marked general symptoms of diarrhea, vomiting and anhydremia may be present despite the fact that local evidences of infection in the mastoid are but slight.

The local evidences in some instances amount to no more than

a swelling of the posterior superior canal wall and adjacent drum-membrane. There may be no tenderness, redness or swelling over the mastoid region. Roentgenograms may reveal a clouding of the antrum and even of the whole mastoid process but inasmuch as there is so much individual variation in normal infants in the degree of pneumatization of the mastoid and size of the antrum, roentgenograms should be interpreted only in comparison with the opposite side and if this also is involved, it may be impossible to reach a conclusion as to the significance of the pictures. In some cases a definite diagnosis of mastoid or mastoid antrum involvement cannot be made. The condition can only be suspected on the basis of the symptoms and by exclusion of other causes for the fever or general symptoms. Not infrequently extensive mastoid involvement is discovered at autopsy when very little evidence of the condition has been present up to the time of death. Occasionally an otitis media may subside entirely, but at autopsy the mastoid is found filled with pus. When mastoid infections of the type described are recognized and suitably treated by surgical drainage, a prompt disappearance of the general symptoms follow in the majority of instances.

There is another type of mastoid involvement which is seen almost exclusively in malnourished infants and which appears to be secondary to gastrointestinal disturbance or to intestinal infection. In these infants the middle ear and mastoid involvement is not present until late in the course of severe diarrhea. The ear infection is not preceded by rhinopharyngitis. The local evidences in the tympanic membrane are slighter than in the case of the primary ear infections and the local evidences of the mastoid involvement are likewise slight even in cases in which an extensive necrosis is found at autopsy. In these secondary types of mastoid involvement, the organisms found are, in most instances, intestinal types, such as strains of the B. coli, the Bacillus of Morgan or members of the paratyphoid or paradysentery group. The organisms obtained on culture from mastoids are often different from those obtained from the middle ear, thus suggesting the possibility of direct blood-stream infection, although it appears probable in most instances that the intestinal organisms reach the ears from vomited material by way of the eustachian tubes.

Coincident with the development of the mastoid infections in infants suffering from diarrhea, there usually occurs an exacerba-

tion in the gastrointestinal symptoms and an elevation of the temperature above the previous level. In these cases, drainage of the middle ear and mastoid does not result in the same degree of improvement in the general and gastrointestinal symptoms as is observed in primary types of ear infection previously described. The mortality in these cases is very high because of the fact that the infants who develop the complication are already in a precarious condition and are still further weakened by the added infection.

The treatment of mastoid infections in infants depends upon the condition of the infant and the severity of the process. Many cases of mastoid infection in well-nourished infants clear up spontaneously or following adequate middle ear drainage. In others, it becomes necessary to drain the mastoid. In cases of antrum infection with extension to the remainder of the mastoid process, antrotomy or "post-auricular drainage" is all that is required. This simple operation is performed under local anesthesia and may be accomplished with very little disturbance or shock. The operation consists merely in the removal of a small button of bone over the mastoid antrum. The pus in the antrum should be gently removed with a swab and a gauze drain should be inserted. Extensive curettage should not be attempted especially in the case of malnourished infants as this may damage the mucosa and adjacent bone and favor extension of the process. The wound should be kept open for a reasonable time by the insertion of a drain. Healing is usually prompt in well-nourished infants, but in athreptic infants is, at times, very slow, there being often but little evidence of repair for a period of weeks. In cases of extensive involvement of the mastoid, more radical operation is, of course, required. In all cases of mastoid infection, suitable treatment of the rhinopharynx and middle ear should be continued, the fluid balance of the body maintained and adequate food given.

Pyelitis

Pyelitis is a frequent cause of nutritional disturbance during infancy and one which is likely to be overlooked, since there may be no symptoms directly referable to the urinary tract. The frequent occurrence of pyelitis in infants may be explained on the basis of lack of immunity to colon bacillus infections and to anatomical peculiarities of the urinary tract. Pyelitis may occur

in well-nourished infants, but is distinctly more frequent in those whose nutrition is below par and in those whose resistance is lowered by the presence of other infections. Thus the development of pyelitis is often observed following acute or chronic rhinopharyngeal or ear infections. The first evidences of pyelitis may appear after an infant has suffered from a gastrointestinal disturbance. It is possible that the intestinal wall becomes permeable to colon bacilli under such circumstances and that the organisms reach the kidney by way of the blood stream or lymphatics. The development of pyelitis is favored by any anomaly of the urinary tract, such as strictures or kinks in the ureters or disturbances in the neuromuscular function of the bladder.

Whatever the cause of pyelitis, the effects on nutrition may be marked. The infant's appetite is lessened and vomiting is of frequent occurrence. There is occasionally diarrhea. Even infants who take fair amounts of food and do not show much gastrointestinal disturbance fail to show a normal gain in weight. Infants with pyelitis are pale and pasty in appearance and are often languid and apathetic.

A definite diagnosis of pyelitis can be made only by urinary examination. A voided specimen of urine is satisfactory for examination in the case of males, but in females a catheterized specimen is preferable because of the possibility of contamination with pus cells from the vagina or labia. The methods of collecting urine are described in Chapter XXXIV. An uncentrifuged specimen should be examined under the high power of the microscope. The diagnosis of pyelitis is warranted if there are more than 8 or 10 leucocytes present to a field and especially if they are in clumps. A single urine specimen may be free from pus and subsequent specimens may contain large amounts. This probably depends upon retention at some point in the kidney or ureter.

The most suitable treatment in most cases consists in the administration of alkali in the form of sodium bicarbonate, sodium citrate or fruit juices in sufficient amount to render the urine alkaline to litmus and to keep it alkaline continuously. Potassium citrate, although frequently recommended, should be avoided as there is a danger of retention of potassium salts in sufficient amounts to exert a toxic effect in those cases in which renal function is impaired as a result of pyelitis. The amount of alkali which is necessary in order to accomplish the desired result varies

greatly in individual infants. There is some danger in giving too much alkali especially in cases in which vomiting is a pronounced feature as these infants through loss of hydrochloric acid in the gastric juice may already be suffering from a condition of alkalosis. In such instances alkali administration may precipitate the symptoms of tetany. The administration of alkali is for the purpose of rendering the urine less irritating to the injured mucosa, not to exert an antiseptic effect, for colon bacilli actually grow better in an alkaline than in an acid urine. Coincident with the alkali administration, there should be an abundant intake of water.

If after a fair trial of alkali treatment, the symptoms do not subside, other methods of treatment should be applied. The most effective of these is the administration of methenamin (urotropin) by mouth. This substance is excreted in the urine where in the presence of acid it is partially decomposed with the liberation of formaldehyde. The dosage of methenamin for an infant is from 3 to 7.5 gr. (0.2 gm. to 0.5 gm.) as often as four times a day. Coincident with the administration of methenamin, alkali administration should be stopped and substances administered which will render the urine acid in reaction in order that the methenamin may be effective. The most suitable substance to use for this latter purpose is ammonium chloride, which may be given in a dosage about equaling that of the methenamin. Methenamin administration, especially in the acute stages of pyelitis, may cause temporary hematuria. It may be necessary to keep up methenamin treatment for long periods before the urine becomes free from pus. In some instances good results are obtained by alternating alkali and methenamin-acid therapy. The above lines of treatment are those which are usually the most satisfactory. Other methods of treatment have been recommended, such as the administration of hexylresorcinal (Caprokol). This latter method has, in our experience, been disappointing and frequently leads to vomiting. Caprokol is said to be more effective in cases of pyelitis due to streptococcus or staphylococcus infection than in the usual type due to the colon bacillus.

In cases which do not respond to medical treatment, there may be present infections elsewhere in the body, as in the adenoids and tonsils. When such infections are present, they should be treated appropriately and this is often followed by clearing of

the pyelitis. In certain chronic cases of pyelitis, anatomical conditions are present, such as ureteral obstruction which can only be diagnosed by cystoscopy and treated by special surgical procedures. Renal calculi, when present, may cause pyelitis, but such calculi are rarely present during infancy.

The diet in cases of pyelitis should be one suitable to build up the nutrition. It need not differ in this respect from that of any other infant of the same age. It is especially essential, however, that cod liver oil be given in order to provide sufficient vitamin A to render the mucous membranes resistant to infection. Transfusion of infants with adult's blood appears to increase the degree of immunity to B. coli and is a valuable procedure.

Tuberculosis

Tuberculosis of young infants is typically an acute disease. It may occur in well-nourished, apparently healthy, infants and runs its course in a few weeks to a fatal termination with meningeal involvement. Even at the time of death, the state of nutrition may still be excellent. In older infants a more chronic form of the disease may occur, which is associated with gradually failing nutrition. In this latter type the infection is localized largely in the glands, especially the peribronchial and cervical glands; less frequently the glands of the mesentery. In this country, tuberculous infection is usually acquired through human contact and the primary lesion is, in the majority of instances, in the lungs, with secondary extension to the peribronchial glands. When the infection occurs in a young infant or when it is a massive one, extension by way of the blood stream to the meninges is common even when the pulmonary involvement is but slight.

In the case of less extensive infections and better resistance on the part of the infant, the primary lesions in the parenchyma of the lungs may become well walled off and calcified, but the bronchial glands draining the affected areas of the lungs usually become involved. The infection may be quiescent for long periods of time in older infants, but may at any time light up when the resistance is lowered due to inadequate nutrition or to superimposed infections, especially those of the respiratory tract. During the quiescent stage, the infant shows no acute symptoms but often fails to gain at a normal rate, is anemic and suffers from lack of appetite. Physical signs in the lungs are slight, there being oc-

casionally signs which would be interpreted as those of a localized bronchitis. The roentgenogram shows enlarged tracheo-bronchial glands and some infiltration about the hilum. The same x-ray picture may, however, be seen in the case of chronic rhino-pharyngeal infections with secondary pulmonary involvement. In cases of this type, the intradermal tuberculin test is of the greatest value in indicating the presence of tuberculous infection. Infants who are in poor nutritional condition at the time the test is made may fail to react unless a fairly large amount of tuberculin is used. The ordinary Pirquet scarification technic is not nearly so trustworthy as the intracutaneous test. For the latter, a fresh solution of "old tuberculin" (O.T.) of a strength of 1:1000 is prepared and 0.1 c.c. injected intracutaneously in the same manner as for the Schick test. This corresponds to 0.1 mg. of tuberculin. A positive reaction is definite indication of the presence of tuberculous infection and in a young infant tuberculous infection usually means active tuberculous disease. A negative test with 0.1 mg. of tuberculin does not always exclude tuberculous infection and where there is a strong suspicion of tuberculosis, larger doses of tuberculin should be employed, up to as much as 1.0 mg. When a positive tuberculin test is obtained on an infant under two years of age, the prognosis is always grave, but not necessarily hopeless provided the infant's nutrition can be maintained.

The tuberculous infant should receive an especially liberal diet. The allowance of milk should be closer to two ounces per pound of body weight than the customary one and one-half ounces. It is often desirable to give a somewhat lesser proportion of carbohydrate than in the case of normal infants to insure a larger intake of milk. In any event, however, the feeding will usually have to be a concentrated one, as an adequate amount of food cannot be given to these infants in the form of dilute formulas. At least 3 teaspoonfuls of cod liver oil should be administered daily. Viosterol is not a satisfactory substitute. Good ventilation of the living quarters and an abundance of fresh air are essential, but direct exposure to sunlight or to artificial sources of ultra-violet light is not advisable in the pulmonary forms of tuberculosis in infancy. In the case of any infant with tuberculosis, the human source of the infection should, if possible, be determined and the infant removed from anyone suffering from active tuberculosis.

If the mother is suffering from the disease in active form the infant should invariably be weaned promptly and the mother should not come in contact at all with the infant.

Congenital Syphilis

Congenital syphilis, like other chronic infections, may influence nutrition profoundly, even though the infection be of the latent type without active clinical manifestations.

Congenital syphilis is not a rare disease. It is estimated that approximately 1½ per cent of all infants in this country are infected with syphilis at the time of birth. The incidence, however, varies greatly among different classes of society. Among negroes an incidence as high as 15 per cent has been reported. Among the lower classes of whites, the incidence approximates 2 per cent and among the better classes something less than one per cent. Among premature infants the incidence of syphilis is especially high, inasmuch as syphilis is an important cause of premature birth. Many infants infected with syphilis are still-born or fail to survive longer than a few days, so that the actual incidence of syphilis after the neonatal period is much lower than the figures given above.

In many instances the congenitally syphilitic infant is apparently normal at birth, no characteristic symptoms developing until after the lapse of several weeks. The most characteristic of the syphilitic manifestations during the first weeks of life are the rash and the rhinitis, but neither of these symptoms may appear and the rhinitis may be mistaken for an ordinary cold in the head. Careful examination of these infants may reveal some thickening of the palms and soles and occasionally desquamation. In a fair proportion of cases of congenital syphilis, the spleen becomes palpable during the first month or so of life and this may be the only clinical manifestation of the disease. The liver is often enlarged somewhat, but inasmuch as there is considerable variation in the size of the liver, even in normal babies, this finding is of less significance. Well-marked cases of clinically active congenital syphilis are easily diagnosed, but the latent cases without clinical manifestations often escape diagnosis unless one's suspicion has been aroused by a family history of syphilitic infection or of repeated miscarriages or premature births.

Infants with latent congenital syphilis often develop in an en-

tirely normal manner and the diagnosis is only made when a routine Wassermann reaction or Kahn test is performed. There are, however, other infants who show none of the marked clinical evidences of congenital syphilis, but who fail to do well even when fed at the breast or given artificial formulas which should be adequate. The true nature of the condition is revealed only by a positive blood test. The Wassermann reaction is of distinctly greater reliability during infancy than during later life. A positive Wassermann after the age of two months may be taken as conclusive evidence of the presence of syphilis and a negative reaction as indicating freedom from the infection. In our experience exceptions to this rule have been so rare as to be negligible. Some infants with congenital syphilis may, however, show a negative Wassermann reaction during the first month or two of life; a positive test on the blood of the mother supplies confirmatory evidence in such cases. There are occasional instances in which the mother's blood is positive and also the infant's blood at the time of birth, but in which the infant's blood later becomes negative and no clinical evidences of the disease develop.

Once the diagnosis of congenital syphilis is established, antisyphilitic treatment should be instituted promptly, whether or not active clinical manifestations are present. With the institution of such treatment and without any change in the character of the feeding, many of these infants begin to thrive and may develop in an entirely normal manner. The earlier treatment is begun, the more satisfactory are the results obtained.

In the treatment of congenital syphilis care should be taken that the treatment is not too vigorous at the outset and mercury should always be administered for several weeks before the use of the arsenicals. In general, mercury should be administered once weekly either by inunction of the blue ointment (*hydrargyrum cum creta*) in doses of one to two grains or by intramuscular injection of a 1 per cent solution of mercury bichloride; this latter is given in the proportion of ½ minim per kilogram (2.2 pounds) of body weight. In addition to the mercury, a course of three intramuscular injections of sulpharsphenamin at weekly intervals should be administered every two months. Sulpharsphenamin is given in the amount of 0.02 grams per kilogram of body weight.

The proper feeding of the syphilitic infant is almost as important as specific antisyphilitic treatment. If the mother is in good

physical condition and has an abundance of milk, the infant should, by all means, be nursed. A latent syphilitic infection on the part of the mother does not influence the character of her milk, nor is there any likelihood of a mother contracting syphilis from the infant. Because of the fact that the nutrition of the syphilitic infant is likely to be poor, even with good feeding, the infant should be given the advantage of breast feeding whenever possible. In case this is not possible, due to inadequacy of the mother's milk supply, or poor health on her part, the artificial feeding given should be a generous one, as these infants suffer more from inability to assimilate food than from digestive disturbances. A concentrated feeding having a high caloric value and containing adequate milk should be given. It is important that the intake of food should not drop below the optimum requirement because of lack of appetite on the part of the infant. Every effort should be made to insure a sufficient intake either by the use of concentrated food or if necessary by resort to gavage. With proper antisyphilitic treatment, the infant's ability to utilize the food given may be expected to increase.

CHAPTER XXIX

RICKETS

Rickets is the most common nutritional disorder affecting infants living in temperate zones. It has been estimated that from 60 to 90 per cent of all infants in the larger cities show some evidences of rickets during the first two years of life. Although rickets may be active at any time during the growing period, it is most frequently observed during infancy, especially between the ages of six months and two years.

Etiology

Severe rickets is of more frequent occurrence in the dark-skinned than in the light-skinned races, especially when removed from their natural environments. Rickets is not common among negroes and Italians in Africa or in Italy but is very common among these same classes living in American cities. The condition is somewhat more frequent in the infants of mothers whose diets during pregnancy and lactation have been faulty, especially in the vitamin D factor. Infants born prematurely are very susceptible to rickets; rapidly growing infants are much more likely to develop the condition than those whose growth rate is slow. Undernourished and athreptic infants rarely show marked evidences of rickets but often develop active rickets when growth is resumed.

Diet plays an important rôle in the causation of rickets. The disease is distinctly more frequent in artificially fed than in breast-fed infants, but severe grades of rickets may be seen in infants who are exclusively breast-fed, especially negroes. The most important single factor in the diet in relation to the development of rickets is vitamin D. Human milk contains no more of this vitamin than does cow's milk, but the majority of artificially fed infants receive less milk than breast-fed infants. There is some evidence that vitamin D is less well absorbed from cow's milk than from human milk, probably due to the fact that a portion of the vitamin is lost along with unabsorbed fat by way of the bowel. When liberal amounts of extra vitamin D are added to the

diets of artificially fed infants, rickets may, in most instances, be prevented. Rickets is as frequently observed in infants fed on raw milk as in those fed pasteurized, boiled, evaporated or dried milk. Rickets is especially common in infants whose diets have contained large proportions of carbohydrate. In these cases the disorder is probably attributable to the proportionately smaller amount of milk taken, rather than to any specific harmful influence of carbohydrate. It has, however, been suggested by Mellanby that certain cereals contain a "toxamine" or "anti-vitamin" which causes rickets, unless the effect is neutralized by the administration of extra amounts of vitamin D. When the diet contains large amounts of fats which are deficient in vitamin D, the development of rickets is favored. Rickets is but little influenced by the amount of protein in the diet.

Although rickets may be produced experimentally in animals by feeding diets deficient in calcium salts, or in phosphates, calcium or phosphorus deficiency in the diet is rarely, if ever, the cause of infantile rickets. Human milk, when taken in sufficient amounts to supply the fuel needs, also provides sufficient calcium and phosphorus. Cow's milk contains from three to four times as much calcium and from seven to eight times as much phosphorus as human milk and even when diluted provides an abundance of these elements; calcium absorption from cow's milk is, however, not so good as that from human milk. The feeding of an excess of calcium when but little phosphate is present in the diet leads to a loss of phosphate from the body and causes the development of rickets in experimental animals.

Of equal or greater importance than diet in the etiology of rickets is exposure to sunlight. Rickets has been aptly described as a "disorder of darkness." Rickets is a rare disease in the tropics and in country districts where babies spend considerable time out of doors. The predisposition of dark-skinned races to rickets is probably in part explained on the basis of the shielding effect of pigment against the sun's rays. It is only certain rays in the sunlight which are effective in preventing rickets, and these are the shorter ultraviolet rays, ranging in length from 250 to 320 millimicrons. "Skyshine" or the light reflected from the sky, clouds and other light surfaces, is from one-half to two-thirds as effective as direct sunlight. Light which has passed through glass contains practically none of the short length antirachitic

waves. For this reason, children who are constantly kept indoors, even though in well lighted rooms, are prone to develop rickets. Ultraviolet light, produced artificially by means of the mercury vapor or carbon arc, has an antirachitic effect comparable to that of sunshine. In general, the effects of ultraviolet rays on the metabolism are the same as those of vitamin D, and a deficiency of vitamin D in the diet may be compensated for by exposure to light. Irradiation of the body is, if anything, more effective than the administration of vitamin D in the prevention of rickets. The greater frequency of the active manifestations of rickets during the late winter and early spring months is probably to be explained on the basis of lesser exposure to sunlight. The effect of ultraviolet light on the body is, in part, due to the activation of lipoids in the skin, which are closely similar or identical with ergosterol. (See Chapter VII.)

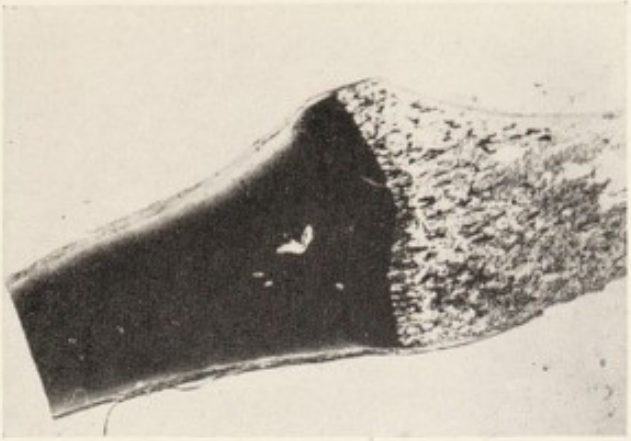
Rickets is not ordinarily associated with disturbances of any of the endocrine glands, but a form of rickets is occasionally observed which is associated with dysfunction of the parathyroid glands. Parathyroid insufficiency is associated with a low calcium content of the blood and tetany. In this condition changes may occur in the bones which are indistinguishable from rickets. The administration of vitamin D in such cases results in an increase in the calcium of the blood and a healing of the rachitic process.

There is no good evidence that rickets is caused by infections or toxic agents.

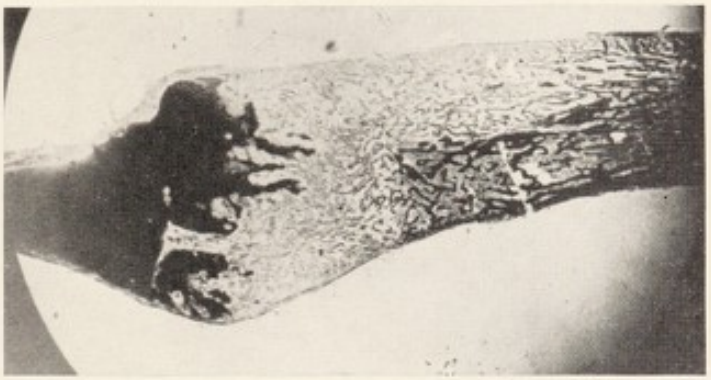
A condition closely resembling rickets is seen in certain cases of chronic renal insufficiency in which there occurs a marked retention or inorganic phosphates in the blood and a coincident decrease in calcium content. This condition of "renal rickets" is observed chiefly during middle childhood, but we have seen instances of it in late infancy when congenital malformations of the kidney were present.

Pathogenesis and Pathology

The most characteristic manifestation of rickets is a failure of calcification of newly formed osteoid in the developing portions of the bones. This failure of calcification is most easily observed at the epiphyseal ends of the long bones. Here, under normal conditions of growth, osteoid or the organic matrix of bone is laid down and promptly filled with a deposit consisting chiefly of



A.



B.

C.

Fig. 26.—Sections of normal (*A*) and rachitic (*B* and *C*) ribs. Note regular line of calcification at the costochondral junction in the normal rib and the irregular line in the rachitic ribs. The softened rachitic ribs show bending inward as the result of external air pressure.

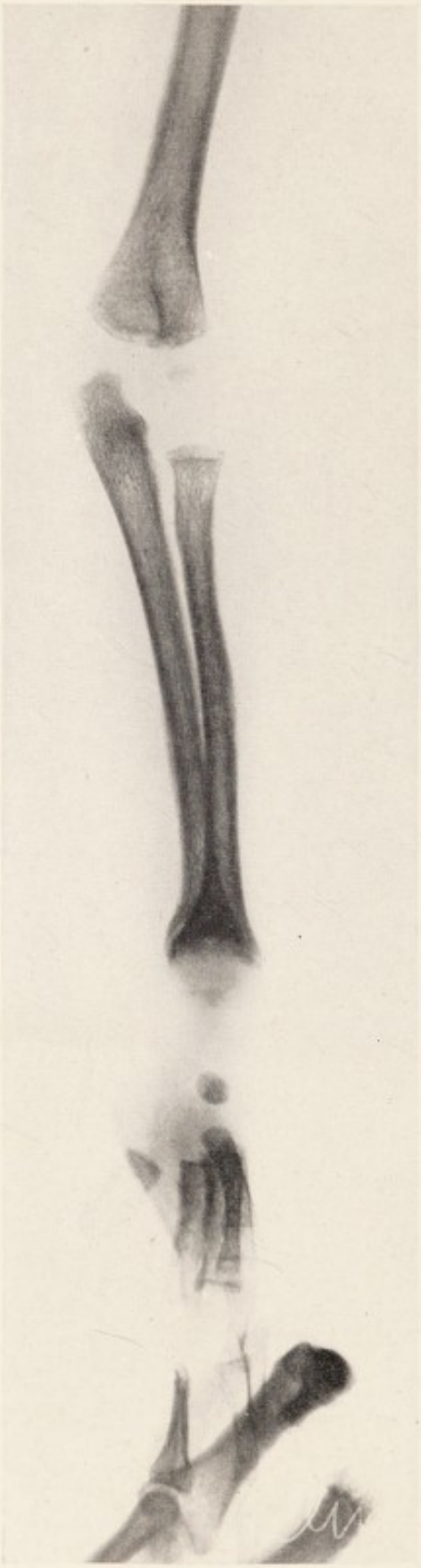


Fig. 27.—Rickets. Irregular line of calcification at lower end of radius, with cup-like contour.

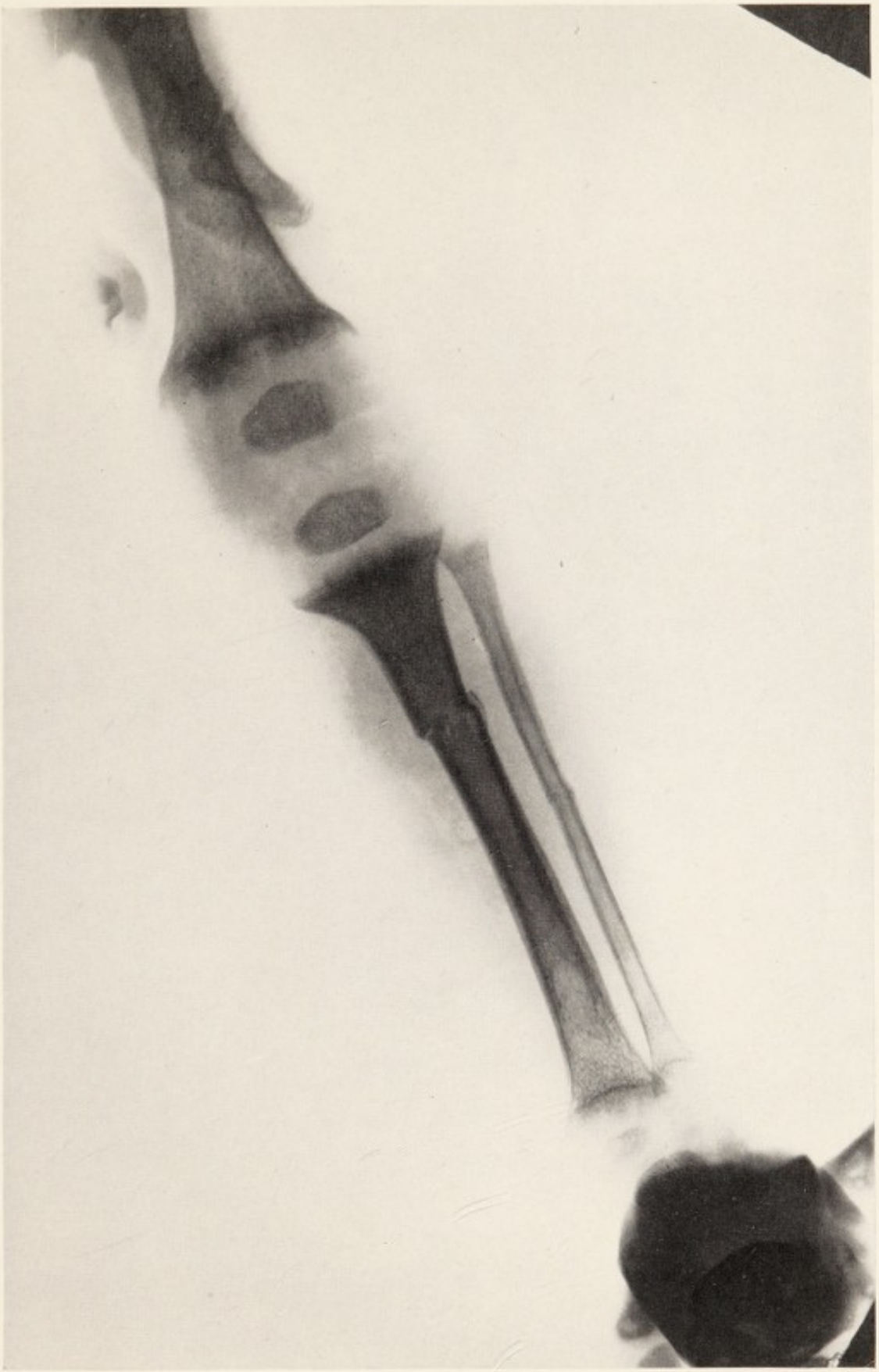


Fig. 28.—Rickets. Irregular calcification at ends of long bones with distortion. Fractures of tibia and fibula.

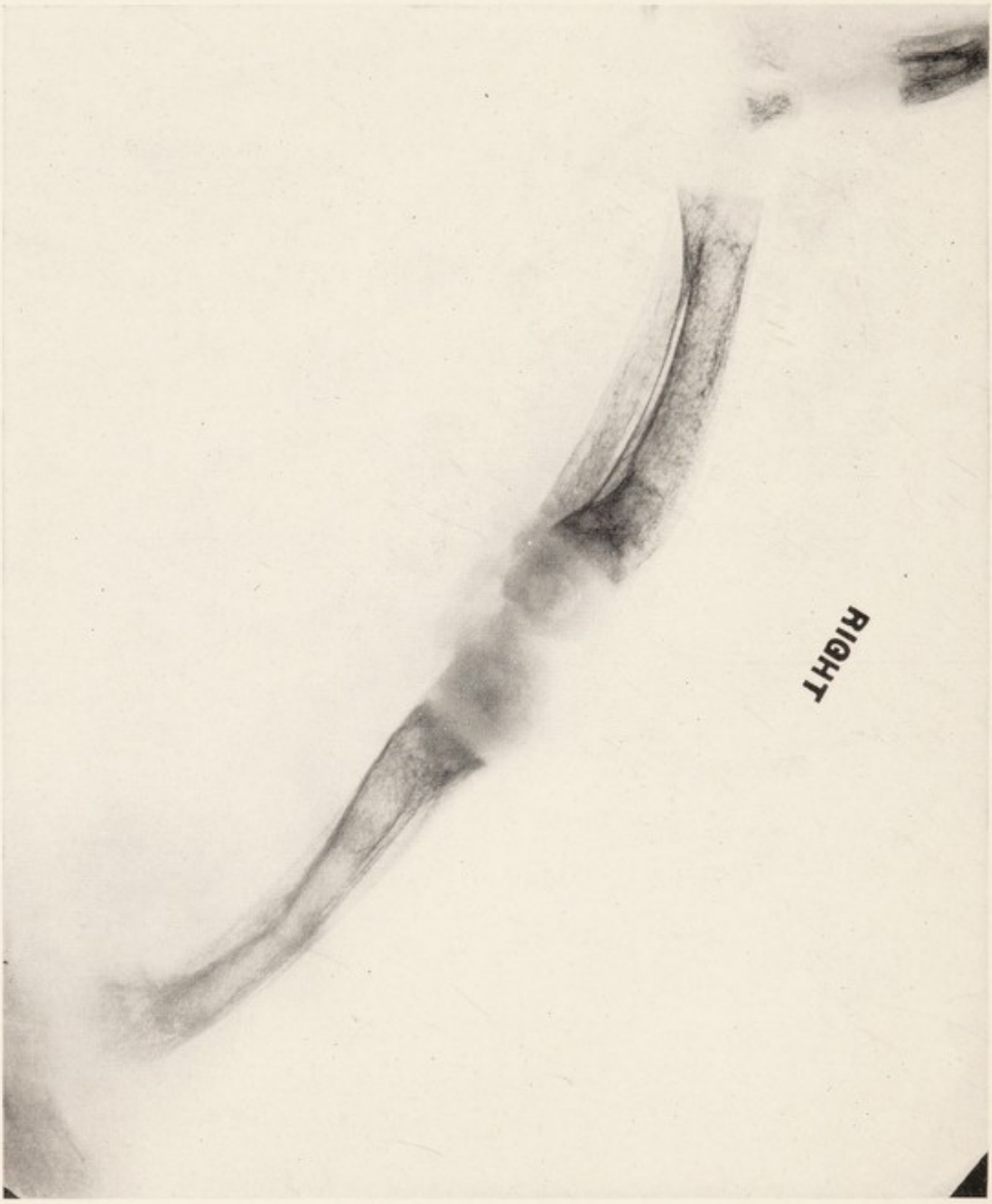


Fig. 29.—Severe rickets. Rarefaction of bones. Incomplete ossification of epiphyseal centers.

calcium phosphate with small amounts of calcium carbonate. In rickets, the osteoid is laid down in an essentially normal manner, but calcification is incomplete and irregular (Fig. 26). This results in the formation of bone, which is soft and easily deformed. X-ray pictures of the epiphyses reveal an irregular line of calcification (Figs. 27 and 28), rarefaction of the bones and delayed or incomplete ossification of the epiphyseal centers (Fig. 29). Fractures often occur (Fig. 28). Incomplete calcification also occurs in the bones laid down by periosteum.

The failure to deposit lime salts is not attributable to lack of calcium in the circulating blood, for except in certain rare forms of rickets the blood calcium content is normal or but slightly reduced. On the other hand, the phosphate content of the serum is markedly diminished. The normal amount of phosphate in the serum of infants, expressed in terms of phosphorus, is from 5 to 6 mg. per 100 c.c.; in active rickets, the amount is reduced and may be as low as 1 or 2 mg. per 100 c.c. The low phosphate content of the blood plasma is not the result of a deficiency of phosphorus in the diet, nor may the amount be raised appreciably by the administration of extra phosphate. It would appear that the blood becomes incapable of carrying phosphate. The administration of vitamin D in the form of cod liver oil or irradiated ergosterol or exposure of the body to sunlight or ultraviolet light is followed by a rise in the phosphorus of the blood plasma and healing of the rachitic process. No appreciable change occurs in the calcium concentration of the blood during the stage of healing rickets, but there is a retention of a larger amount of the calcium of the food; this is, of course, deposited in the bones in the form of phosphate and carbonate.

Although the most demonstrable changes in rickets are seen in the bones, rickets is not merely a disease of the bones but a general disease affecting other parts of the body as well. The muscles throughout the body are soft, flabby and atonic and may show slight microscopic changes at autopsy. The smooth musculature of the intestinal tract is also hypotonic. The ligaments are lax. A catarrhal condition of the mucous membranes of the respiratory and gastrointestinal tract is commonly observed but may be dependent upon associated dietary deficiencies rather than upon the rickets itself. In a fair proportion of cases of rickets, the spleen shows enlargement, and this may be extreme when

a severe degree of secondary anemia is associated with the rickets. Microscopic changes in the spleen are those of simple hyperplasia. The tonsils, adenoids and cervical glands are frequently enlarged, but this enlargement is probably due not so much to rickets as to the chronic infections to which rachitic infants are susceptible. The bone marrow shows some degree of atrophy with a deficiency in cellular elements. This bone marrow atrophy probably accounts for the anemia so often associated with rickets. The anemia is of a secondary type and may be severe. In some cases, the anemia is associated with a lymphocytosis and marked splenic enlargement and corresponds closely to the "von Jaksch's" type of pseudoleucemic anemia or splenic anemia.

Symptoms

The most characteristic symptoms of rickets are referable to the bone changes. The bones throughout the body are not always affected to an equal degree; in some infants the most marked changes are in the cranium, in others, in the extremities or thorax.

The head appears large, due chiefly to the development of thickened areas or bosses in the temporal and parietal regions (Fig. 30). The top of the skull is flat and often depressed toward the middle. The anterior fontanelle does not close at the regular time and may be widely open, even after the age of two years. Areas of softening of the cranial bones (craniotabes) may be detected on palpation. These areas are most frequently found in the neighborhood of the lambdoidal sutures either in the parietal or occipital bones. Such soft areas may be present in the skulls of newly born infants who show no other manifestations of rickets; hence, craniotabes is not of significance unless developing during later infancy.

In the thorax, the costochondral junctions are enlarged and may be felt as rows of beads running parallel to the sternum and curving outward toward the lower end of the chest (the *rachitic rosary*) (Fig. 30). In cases of marked and long-continued rickets, deformity of the chest occurs, with sinking in along the lines of the costochondral junctions. (Figs. 30, 31, 32.) In these cases the bending in of the ribs and cartilages may be sufficient to compress the lungs and interfere with normal expansion. Associated with the sinking in of the thorax is a flaring of the lower ribs covering the liver and upper portions of the abdomen. At the upper edge

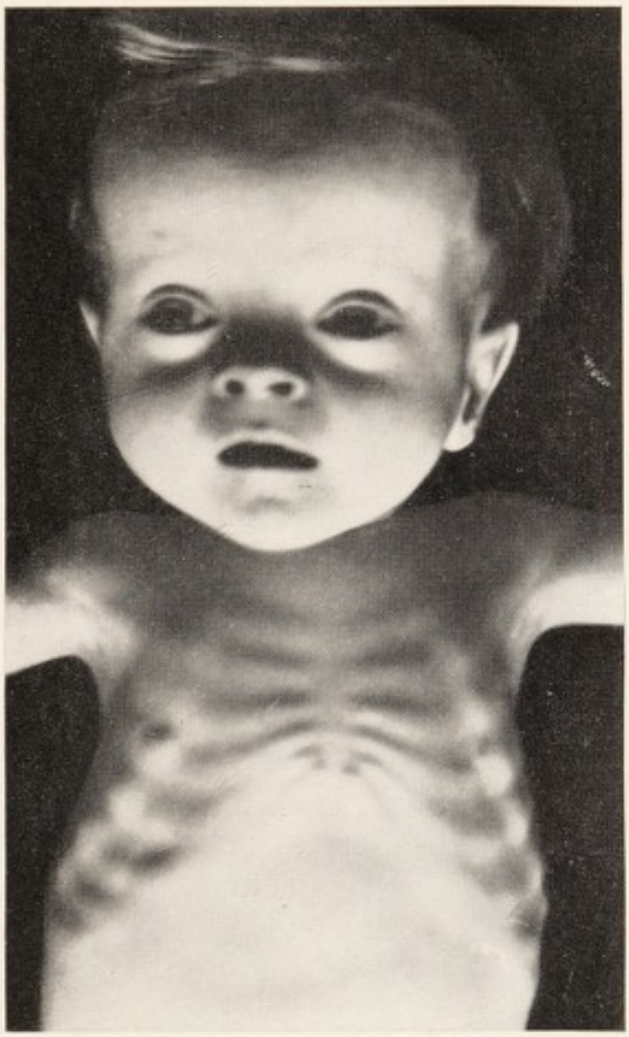


Fig. 30.—Rickets. Square head, frontal bosses, enlarged costochondral junctions ("rachitic rosary"), and Harrison's groove.

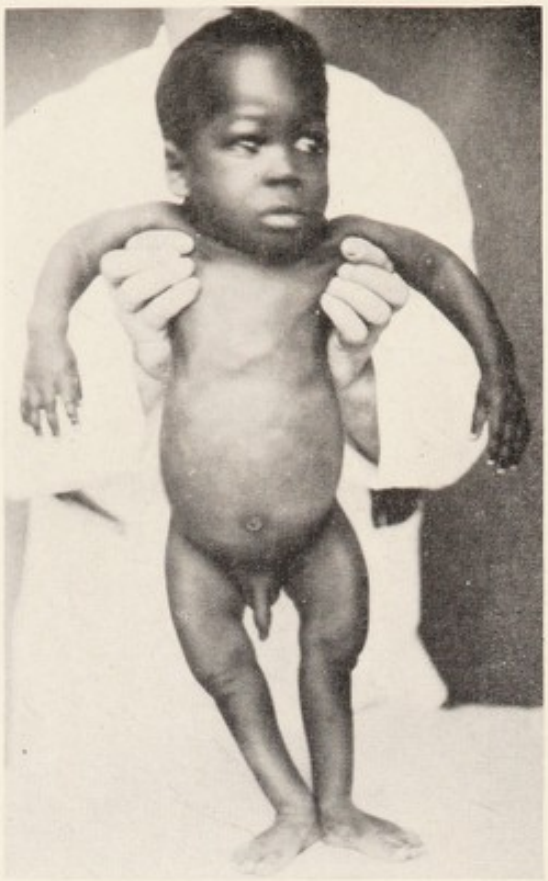


Fig. 31.—Rickets. Deformities of head, chest and extremities.

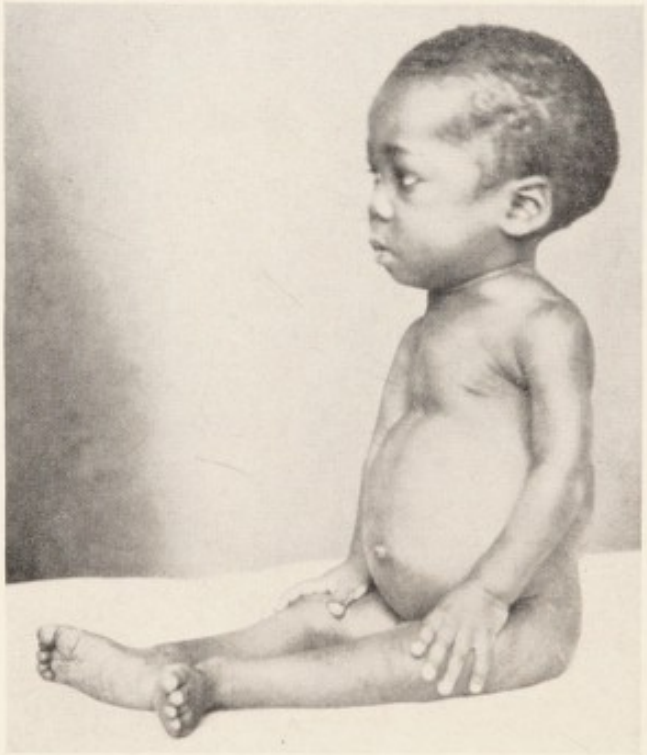


Fig. 32.—Rickets. Characteristic posture, deformities of chest and extremities, "pot-belly."

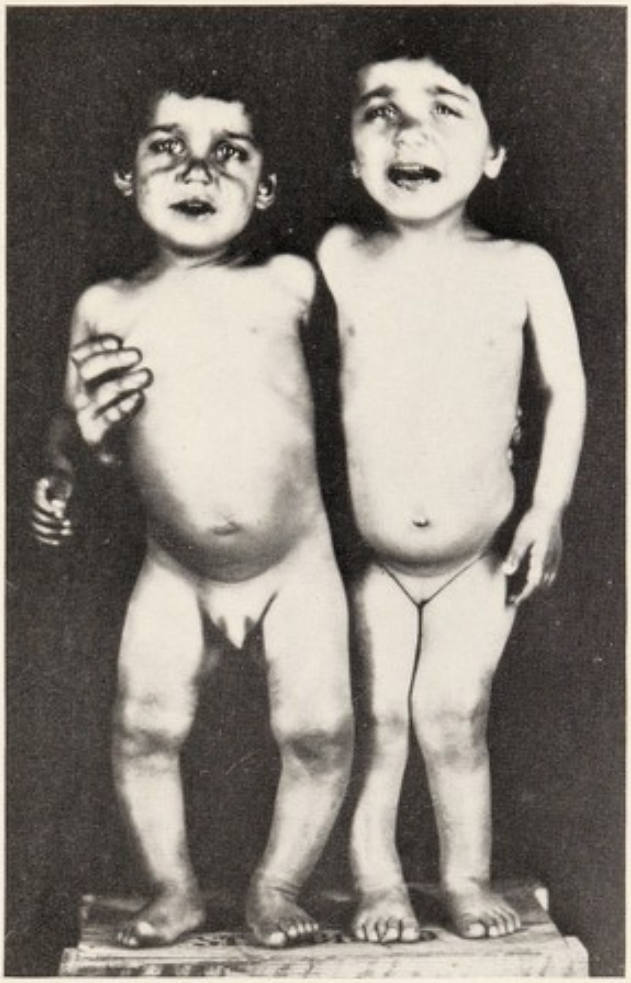


Fig. 33.—Rickets. Deformities of extremities, bowlegs and knock-knees.

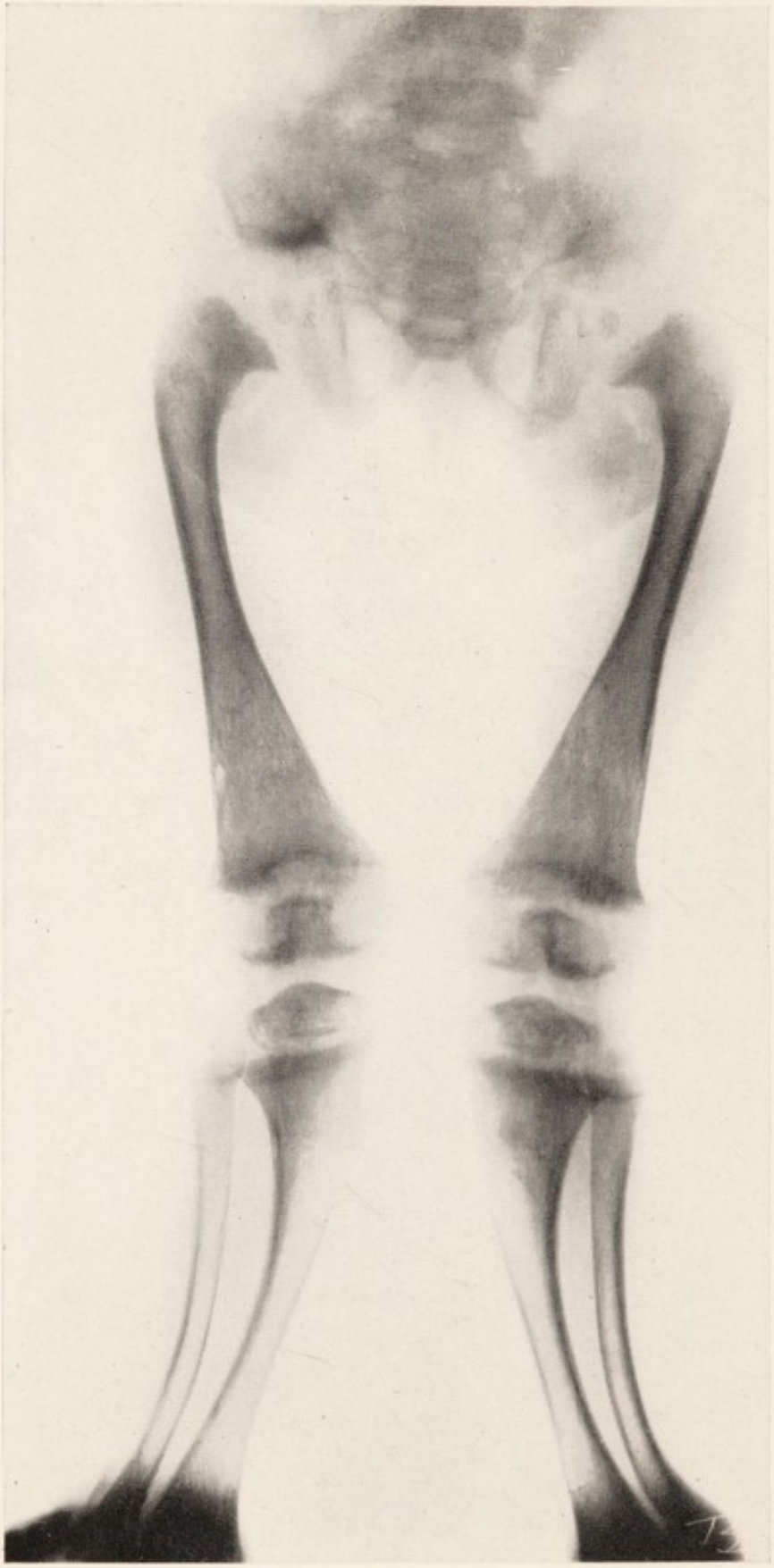


Fig. 34.—Rickets. Rarefaction and deformities of bones. Knock-knees.

Fig. 35.—Rickets. End-result of severe rickets during infancy.

of the flaring following the line of the diaphragmatic attachment a gutter-like depression occurs (Harrison's groove.) The lower end of the sternum may be retracted so as to give rise to a funnel-shaped depression; such a deformity, however, may be congenital or the result of other causes, as for example respiratory obstruction, due to adenoids.

The long bones of the extremities show more or less deformity, depending upon the degree and duration of rickets and the amount of mechanical stress to which the bones have been subjected. In almost all cases of rickets, there is some enlargement of the epiphyses at the wrist. In children who are old enough to sit up or to stand on the feet, bowing of the thighs and legs occurs (Figs. 31 and 33). The rachitic child tends to sit in a cross-legged, tailor-like fashion, resting a part of the weight upon the arms; this causes deformity of both arms and legs (Fig. 32). Instead of being symmetrically bowed, the legs may be twisted in almost any direction (Figs. 33, 34, and 35). Rickets is one of the causes of knock-knees.

Because of the lax musculature, the rachitic infant tends to slump into a heap when an effort is made to sit up. Ultimately this leads to a considerable degree of kyphosis. The laxity of the muscles and ligaments of the feet predisposes to the development of "flatfeet." The lax musculature of the abdomen and of the intestines causes abdominal protuberance or "pot-belly" (Fig. 32). Intestinal atony results in constipation. Diastasis of the rectal muscles and umbilical herniae are common.

Dentition is delayed, but the temporary teeth are not necessarily abnormal in shape or structure, since these teeth are fairly well developed even though unerupted at the time of birth. Rickets during infancy is more likely to cause an alteration in the structure of the permanent teeth which are developing during the time that rickets is active (see Chapter XXXIII).

The rachitic child is fretful, irritable, sleeps poorly, and is restless during sleep. Excessive sweating about the head is common. The infant is pale and pasty in appearance. Tetany is not infrequently associated with rickets, and when present there are the added symptoms of neuromuscular irritability, carpopedal spasm, laryngismus stridulus, or definite convulsive seizures.

The mental development is sometimes retarded in cases of severe rickets, and this retardation may be sufficient to arouse the

suspicion of definite mental deficiency. Following the institution of proper treatment, the mental development may be expected to become entirely normal.

Prevention and Treatment

Rickets is, in most instances, a preventable and a curable condition. The greatest difficulties arise in the case of premature infants in whom both prevention and cure are difficult. The two most important means of preventing and curing rickets are the administration of diets containing sufficient vitamin D and exposure to sunlight or ultraviolet light. It is possible that something may be done in the way of preventing rickets by attention to the mother's diet and hygiene previous to delivery and during the lactation period. The mother's diet should contain liberal amounts of vitamin D in the form of butter, milk and eggs, and this may to advantage be supplemented by the administration of viosterol or cod liver oil. Irradiation of the mother's body, or portions of it, with sunlight or ultraviolet light is of value. The infant should, if possible, be breast-fed, but breast milk is not necessarily a complete prophylactic. Breast-fed infants are not likely to develop rickets if the diet of the mother is good and if she and the infant spend a considerable amount of time out of doors, but even then it is advisable to administer cod liver oil to the infant during the winter months. One-half teaspoonful of cod liver oil twice a day is usually sufficient for the breast-fed infant.

If it is necessary to feed the infant artificially, the diet should be constructed along the lines outlined in Chapters XIV, XV and XVI. The amount of whole milk given should be not less than 1.5 ounces per pound of expected body weight per day, and preferably 2 ounces. The proportion of total carbohydrate, including sugar and starch, should not exceed one-tenth of the amount of milk given. Extra vitamin D should be administered in all instances, preferably in the form of cod liver oil. The dosage of this, necessary to protect against rickets or to cure rickets once it has developed, will depend more upon the size and rate of growth of the infant than upon any other factor. It is indeed difficult to administer sufficient cod liver oil to prevent slight radiologic evidences of rickets in infants who are growing very rapidly. Average bottle-fed infants, growing at an average rate, should receive

from one-half to one and one-half teaspoonfuls of cod liver oil three times a day. A very rapidly growing infant may require as much as two teaspoonfuls three times a day.

Viosterol, 100-D, is a rich source of vitamin D; it does not, however, supply the equally necessary vitamin A; furthermore, viosterol is a potent substance, the use of which is not unattended with danger. If viosterol is used, the daily dose is from 10 to 15 drops. Another excellent source of vitamin D is egg yolk. This is not as rich in the vitamin as is cod liver oil but is easily administered. One yolk a day, mixed with the milk, provides sufficient extra vitamin to protect most average-weight infants against rickets, but not enough for rapidly growing infants, prematures or negroes.

Exposure of the body to sunshine "skyshine" or artificial sources of ultraviolet light is at least of as great value as the administration of vitamin D. There are some instances in which irradiation proves more effective than cod liver oil administration; this is especially true of premature infants. In resorting to any form of irradiation, the exposure should be sufficient to tan the skin gradually, never to cause burning.

The above means are the only really effective ones for the prevention or treatment of rickets. There is no good reason for the administration of extra lime salts except in the very rare cases of "low calcium rickets"—indeed, the addition of extra calcium salts to the diet may actually be detrimental. Nothing is to be gained by the administration of extra phosphate or of phosphorus in any other form. Glycerophosphates and hypophosphites are useless, and elementary phosphorus is essentially a poison and probably of no value in the treatment of rickets. (Elementary phosphorus seems to be of value in some other forms of bone disease.) Parathyroid extract (parathormone) is of value only in those cases of rickets associated with tetany or parathyroid deficiency.

Recovery from the secondary anemia associated with rickets is hastened by the feeding of green vegetables and iron salts.

CHAPTER XXX

TETANY

(Spasmophilia)

Tetany or spasmophilia is a condition characterized by neuromuscular hyperexcitability dependent upon a disturbance in mineral metabolism. The neuromuscular excitability leads to characteristic tonic spasms of the muscles of the extremities and of the larynx, resulting in carpopedal spasm or laryngismus stridulus, or in severe cases to generalized convulsions. The disorder may exist in all degrees of severity. In the latent form it is manifested only by increased excitability of the motor nerves to the galvanic current or to mechanical stimuli. Latent tetany may at any time become active as the result of infections or of dietetic errors. Tetany is the underlying cause in perhaps the majority of cases of infantile convulsions occurring after the age of four or five months.

Etiology

Tetany is, to some extent, a familial or constitutional disease. It is seen with especial frequency in families of the nervous type. The parents of infants with tetany not infrequently show certain latent manifestations of the condition, such as the facial phenomenon (Chvostek's sign). The condition is more frequent in the dark-skinned than in the light-skinned races.

Tetany is seen but rarely during the first three or four months of life and is not of frequent occurrence after two years of age. The majority of cases of active tetany occur during the late winter and spring months. This seasonal variation appears to be dependent upon the amount of sunlight to which the infant is exposed.

Diet plays the most important rôle in the etiology of tetany. The condition occurs almost exclusively in artificially fed babies, though it is sometimes observed in negro and in premature infants who have been entirely breast fed. It occurs in well-nourished as well as in under-nourished infants, and in the former is often associated with rickets. Tetany is not directly attributable to a

deficiency of vitamin D in the diet, but the administration of this vitamin does exert a curative effect. None of the other vitamins appear to be concerned. Tetany is especially likely to occur in infants who are receiving large amounts of sweet cow's milk together with relatively little carbohydrate and in those who suffer from a tendency to constipation. It is not frequently seen in infants receiving acid milk mixtures.

It has been suggested that infantile tetany is the result of parathyroid deficiency. When the parathyroid glands are extirpated or are the seat of lesions interfering with their function, all of the manifestations of tetany occur; furthermore, the symptoms of infantile tetany are relieved by the administration of parathyroid extract. On the other hand, no constant changes are demonstrable in the parathyroid glands of infants who have died of tetany.

Pathogenesis

The most important factor in the pathogenesis of tetany is an absolute or relative decrease in the amount of ionized calcium present in the blood. In ordinary cases of "idiopathic" infantile tetany, the calcium content of the blood serum is lowered. The normal calcium content is from 10 to 11 mg. per 100 c.c.; in cases of latent tetany the calcium content falls to 7 or 8 mg., and in active tetany, usually below 6 mg. The low calcium content of the serum is not referable to a deficiency of calcium in the diet; for the condition is most frequently seen in the case of infants fed on cow's milk, which has a much higher calcium content than human milk. The calcium is either poorly absorbed or is rapidly removed from the blood. The relatively large proportion of phosphate in cow's milk undoubtedly interferes to a considerable extent with calcium absorption, and furthermore the feeding of large amounts of cow's milk mixtures with the addition of relatively small amounts of fermentable carbohydrate results in a condition of alkalinity in the intestinal tract which is unfavorable for calcium absorption. Decreased intestinal absorption, however, is certainly not the only factor involved. Tetany sometimes develops during the stage of healing of rickets possibly because the circulating calcium in the serum is removed by deposition in the calcifying bone.

In infantile tetany, the phosphate content of the blood is either normal or slightly high, differing in this respect from rickets, in

which the phosphate content is low. The phosphorus in most cases of tetany is, however, not sufficiently high to explain the low calcium content, although a reciprocal relationship is known to exist. The injection of large amounts of phosphate or the retention of phosphate in the serum as the result of renal insufficiency is followed by a reduction in the calcium content of the serum and often by the appearance of the symptoms of tetany.

All of the symptoms of tetany may occur in individuals in whom the total calcium content of the serum is normal, but in whom the alkalinity of the serum is increased. Thus tetany is seen in infants who, as the result of vomiting, have developed alkalosis, and it is also seen following the therapeutic administration of alkalies. Pulmonary hyperventilation caused by voluntary deep breathing, crying or occurring as the result of fever brings about a removal of carbonic acid from the blood with a shift of the reaction toward the alkaline side; this is often associated with the appearance of all of the symptoms of tetany. The suggested explanation of the occurrence of tetany in the presence of alkalosis is that the ionization of calcium is decreased as the alkalinity of the medium is increased. The administration of acid or acid producing salts leads to a disappearance of the symptoms of tetany associated with alkalosis without at the same time bringing about any great change in the calcium content of the serum.

Symptoms

The characteristic symptoms of active tetany are carpopedal spasm, laryngismus stridulus and generalized convulsions.

Carpopedal spasm is a pathognomonic sign. This consists of a tonic spasm of the muscles of the hands and feet. The position of the hands is characteristic; the wrists are flexed and the thumbs strongly adducted toward the palms of the hands; the fingers are flexed at the metacarpo-phalangeal joints, the other joints being extended or but slightly flexed (Fig. 36). The feet are extended, the dorsum is sharply arched, and the toes are flexed. In addition an equinovarus position may be assumed. The tonic contractures of the hands and feet may be transitory or may continue for days at a time. The contractures are distinctly painful. In those cases in which carpopedal spasm has not occurred spontaneously, the spasm may often be brought about by a compression of the extremities with an elastic band (Trousseau's sign). This sign is

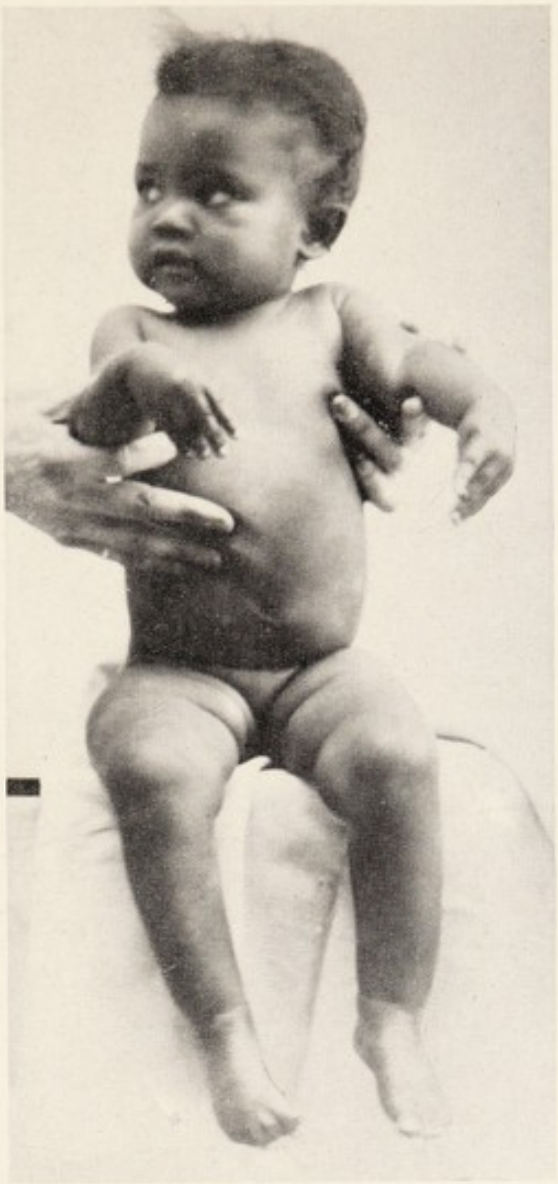


Fig. 36.—Tetany. Carpopedal spasm.

Fig. 37.—Tetany. Appearance during a convulsive seizure.

best elicited in the upper extremities. A flat rubber bandage or a blood pressure cuff is applied to the upper arm with sufficient pressure to obliterate the radial pulse for a few minutes. The hand gradually assumes the characteristic spasm.

Despite the marked carpopedal spasm often seen in the presence of tetany, there is no general muscular hypertonicity. The arms and legs are freely movable, except in the presence of convulsions; the knee jerks are not exaggerated and the Kernig sign is not present. There is no abdominal rigidity and no opisthotonus except during convulsions.

Laryngismus stridulus is observed in a fair proportion of cases of tetany. This is the result of a spasm of the laryngeal muscles. The condition may become evident only when the infant cries, during which time there is a high-pitched inspiratory crow. The laryngeal spasm may be sufficient to interfere with respiration to such an extent that deep cyanosis results.

The severest manifestations of tetany are generalized convulsions. These may occur at frequent intervals. The convulsive movements are at first tonic and toward the close of the seizures, clonic. The hands are tightly clenched or assume the position of carpal spasm. The head is thrown back, and the corners of the mouth are drawn down (Fig. 37). Death may occur during the convulsions.

Not all patients with tetany show the marked symptoms described above. In some, the condition is latent and may be recognized only by evidences of increased electrical excitability of the motor nerves (Erb's sign). In order to elicit this sign, a galvanic battery with a milliammeter is required. The positive (or anodal) terminal is applied over the abdomen and the negative (or cathodal) terminal over the peroneal nerve near the head of the fibula. A contraction of the muscles supplied by the nerve when the circuit is opened, following the passage of a current of less than 5 milliamperes, is the characteristic finding in tetany. The reaction on closing the current is of less significance, as are the reactions occurring when the anodal terminal is applied over the nerve. Considerable experience is required in determining the electrical reactions and in interpreting the results.

A more easily demonstrable sign of latent tetany is the Chvostek sign, or the facial phenomenon. This sign is elicited by tapping lightly with a small percussion hammer over the facial

nerve, just anterior to the ear. A contraction of the facial muscles is interpreted as presumptive evidence of tetany.

In patients with latent tetany the manifestations may all become active in the presence of infection. Many of the convulsions occurring at the onset of febrile affections during infancy are in reality due to tetany which may have been latent and unrecognized.

Tetany itself is not accompanied by fever.

Treatment and Prevention

The two most effective means of preventing tetany are breast feeding and exposure to sunlight or ultraviolet light. The administration of extra vitamin D in the form of cod liver oil or viosterol is also a valuable prophylactic measure. In the case of artificially fed infants, the use of acid milk, and especially of hydrochloric acid milk (see Chapter XVI), is preferable to sweet milk. The diet should not contain an excessive amount of milk in proportion to other constituents; sugars or cereals should be given in an amount approximating one-tenth of the amount of milk given, and vegetables and fruit juices should be included in the diet. Suitable means should be taken to remedy any tendency to constipation, as this predisposes to tetany.

Once the condition of tetany has developed, the most effective means of treatment are ultraviolet irradiation and the administration of calcium salts, acids and viosterol. Irradiation with ultraviolet light results in a rise in the calcium content of the serum and coincident with this a gradual cessation of the symptoms of tetany. Irradiation therapy is more effective if calcium salts are administered at the same time. Calcium chloride is to be preferred to the organic calcium salts, such as the lactate or acetate, as it is more readily absorbed and in addition is metabolized in such a way as to liberate hydrochloric acid. The dosage of calcium chloride is from 10 to 15 grains (0.7 to 1.0 gm.) four to six times daily. This is best administered in milk, as it is then less likely to irritate the stomach than when given alone. Calcium chloride possesses the disadvantage of being bitter and may make the milk distasteful to the infant or cause vomiting. In cases in which calcium chloride is not well taken, the lactate or acetate may be substituted, or the newer preparation, calcium gluconate (Sandoz). This latter preparation has very little taste and is not

irritating. In using the organic calcium salts, it is necessary to give about twice the dose in order to produce the same therapeutic effects as when the chloride is used. Calcium salts may be given by injection; the chloride is very irritating and cannot be given subcutaneously or intramuscularly but may be given intravenously in the form of a 5 per cent solution. The amount of this injected should not exceed 0.5 c.c. per kilogram of body weight. Calcium gluconate may be given subcutaneously or intramuscularly in the form of a 10 per cent solution. Such a solution is now available in the form of ampoules. The effects of the administration of calcium salts are not immediate. The blood calcium content slowly rises over a period of days; it may not fully reach the normal level, but a gradual disappearance of the symptoms of tetany occurs as the normal is approached. If the administration of calcium is discontinued, the blood calcium falls again and the symptoms return; consequently calcium administration must be continued for long periods of time, unless other means are taken to remedy the condition. The calcium administration may usually be discontinued during the summer months when the manifestations of tetany tend to become latent.

Both cod liver oil and viosterol are of specific value in the treatment of tetany; more immediate results are obtained with viosterol. This may be given in doses of from 10 to 20 drops a day until the active manifestations have disappeared, after which the dosage may be diminished. In this connection, it should be noted that the administration of viosterol to infants with active rickets is sometimes followed by a temporary fall instead of a rise in blood calcium with the coincident development of the manifestations of tetany. This appears to be due to a fixation of circulating calcium in the bones.

Acid administration is an effective means of relieving the manifestations of tetany, especially in those cases in which the tetany is the result of alkalosis. Hydrochloric acid may be added to the milk in the proportion of 1 dram (4 c.c.) of the concentrated acid to the pint (this must be added very slowly), or the milk may be mixed with from ⅕ to ⅓ of its volume of tenth normal hydrochloric acid. The acid administration should be continued for a number of weeks and then the amount of acid gradually decreased or substituted by lactic acid. The administration of acid does not necessarily increase the calcium of the blood. In place of acid, certain salts may be given which are metabolized with the libera-

tion of hydrochloric acid. The two most effective salts are calcium chloride and ammonium chloride. Calcium chloride may be given in the doses already recommended, and this provides both acid and calcium. According to Gamble, 1 gram (15 grains) of calcium chloride is the approximate equivalent of 75 c.c. of N/10 hydrochloric acid. Ammonium chloride may be given in the same doses as calcium chloride and has about the same acid producing effect. When an immediate acid effect is desired, as in the presence of very active manifestations of tetany, inhalation of carbon dioxide may be employed. The breathing of a few whiffs of a mixture of 30 per cent carbon dioxide in oxygen is often followed by a prompt but temporary disappearance of the acute symptoms; following this, a mixture of 5 per cent carbon dioxide in oxygen may be inhaled almost continuously until the other therapeutic measures adopted have had a chance to act.

Collip has prepared an extract of parathyroid gland (parathormone) which when injected causes a rise in blood calcium content in both normal individuals and in those suffering from tetany. This extract may be employed as a temporary and emergency measure, but its continued use is inadvisable, as it has a cumulative effect, and overdoses result in serious symptoms. The initial dose in an infant should not exceed 5 units. If no untoward symptoms result, a dosage up to 10 or 15 units may be given; repeated small doses are, however, preferable to a single large one.

Magnesium sulphate administered subcutaneously exerts a definite sedative effect in cases of tetany. This form of medication is especially indicated in those cases in which convulsions occur. For subcutaneous or intramuscular injection a 10 per cent solution is employed. This is prepared from the anhydrous magnesium sulphate (twice the amount of the crystalline epsom salts must be used). The maximum daily dosage of the 10 per cent solution should not exceed 2 c.c. per kilogram of body weight, and it is advisable to give not more than 1 c.c. per kilogram at a single dose. The effect of magnesium sulphate in controlling the convulsive manifestations is fairly prompt and may last for a number of days. This form of treatment is purely symptomatic and should not replace the specific measures described above.

When the convulsions of tetany are severe, it may become necessary to resort to the use of chloroform inhalations or to the administration of morphine or chloral as in the treatment of convulsions due to other causes.

CHAPTER XXXI

SCURVY

(Scorbutus)

Scurvy is a nutritional disorder resulting from a deficiency of vitamin C in the diet. It is characterized by failure of nutrition and by a tendency to hemorrhages throughout the body and especially in the bones. The condition is a relatively rare one at the present day.

Etiology

Scurvy may occur at any age and in any individual deprived of vitamin C, even though the diet and hygienic conditions may be excellent otherwise. Scurvy is seen more frequently in infants than in older individuals because of the necessarily restricted character of the diet. The period of the greatest incidence of scurvy is between the seventh and tenth months of life. The disease is but rarely seen before the fifth month. Scurvy may occur in breast-fed infants when the mother's diet has been deficient in vitamin C; it is, however, much more frequent in those who are artificially fed, because of the fact that the food is usually subjected to heat treatment which destroys a portion or all of the antiscorbutic vitamin C. Raw cow's milk contains a variable amount of vitamin C which may not be sufficient to protect the artificially fed infant against scurvy unless other vitamin C containing foods are added to the diet. Any form of heat treatment of milk, whether pasteurization, boiling, drying or evaporating, results in some destruction of the vitamin. Alkalinization of milk, even in the absence of heat treatment, also destroys the vitamin; hence scurvy is especially likely to be seen in those infants fed on formulas prepared from the proprietary infant foods containing added alkali.

The effects of feeding diets deficient in the antiscorbutic vitamin are not immediately manifest. Infants at birth appear to possess a store of antiscorbutic vitamin, which is not exhausted for a number of months, and it is probably for this reason that the symptoms of scurvy are not usually seen in infants before the age of four or five months, even though they are fed on deficient

diets. Infants vary in their susceptibility to scurvy; of a group of infants of the same age, all of whom are fed on sterilized milk mixtures without the addition of vitamin C containing foods, some may develop marked evidences of scurvy and others only the slightest evidences. Infants with rickets appear to be especially susceptible to scurvy, and this is also true of infants whose nutrition in general is poor. There is no evidence that any infectious or toxic agent is a causative factor in scurvy.

Pathology

In scurvy the cells of most of the organs in the body are probably affected to some extent; the most marked changes, however, are observed in the endothelial linings of the capillaries. This capillary damage leads to stasis and to hemorrhages. The most marked effects are seen in the osseous system. The periosteum becomes swollen, and hemorrhages occur between the periosteum and bone. Hemorrhages and circulatory stasis at the epiphyseal lines frequently result in epiphyseal separation and interference with normal bone formation. The marrow cavities are congested and hemorrhagic. Some of the hemorrhage may find its way into the joints or the periarticular tissues. Ultimately the hemorrhagic extravasations under the periosteum and about the epiphyses become organized and calcified. There is no change in the coagulation time of the blood, and there is no deficiency in blood platelets or calcium. Hemorrhages also occur in the subcutaneous tissues, in the gums, into the intestinal tract and at times into the pericardial and pleural cavities, and over the surface of the brain.

Symptoms

The symptoms of scurvy develop gradually and insidiously. An infant who previously may have been thriving, begins to take the feedings poorly. Gain in weight ceases, or there may be an actual loss. The infant loses his color, becomes progressively paler, is fretful, peevish and irritable, and resents handling. There is little or no fever. One of the early signs of scurvy, as pointed out by Alfred Hess, is an increase in the pulse rate to 150 or more and a coincident increase in the respiratory rate, but not to a proportionate degree.

The first symptom which usually arouses the definite suspicion of scurvy is tenderness of the extremities, especially of the lower

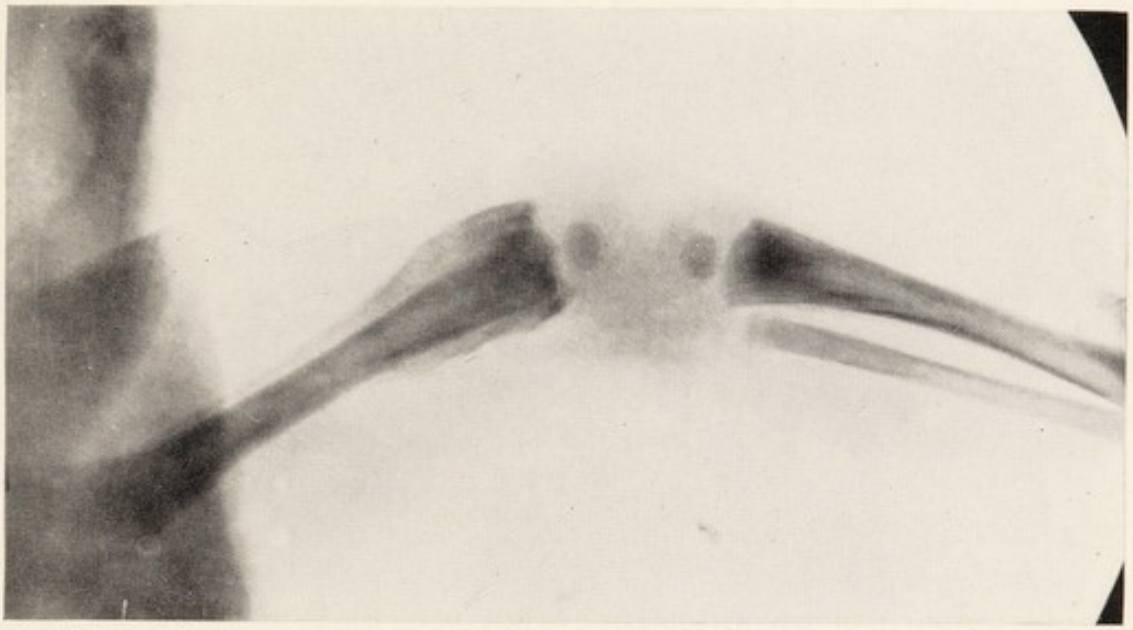


Fig. 38.—Scurvy. Subperiosteal hemorrhage.

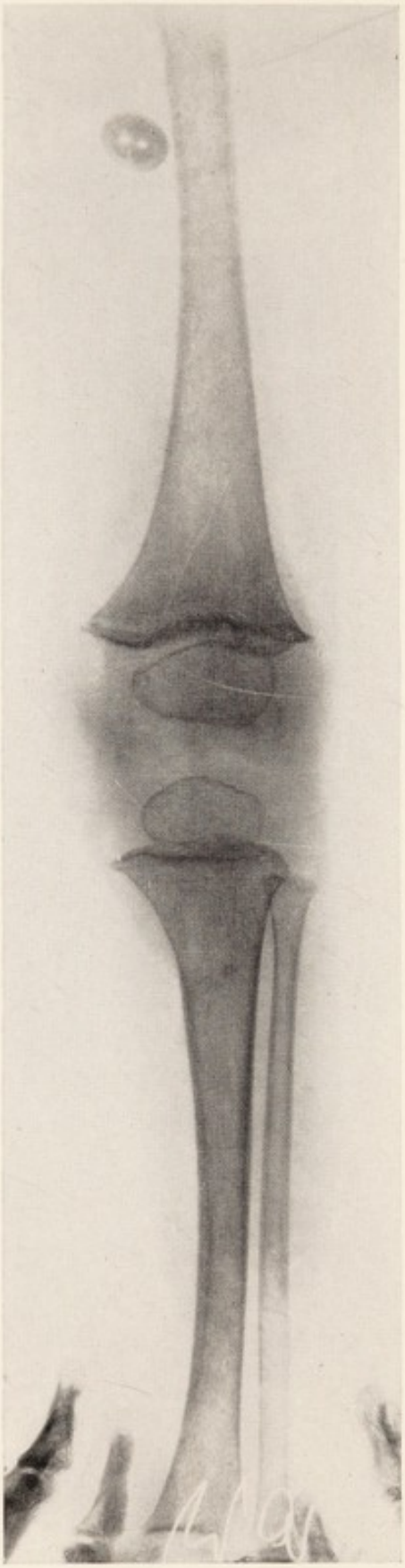


Fig. 39.—Scurvy. Early radiological signs. Note ground-glass appearance of shafts, narrow dense ring around epiphyseal centers, broadened epiphyseal lines with areas of decreased density just beyond, spurs at outer edges of epiphyses.

extremities. The child cries out with pain when the diaper is changed or when the legs are moved. A slight degree of swelling over the tender extremities may be present. Coincident with the appearance of tenderness, a tendency to hemorrhages is noted. The skin bruises easily, even slight trauma being followed by more or less extensive ecchymosis. Hemorrhage in the periorbital tissues occasionally occurs, leading to a typical "black eye." The gums are congested, especially along the edges. If the teeth have erupted, or if they are just under the surface of the gums, hemorrhage may occur into the tissues of the gums, making them swollen and purple. A frequent location of hemorrhages is under the mucous membrane covering the hard palate.

Even in the early stages of scurvy, red blood cells are usually present in the urine and may be detected by microscopic examination. In those cases in which the symptoms are recognized as being indicative of scurvy, and suitable antiscorbutic treatment is instituted, all of the symptoms rapidly disappear, but if the condition is untreated, it progresses.

In a fully developed case of scurvy, the symptoms are unmistakable: the affected extremities are swollen over the areas of subperiosteal hemorrhages and about the joints; there is no voluntary motion, and extreme pain is occasioned by passive movements. Hemorrhages occur, not only under the skin, but often from the mucous membranes, the nose and intestinal tract. The mouth is often the seat of ulcerative stomatitis, and the breath is foul; the teeth are loosened and may be lost. Hemorrhage from the kidneys may be sufficiently great to lead to macroscopic hematuria, the urine being smoky. Albumin, casts and white blood cells are generally present in the urine when there is much hematuria. Some fever is almost invariably present in severe cases of scurvy; the temperature may be as high as 101° or 102° F. A severe degree of anemia occurs. We have observed a red blood cell count as low as 900,000 with a hemoglobin of 10 per cent. Emaciation is characteristic of cases of scurvy of long duration.

Much aid in the diagnosis of scurvy is to be obtained from x-ray pictures of the bones. The characteristic evidences in advanced cases are subperiosteal hemorrhages, epiphyseal separation and an increased density of the shadows in the epiphyseal lines. The subperiosteal hemorrhages, when fresh, may throw no shadow; but as the extravasations organize and calcify, the shadow becomes

apparent (Fig. 38). Not all cases of scurvy show these marked changes, but even in the early stages some bone changes are observable. Pelkan has described these early changes in detail. They consist of: (1) a ground-glass appearance of the shaft, due to the disappearance of the normal shadows of the trabeculae; (2) an increased density along the edges of the epiphyseal centers of ossification, giving the appearance of a circumscribing ring; (3) a very thin bone cortex, appearing often only as a narrow line; (4) an irregular broadened and calcified epiphyseal line with an area of decreased density immediately back of the line; (5) a small spur at the outer edge of the epiphyseal line and occasionally a dislocation of the entire epiphyses. These changes are strikingly shown in Fig. 39.

Treatment

The treatment of scurvy consists in the administration of adequate amounts of the antiscorbutic vitamin C. Although this vitamin is present in small quantities in raw milk, green vegetables, potatoes and meat, none of these articles of food can be depended upon to control a well-marked case of scurvy. Orange juice and tomato juice are the richest sources of the vitamin and should be used in all cases, irrespective of the character of the remainder of the diet. From one to four ounces of orange juice should be given daily. This may be mixed with the milk. The juice of raw tomatoes is about as effective as orange juice but is somewhat more likely to cause digestive disturbance. The juice of canned tomatoes is only a little less effective than the raw juice. The juice should, in all instances, be strained through cheesecloth.

Because of the coincident anemia, iron salts and green vegetables are of value in severe cases. Blood transfusion is, however, the most effective method of overcoming the anemia quickly. In severe cases with epiphyseal separation and bony deformity, orthopedic procedures are ultimately required. This is but rarely necessary. The administration of sufficient orange juice leads to prompt healing of the bony lesions and the complete disappearance of all symptoms of scurvy.

CHAPTER XXXII

ANHYDREMIA, ACIDOSIS, AND ALKALOSIS*

During the course of nutritional disturbances in infancy there frequently occurs a deficit of water or of certain of the mineral constituents of the body such as the fixed bases, sodium and potassium, or the acid anions such as chlorides. A deficit of any one of these essential constituents leads to more or less general disturbance of the ionic balance in the fluids and cells throughout the body and is accompanied by a variety of clinical symptoms, some of which may be severe and eventuate in death. The terms *dehydration, acidosis,* and *alkalosis* are used to designate conditions in which water deficit, fixed base deficit, and acid deficit respectively are the chief factors involved. Anhydremia necessarily results in some alteration in the fixed base content of the body, and both acidosis and alkalosis are usually associated with disturbance in the water balance. The restoration of a normal water balance of the body in cases of anhydremia is likely to be followed by the establishment of a normal base balance, and likewise suitable treatment of an existing acidosis is an important measure in the relief of a coexisting anhydremia.

Although closely interrelated the etiologic factors involved in bringing about the three conditions are very diverse. The symptomatology is in many particulars quite different.

Anhydremia

(Dehydration, Exsiccosis)

When for any reason the amount of water eliminated from the body becomes greater than the amount taken in, desiccation of the blood and tissues necessarily results. When any considerable degree of desiccation has occurred, characteristic symptoms become manifest which are directly attributable to the concentration of the blood (anhydremia).

One of the common causes of anhydremia is severe diarrhea, especially the type often designated by the term "cholera infantum" in which the stools are frequent, copious and watery. Under such conditions large amounts of water are lost by the bowel,

*This chapter was written in collaboration with A. F. Hartmann and includes material from an article by A. F. Hartmann entitled "Acidosis, Alkalosis, and Dehydration," published in Colorado Medicine, xxvi, 373, 1929.

and as vomiting is also frequent, the fluid intake is diminished. The result is a negative water balance and a desiccation of the blood. The fact that the administration of an excessive amount of food to infants suffering from diarrhea increases the severity of all of the symptoms has led many to suppose that the symptoms were due to the absorption of some poisonous substance from the food, and hence the clinical picture has been described as gastro-intestinal intoxication, acute autointoxication, food poisoning, or alimentary intoxication. We now recognize the fact that the severe symptoms are due to a loss of water with consequent desiccation of the blood and tissues, rather than to the absorption of toxic material. The term "anhydremia" is one more accurately descriptive of the condition.

Exactly the same clinical condition may occur in the absence of diarrhea and when the character of the food is not such as to cause a gastrointestinal disturbance. The condition is not uncommon in young infants who are nursing dry breasts. It is seen in idiots as a result of their refusal of food and water. Diminished fluid intake as a result of vomiting from any cause, whether directly referable to the gastrointestinal tract or not, for example, the central vomiting of intracranial lesions, may lead to the condition. Anhydremia more frequently occurs in the summer than during the winter. This is partly due to the fact that high external temperatures cause a greatly increased loss of water by way of the lungs and skin.

Anhydremia occurs during the course of certain infections, such as influenza and pneumonia. It is especially likely to occur in infants suffering from infections of the middle ear and mastoid due to certain strains of hemolytic streptococcus.

The *symptoms* of anhydremia are largely dependent upon a decrease in the volume of the blood and are, therefore, especially likely to occur in the case of athreptic infants in whom the volume of blood is already diminished. Some of the symptoms of the two conditions are identical.

One of the first indications of anhydremia is a loss of body weight, which is often extreme and rapid. A small infant may lose a half pound or a pound in a single day, the loss being chiefly water. Coincident with the loss in weight, the appearance of the patient changes greatly. The features become sharpened, the eyes sunken and often fixed in a far-away stare. Later the conjunctivae

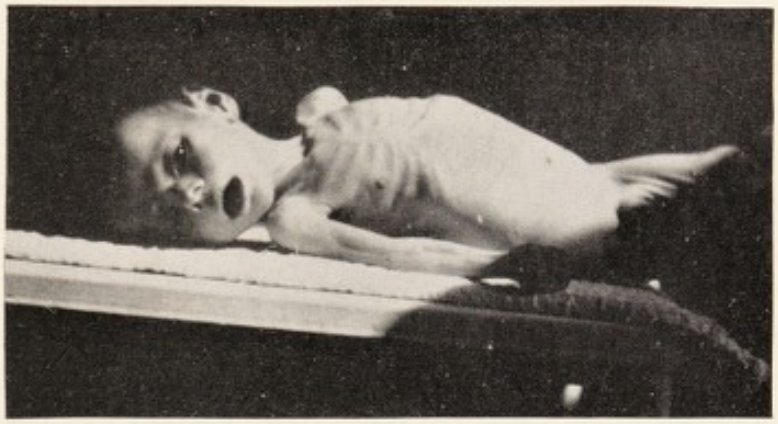


Fig. 40.—Anhydremia and acidosis. (''Alimentary intoxication.'')

lose their luster and are coated with a lusterless film. The eyes are likely to be turned up under the half-closed upper lids. The fontanelle is depressed; the skin has a peculiar pallor and often a characteristic grayish color like that of wet ashes. This color of the skin occurs because there is arteriolar constriction with piling up of red corpuscles in the capillaries. The capillary blood count is distinctly higher than that of the venous blood. The arteriolar constriction is one of the results of a diminished blood volume. The skin over the body is dry and may be picked up into folds which remain an appreciable interval before flattening out. The lips are dry, parched, and often of a peculiar cherry red color. The mouth is held partly open, the tongue is dry. (Fig.40.)

The pulse is small, sometimes almost imperceptible, often rapid and irregular. The volume flow of the blood is greatly diminished as the result of its concentration. The blood is thick, does not flow easily, and when centrifuged separates relatively little serum. The concentration of protein in the serum is invariably high and the water content low. Leucocytosis of a moderate degree is frequently present.

The urine is very scanty, highly concentrated, contains numerous granular casts and some albumin. Occasionally it reduces Fehling's solution. The scanty urine is the result of desiccation of the blood. The kidney becomes functionally inactive, although there are usually no demonstrable pathologic changes in the kidney at autopsy. This alteration in the functional capacity of the kidney results in the accumulation in the blood of products ordinarily eliminated by the urine.

The total nonprotein nitrogen of the blood increases and may be as high as in cases of uremic coma. Amounts above 200 mg. per 100 c.c. are not uncommon. The blood chlorides are increased in most severe cases and may be very high.

In most cases of anhydremia there occurs a reduction in the blood bicarbonate content, and this reduction may be extreme. We have not infrequently observed cases in which the volume percentage of CO_2 had fallen lower than 10, indicating a reduction of bicarbonate to one-fifth of the normal. The bicarbonate reduction is associated with the development of all of the symptoms and associated findings of acidosis. (For further discussion, see page 312.)

Some degree of fever is usually present; the temperature may be high in severe cases. In some instances the fever is due to infection; in others, however, it seems to be due to a disturbance of the heat-regulating mechanism as the result of an insufficient amount of water in the body. It may subside when sufficient fluid has been administered.

The mental condition of these patients is at first one of restlessness and excitement. Later, if the condition is untreated, the patient frequently lapses into a state of coma. Convulsions are not infrequent and often close the scene. Collapse symptoms may occur at any time. Vomiting often occurs as the result of anhydremia. It is seen in cases in which the anhydremia is the result merely of a diminished fluid intake. It occurs in animals fed exclusively on solid food. The vomiting often ceases when the water content of the body once more becomes normal.

The *prognosis* in anhydremia depends upon the underlying causes, the degree of desiccation and the previous nutritional condition of the infant. Anhydremia resulting from diarrhea or the combination of vomiting and diarrhea is more serious than that due to other causes. The prognosis is especially serious in the case of infants already athreptic. The presence of acidosis renders the prognosis exceedingly grave, not so much on account of the acidosis itself, for that can be cured, but because the presence of acidosis indicates that extreme desiccation has occurred with profound disturbance of the metabolism. When anhydremia is due simply to insufficient fluid intake, it usually clears promptly after a sufficient amount of water has been taken by mouth.

Treatment.—Much can be done to prevent the occurrence of anhydremia. It is important that those conditions which are likely to lead to water loss be treated promptly. Excessive loss of water from any cause must be checked and the intake of fluid increased. The water balance must remain positive.

Diarrhea should be treated in the manner previously outlined. When the stools are frequent, large and watery, one should not hesitate to use opium in reasonable doses. Water should be given freely and at frequent intervals and a record kept of the amount actually retained. An infant's weight should be followed carefully, and sudden or great loss should suggest the possibility of a developing anhydremia and serve as an indication for active treatment.

Once anhydremia has developed, the essential indication is to supply water. This should be given by mouth in as large amounts and at as frequent intervals as possible. If water is vomited, more should be immediately offered, as under these conditions it is sometimes retained. Very often the water deficit of the body is so great that it is impossible to administer a sufficient amount of water by mouth to supply the need. Even when vomiting does not occur, the water may be very poorly absorbed from the intestinal tract. For these reasons it frequently becomes necessary to administer water by other means. A small amount can be given intravenously in the form of the saline solutions or dextrose, but not a sufficient amount to accomplish any lasting effect. Larger amounts of fluid may be introduced subcutaneously than intravenously, but it is not always possible to introduce enough even by this means.

A very efficient means of introducing fluid is by way of the peritoneal cavity. Large amounts of fluid may be given in this way and be rapidly and completely absorbed. The technic of intraperitoneal injection is given in Chapter XXXIV. It is often possible to inject as much as 400 or 500 c.c. (1 pint) into a small infant. The injection may be repeated within five or six hours if the fluid previously administered has been absorbed. When sufficient fluid has been taken up by the blood and tissues to restore normal conditions, absorption from the peritoneal cavity becomes much slower. At about the same time the secretion of urine is resumed. The skin becomes elastic, and all of the symptoms tend to disappear. The weight becomes almost the same as before the development of the condition. The fluid usually used for intraperitoneal injection in these cases is Ringer's solution (NaCl 7.0 gm., KCl 0.1 gm., $CaCl_2$ 0.2 gm., water to 1000 c.c.). This solution has the advantage of supplying some of the mineral matter lost from the body. It, however, has the disadvantage of containing a higher concentration of NaCl than that of the blood, and increase of anhydremia, associated with oliguria and chloride retention, may cause a further increase in the blood chloride and a corresponding fall in blood bicarbonate.

A preferable solution to use for injection is Hartmann's solution (see page 322). This has the advantage of supplying potential base to replenish the depleted blood bicarbonate and at the same time does not increase the chloride content.

In all severe cases of anhydremia it is advisable to give dextrose intravenously in the form of a 10 or 15 per cent solution. (For technic, see page 351.) Although solutions of this strength are hypertonic, dextrose is so rapidly oxidized or stored that the injection of such a solution is almost equivalent to the injection of water. The injections may be repeated twice daily or more often. Such injections increase the blood volume and improve the circulation. The glucose supplies a certain amount of food and also acts as a diuretic. Blood transfusions are of great value in restoring a normal blood volume.

When it is possible to restore the normal water content of the body and establish diuresis, the acidosis accompanying anhydremia may be expected to disappear without further treatment. When, however, the acidosis is of sufficient severity to threaten life and diuresis cannot be promptly established, the administration of sodium bicarbonate is indicated. (For further discussion, see page 321.)

It is not always possible to restore the normal water content of the body. In many infants despite all therapeutic measures, the blood remains concentrated. In such instances a fatal outcome cannot be prevented. In the treatment of anhydremia it is important to realize that it is necessary not only to restore normal conditions, but to maintain them. An infant, after the administration of large amounts of fluid according to the methods described above may appear well on the road to recovery and yet a few hours later may once more lapse into a moribund condition. The treatment must be kept up until the causative factor is no longer operative. An infant suffering from an infection may develop anhydremia and die as the direct result of the anhydremia. On the other hand, even if the anhydremia is properly treated and cured, the infant may ultimately succumb to the infection.

Acidosis

In the course of normal metabolism, large quantities of acids are formed in and eliminated from the body. These acids are chiefly carbonic, lactic, phosphoric, and sulphuric. In order that the blood and other body fluids may preserve their normal reaction (that of faint alkalinity corresponding to pH 7.40) which is essential for health and life, the greater part of these acids, with the exception of carbonic acid, as soon as formed, and while re-

maining in the body, must be almost completely neutralized by base of the cells, intercellular fluids and blood plasma. Release of base for this purpose must not be attended by any appreciable change in acidity, and excretion of the excess acids must not remove from the body fixed base (i.e., Na, K, Ca, or Mg) in an amount sufficient to cause a negative base balance. In health this does not occur, and it is surprising with what little variation the body fluids maintain their normal hydrogen ion, electrolyte and total osmolar concentrations. The normal blood plasma, for example, is made to adhere rather strictly to the following composition:

	Average
BCl = 550-600 mg. % NaCl	= 100.0 M. eq. B^+
$BHCO_3$ = 200-250 mg. % $NaHCO_3$	= 25.0 " " "
H_2CO_3 = 40 mm. Hg.	= 3 vol. % CO_2
pH = 7.35-7.45	
Protein = 6.5-7.5 per cent	= 15.0 M. eq. B^+
Inorganic P = 3.5-5.5 mg. %	= 2.0 " " "
Inorganic S = 1.0-2.0 " "	= 1.0 " " "
B. lactate = 15.0-20.0 " "	= 2.0 " " "
Residual electrolyte (BR)	= 6.0 " " "
Total fixed base (Na + K + Ca + Mg)	= 151.0 " " "
Dextrose = 80-120 mg. %	
Urea = 10-20 mg. %	
H_2O = 93-94 gm. per 100 c.c.	
Total osmolar concentration = 320 osmolar mm.	

The H^+-ion concentration depends upon the relative concentrations of H_2CO_3 which dissociates into H^+ ions as follows:

$$H_2CO_3 \rightleftharpoons H^+ \overline{HCO_3}$$

and $BHCO_3$ which dissociates into $\overline{OH}$ ions:

$$BHCO_3 \rightleftharpoons B^+, \overline{HCO_3}$$

$$H_2O \rightleftharpoons \overline{OH}, H^+$$

$$H_2CO_3$$

Whenever the ratio $\frac{BHCO_3}{H_2CO_3}$ is about $\frac{20}{1}$ the H^+-ion concentration equals pH 7.40.

The "defensive" mechanisms capable of preventing changes in reaction are as outlined below:

(1) *Buffer Action:* the conversion of "strong" acids or alkalies (dissociating to a large extent into H^+ and $\overline{OH}$ ions) into "weak" ones (which do not dissociate much).

(a) $BHCO_3$: If relatively strong acids such as lactic, diacetic, or hydrochloric are allowed to react with a bicarbonate salt such as sodium bicarbonate the following reaction will take place:

$$Na^+, \overline{HCO_3} + H^+, \overline{Cl} \rightleftarrows Na^+, \overline{Cl} + H_2CO_3$$

As long as sodium bicarbonate remains in excess, no great acidity will develop because of the weakness of carbonic acid. In the normal body fluids $NaHCO_3$ is maintained at a concentration of approximately 2.0-2.5 gm. per liter (equivalent to 250-300 c.c. N/10 acid per liter).

(b) H_2CO_3: If strong alkali such as sodium hydroxide is allowed to react with an excess of carbonic acid, sodium bicarbonate, a much weaker alkali than sodium hydroxide, will be formed.

$$Na^+, \overline{OH} + H_2CO_3 \rightleftarrows Na^+, \overline{HCO_3} + H_2O$$

(c) Phosphate $\frac{B_2HPO_4}{BH_2PO_4}$: At the normal body fluid reaction pH 7.40 the ratio of dibasic to monobasic phosphate is 4:1. At a pH of 6.0 the ratio is 1:9. It is thus evident that a relatively small shift in hydrogen-ion concentration attends the almost complete conversion of the dibasic salt to the monobasic form, or vice versa, the results of neutralization with strong acid or alkali. The chemical reactions involved in these transformations are as follows:

$$(1)\ Na_2HPO_4 + HCl \rightleftarrows NaH_2PO_4 + NaCl$$
$$(2)\ NaH_2PO_4 + NaOH \rightleftarrows Na_2HPO_4 + H_2O$$

The buffer effect of inorganic phosphate in the blood plasma and intercellular fluids is not great, owing to its small concentration. It is of more significance within the cell where its concentration is considerably higher.

(d) Protein: Because of its structure, protein can combine with either acid or alkali, due to its amino (NH_2) and carboxyl (COOH) groups. At the normal body reaction, protein behaves as an acid

and binds base. Its value as a buffer in blood plasma or lymph, while appreciable, is not of great significance, because of its relatively small concentration. In the fixed tissue cell, however, as well as in the erythrocyte, it is of very great importance. Hemoglobin, while shifting in reaction from pH 7.40 to 7.00, releases a relatively large amount of base. Since the membrane limiting both the erythrocyte and fixed tissue cell is relatively impermeable to passage to or fro of the cations, base so released by protein can be of help as buffer to acid only after passage of freely permeating anions into the cell from the lymph or plasma. That this process occurs and is of great importance in the normal transfer of carbonic acid from the tissues to the lungs has been shown particularly by the work of Van Slyke.

(2) *Respiratory Activity:* By means of the buffer mechanisms cited above, *gross* changes in H^+-ion concentration are prevented. They do not prevent, however, large fluctuations in the $\frac{BHCO_3}{H_2CO_3}$ ratio, which are automatically attended by physiologically significant shifts in H^+-ion concentration. When this ratio, which is normally 20/1, decreases (acidosis) as a result of reduction in $BHCO_3$ with or without actual increase in H_2CO_3, a normal ratio may again be restored by hyperventilation which causes a more rapid loss of carbonic acid through the lungs. If, on the other hand, the ratio is increased (alkalosis) due to increase in $BHCO_3$, slow, shallow breathing tends to diminish loss of carbonic acid and therefore aids in maintaining a normal ratio.

Respiratory activity, therefore, may be looked upon as the *fine adjuster* of acidity.

(3) *Renal Activity:* It is obvious that the buffer and respiratory defenses would sooner or later be overtaxed if acid excess continued. Normal renal activity, however, aids immensely during periods of acidosis by providing a means of excretion of excess acid bound to relatively small amounts of fixed base, thus conserving buffer substance. On the other hand, during periods of alkali excess, the normal kidney is capable of excreting $NaHCO_3$ in five times the concentration of the normal plasma sodium bicarbonate.

(a) Regulation of Urinary pH (Gamble): The normal kidney is able to secrete urine as acid as pH 5.0 or as alkaline as pH 8.0. At the latter reaction there is no free acid present with the

exception of carbonic acid which remains constant at a concentration equal to that in the venous blood of the kidney (i.e., about 4-5 volumes per cent); phosphate is present as the dibasic salt, and $BHCO_3$ is present in a concentration equivalent to 15 gm. of $NaHCO_3$ per liter. At pH 5.0 considerable amounts of the weaker organic acids are unneutralized (e.g., 5 per cent of diacetic, 20 per cent of B-oxybutyric, etc.); phosphate is in the form of the monobasic salt and $BHCO_3$ is almost absent (0.015 gm. per liter).

(b) Substitution of Ammonia for Fixed Base: In addition to practicing fixed base economy, in times of need, by regulation of acidity, the normal kidney also has the ability to manufacture ammonia (probably from urea, a neutral substance) and substitute it to a large measure for the fixed bases bound to acids coming to the kidney for excretion.

$$Na^+, Cl^-, + NH_4^+ \longrightarrow NH_4Cl + Na^+$$

The conserved fixed base, in this instance the Na^+ ion, will remain in the blood, leaving the kidney largely as $NaHCO_3$.

In disease, factors may operate in such a manner as to overcome the defensive mechanisms of the body and to lead to serious or fatal changes in the composition of the body fluids, which we often speak of as acidosis, alkalosis, or dehydration. The immediate etiologic factors responsible for the production of the more common and important forms of acidosis are outlined in Table XVIII:

TABLE XVIII

ACIDOSIS

(Tendency toward or actual reduction of ratio—$\frac{BHCO_3}{H_2CO_3}$)

I. *Increase of H_2CO_3*: due to abnormal respiration or circulation. (Depressed breathing, high CO_2 content of inspired air, pulmonary atelectasis, venous stasis.)

II. *Decrease of $BHCO_3$*

A. Replacement of HCO_3 because of increase of other anions.

1. Lactate ion: due to its increased formation or insufficient removal (strenuous muscular exertion, convulsions, hypo-oxidation because of poor circulation due to anoxemia or anhydremia).
2. Cl: Due to overadministration of HCl, NH_4Cl, $CaCl_2$, or NaCl, or to insufficient urinary excretion.
3. HPO_4 } Due to overadministration or insufficient urinary excretion.
4. SO_4 }
5. Diacetate ion } Due to abnormal fatty acid: glucose oxidation.
6. Beta-oxybutyrate ion }
7. Salicylate ion: due to overadministration or insufficient urinary excretion.

B. Loss from body of $BHCO_3$

1. By way of intestinal tract (failure of reabsorption of alkaline digestive juices because of obstruction, fistula, or diarrhea).
2. By way of urinary tract (polyuria, kidney damage).

It should particularly be noted that the chemical changes leading either to acidosis are in turn dependent on such factors as vomiting, diarrhea, anhydremia, anoxemia, inadequate dextrose oxidation, and circulatory or renal insufficiency. When a number of such factors are simultaneously at work the resultant type of

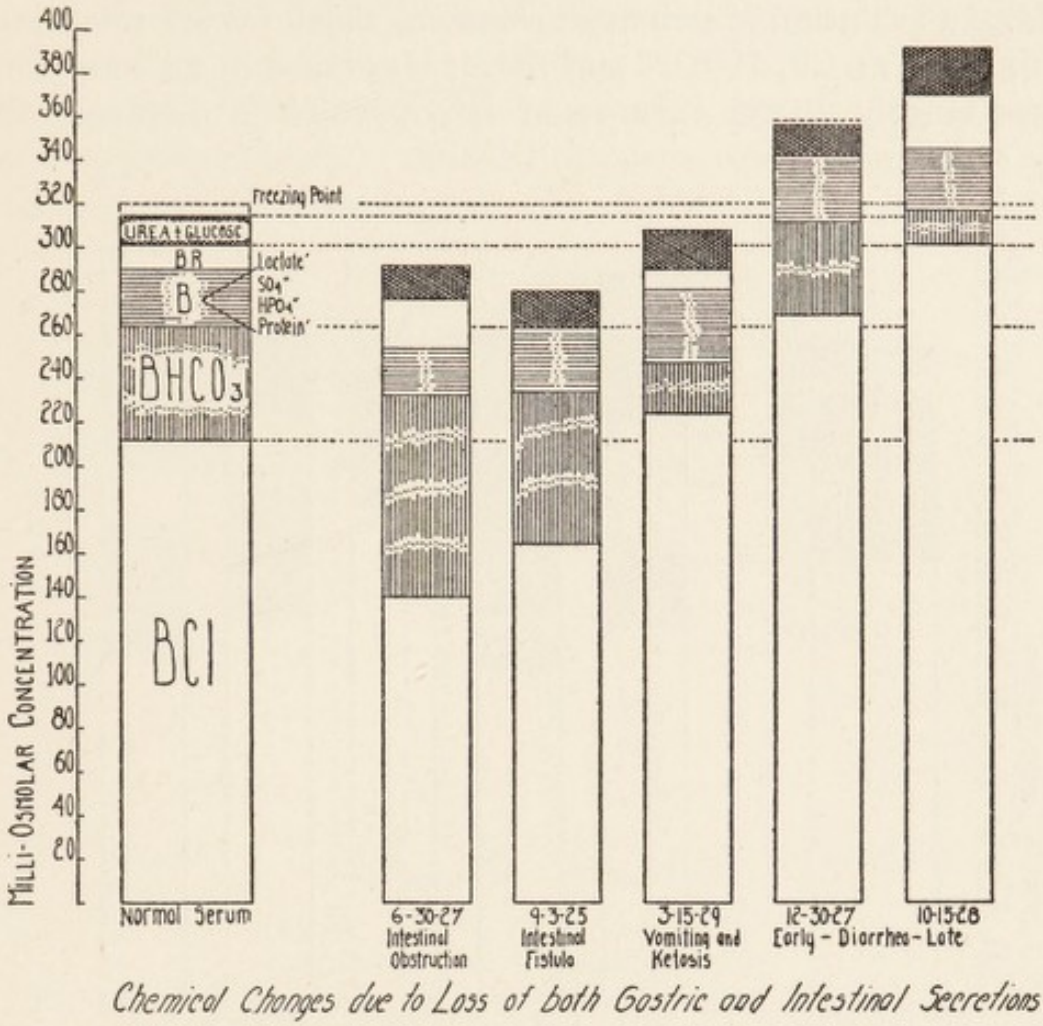


Fig. 41.—Types of chemical change (alkalosis or acidosis) due to loss of both gastric and intestinal secretions. (Hartmann.)

acidosis may be a very complex one (Figs. 41 and 42). Our present conceptions of the pathogenesis and treatment of the more common and important forms of acidosis are as follows:

Acidosis Associated With Severe Diarrhea of the Cholera Infantum Type

Severe watery diarrhea tends to produce changes in the direction of acidosis because of: (1) extensive loss of body water, (2) loss of body salts, and (3) starvation. The mechanism is as follows:

(1) *Loss of Water.*—Anhydremia, Dehydration: Loss of water from the blood leads to reduction in blood volume, which in turn leads to reduction in the volume of blood flowing through vital organs in a given time. Such circulatory insufficiency leads to anoxemia which favors the accumulation of lactic acid, and also leads to diminution of urinary excretion, which favors retention of such anions as Cl', HPO_4'' and SO_4''. Increase in all four anions occurs largely at the expense of $\overline{HCO_3}$ which is displaced from

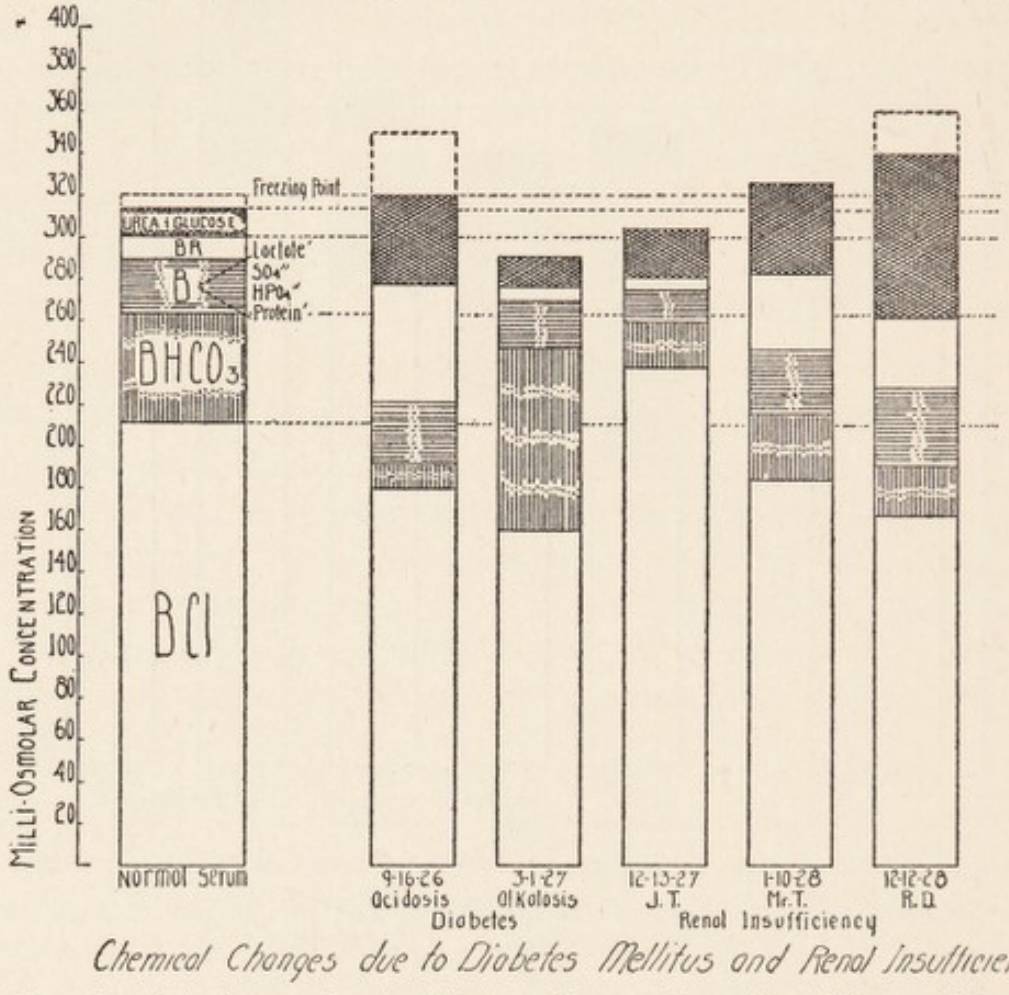
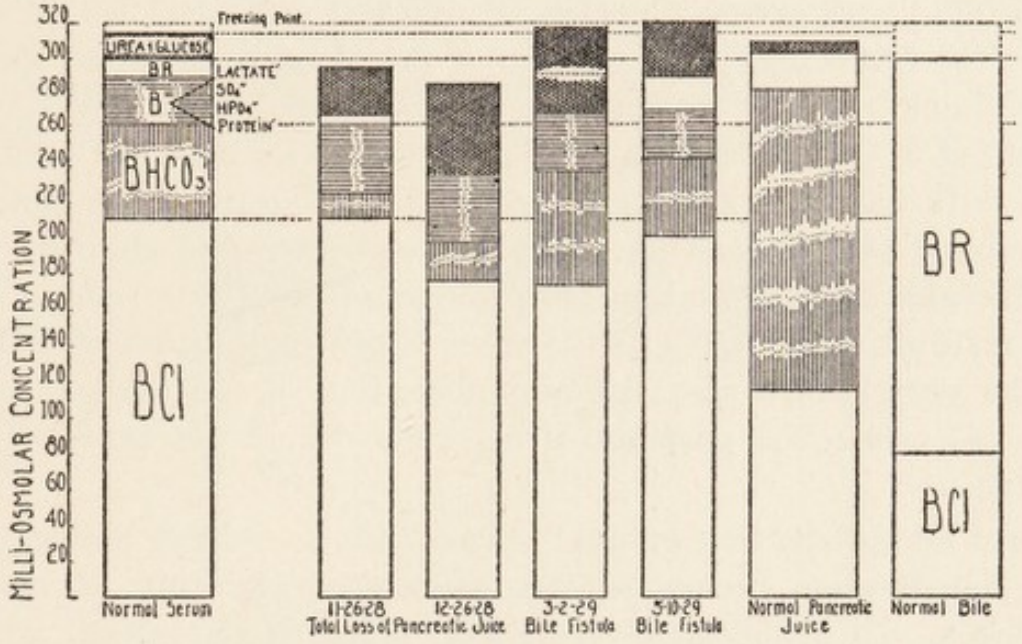


Fig. 42.—Chemical changes due to diabetes mellitus and renal insufficiency. (Hartmann.)

combination with base. As a result, the ratio $\frac{BHCO_3}{H_2CO_3}$ tends to diminish because of decrease in the numerator and increase in the denominator.

(2) *Loss of Body Salts.*—Associated with the failure of water reabsorption by the intestine there also occurs a failure of reabsorption of the salts of the digestive juices (Fig. 43). In the entire combined secretions of the normal gastrointestinal tract the excess of fixed base over fixed acid is apparently no greater than

that of the plasma or lymph. A similar ratio exists in the normal and diarrheal stool according to Hoag. The total amount of excess of fixed base over fixed acid is increased in the diarrheal stool, however. Metabolism, during periods of both starvation and adequate caloric intake in the form of the usual diet, is such as to leave an excess of fixed acid over fixed base bound for excretion. Renal activity, however, is normally such as to compensate by excreting more fixed acid than base. During periods of severe diarrhea accompanied by marked oliguria, acid excretion relative to base retention must diminish. In addition, during such periods



Chemical Changes due to loss of Pancreatic Juice and Bile by Intubation Fistula.

Fig. 43.—Type of acidosis due to loss of pancreatic juice. (Hartmann.)

evidence of kidney damage appears not only in the presence of considerable amounts of albumin and large numbers of casts in the urine, but also in the diminished urinary acidity and ammonia concentration relative to the concentration of fixed base. Furthermore, in the most severe forms of watery diarrhea, it would seem to us at least that a greater loss of alkaline pancreatic juice than acid gastric juice occurs, possibly due to the diminished secretion of the latter. Without such a qualitative change, however, loss of digestive juices coupled with normal or abnormal metabolism, restricted mineral intake, and renal insufficiency due to kidney damage or simply to oliguria would lead to reduction in $BHCO_3$.

(3) *Starvation.*—Aside from the failure to replenish minerals lost from the body in the stools and urine, with starvation there also occurs more rapid destruction of body tissue with increased liberation of such acids (relative to fixed base) as phosphoric and sulphuric. In addition, the organic ketone acids may accumulate if carbohydrate starvation becomes sufficiently pronounced. Ketosis, however, is much more commonly associated with bacillary dysentery than it is with diarrhea of the cholera infantum type due either to enteral or parenteral infection.

Based on such a conception of the pathogenesis of the acidosis associated with severe diarrhea, the logical treatment would seem to be the following: (1) dilution of the blood in order to restore its volume and flow so that the accumulated lactate ion may be disposed of in a normal fashion and release its base for combination with carbonic acid; (2) production of diuresis, so that accumulated anions such as phosphate, sulphate and chloride may be excreted in combination with ammonia, and thus release base for restoration of $BHCO_3$; (3) more rapid restoration of $BHCO_3$ in the very severe cases by administration of that salt as such; and (4) whenever possible direct removal of the cause of the diarrhea.

Oral administration of fluids often fails to relieve anhydremia and dehydration because of the associated vomiting as well as diarrhea. In the more severe cases, repeated parenteral administration is necessary. Tap or distilled water, even though sterile, cannot be used because if given subcutaneously or intraperitoneally it would be extremely irritating and if given intravenously it would cause hemolysis. Perhaps the most widely used method for parenteral administration of fluid is the intraperitoneal injection of normal or physiologic salt solution (sterile 0.85 per cent NaCl in distilled water). Such a solution is isotonic with the normal body fluids and therefore does not lead to hemolysis and is nonirritating even though given repeatedly and in large amounts into the peritoneal cavity, from which it is rapidly absorbed into the blood stream. When severe watery diarrhea persists, however, salt solution administration alone obviously will not be sufficient to relieve marked acidosis if anyhydremia and oliguria with renal insufficiency persist (Fig. 44). In such instances both anion and cation will be retained in the body, and the

plasma chloride concentration may reach enormous values (e.g. 900 mg. per cent NaCl). Such increase in salt concentration and osmotic pressure in the blood leads to further loss of plasma $BHCO_3$. The urine, for example, becomes more alkaline. In addition, it is probable that increased secretion of pancreatic juice richer in $BHCO_3$ also occurs. Isotonic dextrose (6 per cent) solution possesses some advantages over physiologic saline. The solute (dextrose) can be stored as glycogen or oxidized to furnish energy and relieve ketosis, if present. It does not tend, therefore, to increase permanently the osmotic pressure and cause further excretion of $BHCO_3$. A disadvantage is its occasional tendency to cause abdominal distention when injected intraperitoneally. This disadvantage may be overcome if the glucose is sterilized in the dry state before it is dissolved in the sterile distilled water.

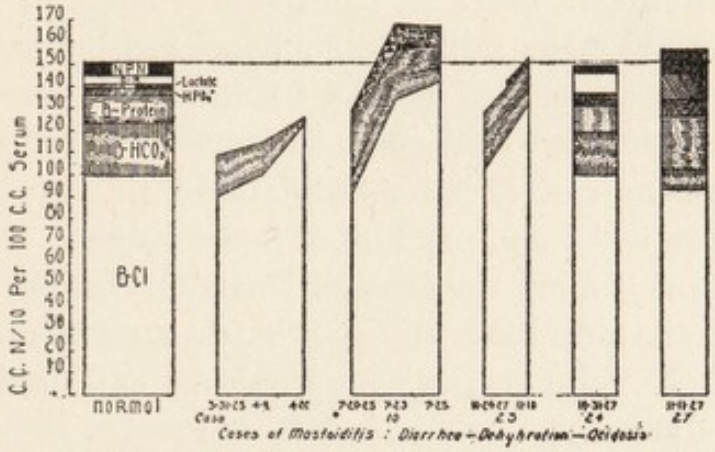


Fig. 44.—Acidosis and dehydration unrelieved by administration of Ringer's solution. (Hartmann.)

The combined use of physiologic salt and dextrose solution is often more effective in producing diuresis and thus securing the effect of renal activity than is the administration of either alone. When used in conjunction with each other, it is customary first to fill the abdomen with the saline solution and then to inject hypertonic (10 to 20 per cent) dextrose solution intravenously, in a dosage of 20 to 30 c.c. per kilo of body weight.

In the event, however, that sufficient $BHCO_3$ is not restored by the maximal dilution of the blood and increase in renal activity obtainable by such methods, alkali may have to be introduced more directly into the body. Administration by mouth is unreliable because of the uncertainty of absorption in the presence of vomiting and diarrhea. Parenteral administration requires consider-

able care both in the preparation of the solution and in dosage. For intravenous injection, a solution may be made most simply as follows:

Weigh out on a clean filter paper, taken from the inside of a package, 5 gm. of $NaHCO_3$ removed from a clean package by means of a metal spatula previously flamed, and dissolve in 100 c.c. previously cooled, sterile distilled water. Such a solution, while not absolutely sterile, can safely be given intravenously. For subcutaneous or intraperitoneal administration, however, the solution not only must be sterile, but also must be approximately isotonic and adjusted to the correct reaction, or else it will be too irritating. Since boiling decomposes the bicarbonate salt into the much more caustic carbonate, the sterilization must be accomplished by Berkefeld filtration. A satisfactory method of preparation is as follows: make 1½ per cent (isotonic) $NaHCO_3$ in distilled water, add a few milligrams phenol red indicator, sterilize by Berkefeld filtration, and before using bubble carbon dioxide through the solution until the color changes from red to orange.

The usual dosage of alkali administered in any of the ways mentioned above is 0.5 gm. per kilo of body weight. This is equivalent to about one-fourth the normal $NaHCO_3$ content of the body fluids. After the administration of such an amount a plasma CO_2 content of 15 volumes per cent would be expected to increase to about 30 volumes per cent. A larger dosage, should diarrhea cease simultaneously, might precipitate serious or fatal alkalosis by increasing the ratio $BHCO_3/H_2CO_3$ too much (i.e. $BHCO_3$ is doubled or tripled almost instantaneously while H_2CO_3 can be increased only by depressed breathing—a much slower process). On the other hand, should profuse diarrhea continue, further administration of alkali would be required.

Despite persistent diarrhea, by the continued judicious combined administration of saline, dextrose and alkali, it is usually possible to keep the acid-base and water balances more or less normal (Fig. 45). It must be admitted, however, that sometimes this is very difficult to accomplish without frequent chemical examinations of the blood.

Results equally good, if not better, have been obtained without the necessity of blood chemical examinations, through the use of a single "combined solution" devised by Dr. A. F. Hartmann, of

this clinic, and designed to supply water, BCl, $BHCO_3$ and anti-ketogenic effect, as needed in the restoration of lost body fluids.

Hartmann's solution is prepared as follows:*

Into a liter volumetric flask, measure 60 c.c. C.P. lactic acid (e.g. Mallinckrodt C.P. lactic acid 85 per cent).

Add a small amount of dry phenol red indicator (sufficient to make color change easily perceptible) and

Neutralize with strong carbonate free NaOH (e.g. saturated NaOH from which sodium carbonate has settled out on standing).

Then add 150 grams of NaCl

10 grams of KCl and

5 grams of $CaCl_2.2H_2O$

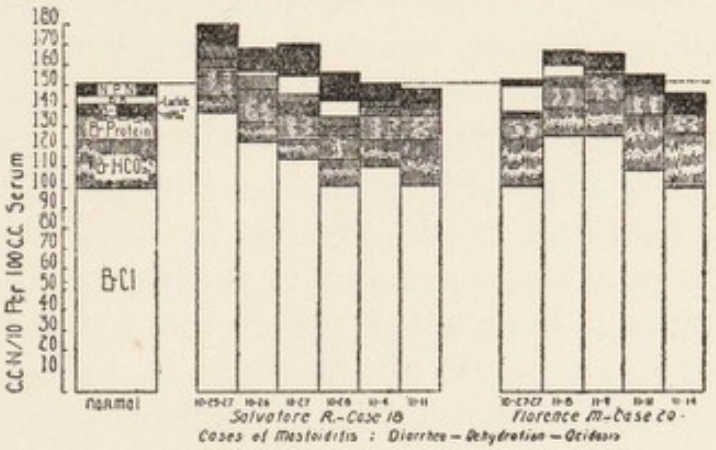


Fig. 45.—Relief of acidosis and dehydration by combined administration of sodium bicarbonate, dextrose and Ringer's solution. (Hartmann.)

Make to mark with freshly distilled water. Boil for thirty minutes to hydrolyze the lactic acid anhydride present; reneutralizing with strong NaOH as often as the solution becomes acid. Bring back to original volume with distilled water, filter and place in test tubes, autoclave at 15 lb. for thirty minutes and seal from air by drawing out the test tube. Rubber stoppered vials may be used; if so, the air should be aspirated with a fine needle and syringe before sterilizing, or else the stoppers will be blown out on heating.

Before using, the solution is diluted 25 times (e.g. 5 c.c. to 125, 10 c.c. to 250, etc.) with fresh sterile distilled water. It may then be given intravenously, subcutaneously or intraperitoneally.

*The solution may be obtained in sterile ampules from Eli Lilly and Company, Indianapolis.

When suitably diluted the composition of the solution corresponds to:

Na lactate	= 25	m. eq.	B^+
NaCl	= 100	"	"	"
KCl	= 5	"	"	"
$CaCl_2$	= 2.5	"	"	"

This solution supplies the chlorine ion if needed; sodium is balanced by potassium and calcium; the sodium lactate is converted at a relatively slow but still sufficiently effective rate into $NaHCO_3$ which may be retained if needed or excreted if not; and because of its hypotonicity, it tends to dilute the body fluids and promote diuresis when given in sufficient quantity. It should be administered subcutaneously, intraperitoneally or intravenously in amounts sufficient to maintain elasticity of the skin. If marked circulatory failure persists, however, sodium lactate may not be converted rapidly enough into $NaHCO_3$, and the latter will have to be given in addition for more immediate effect.

It must be borne in mind that the above measures are expected only to relieve dehydration and acidosis (or alkalosis, as we shall see later) and are not expected to stop diarrhea. They are often life-saving measures, however, during the period of severe water and mineral loss. Removal of the cause of the diarrhea is of course the ultimate aim, and includes treatment or removal of foci of infection and proper feeding. Blood transfusion constitutes one of the most effective means of aiding in the establishment of immunity to infection and is particularly valuable in the athreptic infants with diminished plasma volumes due to diminished plasma protein. (See Chapter XIX.)

Diabetic Acidosis

From detailed blood chemical studies the following conception of the pathogenesis of diabetic acidosis has been gained:

(1) *Rôle of Dehydration.*—As the ability to oxidize glucose diminishes, hyperglycemia develops, which is followed by glycosuria. Excretion of large amounts of sugar requires a large urine volume. For a while increased water intake compensates for polyuria but sooner or later dehydration results. Occasionally (particularly when coma intervenes) a decided decrease in fluid intake contributes to such dehydration, but frequently dehydration occurs despite a continued large water intake. Such dehydration

seems referable chiefly to loss of electrolyte from the body. To begin with, the mineral content of the cells probably tends to be minimal because of the failure of the cells to maintain their normal supply of glycogen. Also the continued polyuria in itself seems to lead to an appreciable loss of BCl and $BHCO_3$. Continued excretion of diacetic and B-oxybutyric acids leads to loss of fixed base from the body. Diminution of extracellular water (lymph and plasma water) occurs in favor of intracellular water when acidity increases because of a shift of anions with water in accordance with the principle of the Donnon equilibrium. If vomiting becomes marked, to reduction of fluid and mineral intake will be added loss of fluid and mineral in the form of gastric (and intestinal) secretions. As a result of such factors, dehydration and particularly anhydremia may become extreme and approach in magnitude the desiccation seen in infants with cholera infantum. In turn, circulatory and renal insufficiency result and lead to $BHCO_3$ reduction as discussed previously.

(2) *Rôle of Ketosis.*—Incomplete oxidation of fat leads to ketosis frequently with considerable accumulation of diacetic and B-oxybutyric acids in the body fluids. As mentioned in the beginning, as long as such relatively strong acids are in the body, they must be fully neutralized by base. Base for this purpose is at first entirely supplied by $BHCO_3$, which is consequently reduced. When reduction of $BHCO_3$ is not followed by proportional reduction of H_2CO_3 (i.e. when acidity actually increases and "uncompensated" acidosis is present), automatically phosphate and protein begin also to yield base. Renal activity tends to conserve such base by excreting a large part of the anions bound either to the H^+ or NH_4^+ ions. Such a defense, however, even in the presence of a normal urine volume and dietary intake is not perfect, and eventually permits extensive loss of electrolyte from the body. BCl and $BHCO_3$ suffer the greatest diminution.

Based on such a conception of the pathogenesis of diabetic acidosis, the logical treatment should include the following:

(1) Very rapid or immediate relief of increased acidity in those cases in which acidosis is extreme and death is feared.
(2) Complete restoration to normal of altered electrolyte, water and total osmolar concentrations of the body fluids, which includes

(a) addition of electrolyte (particularly BCl and $BHCO_3$) and water
(b) reduction of glucose
(c) abolition of ketosis.

(1) *Restoration of pH and $BHCO_3$.*—Although sufficient insulin administration with resultant glucose oxidation leads eventually to increase in the $BHCO_3$ content (and consequently pH) of the body fluids (Fig. 46), such relief of acidosis may be too slow to prevent death in the most severe cases; in the presence of very marked reduction in plasma $BHCO_3$ (e.g. to 15 vols. per cent or

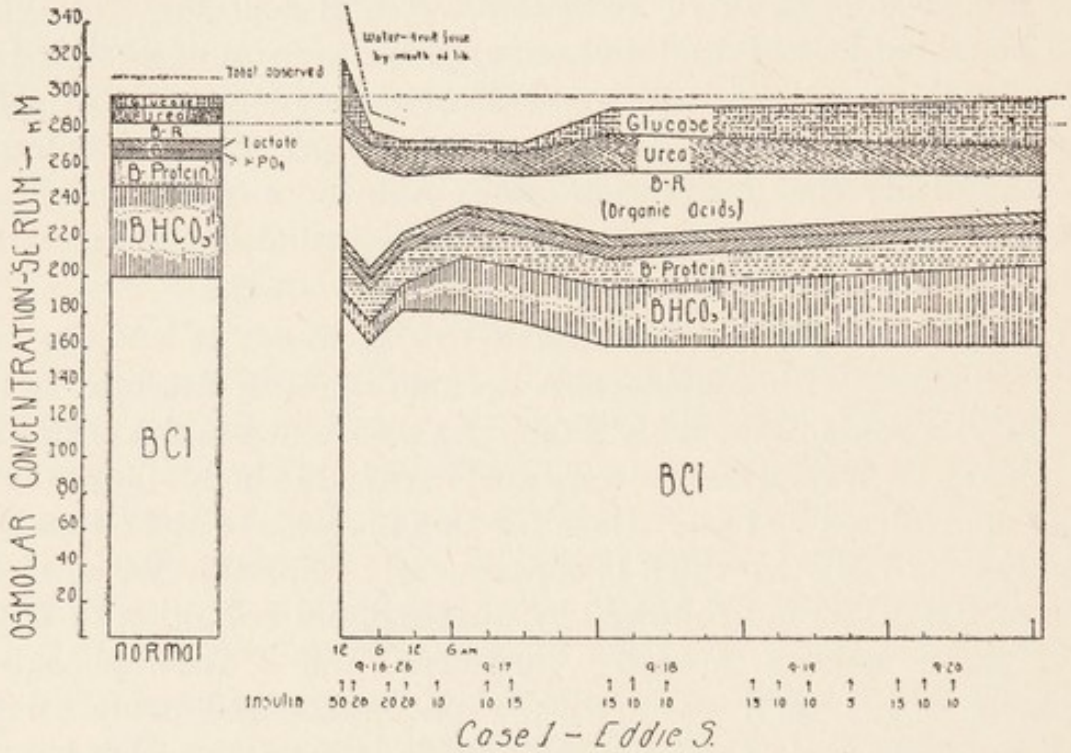


Fig. 46.—Slow recovery from diabetic acidosis because of failure to give $NaHCO_3$ or its equivalent. (Hartmann.)

less), particularly when marked insufficiency of circulation and renal activity are present, administration of $NaHCO_3$ is indicated. A safe yet effective dosage is 0.5 gm. per kilo of body weight (roughly one-fourth the normal entire body content) which is most easily given intravenously in 5 per cent solution prepared as outlined above. In contrast to "diarrheal acidosis" alkali need not be repeated in diabetic acidosis if the other measures indicated are properly carried out. (Fig. 47.)

(2) *Complete Chemical Restoration of Body Fluids.*—Electrolyte and water addition is best accomplished by subcutaneous or intraperitoneal administration of Ringer's solution, which fur-

nishes the Cl ion together with Na, K and Ca in proper proportions. Such fluid is given in an amount sufficient to restore the lost turgor of the skin. Ketosis is abolished by the repeated administration of insulin. The first administration should be a combined intravenous and subcutaneous injection. The customary dosage of each is 1-2 units per kilo of body weight. Subcutaneous insulin administration should then be repeated every four hours (dosage equals 1 unit per kilo of body weight) until ketosis dis-

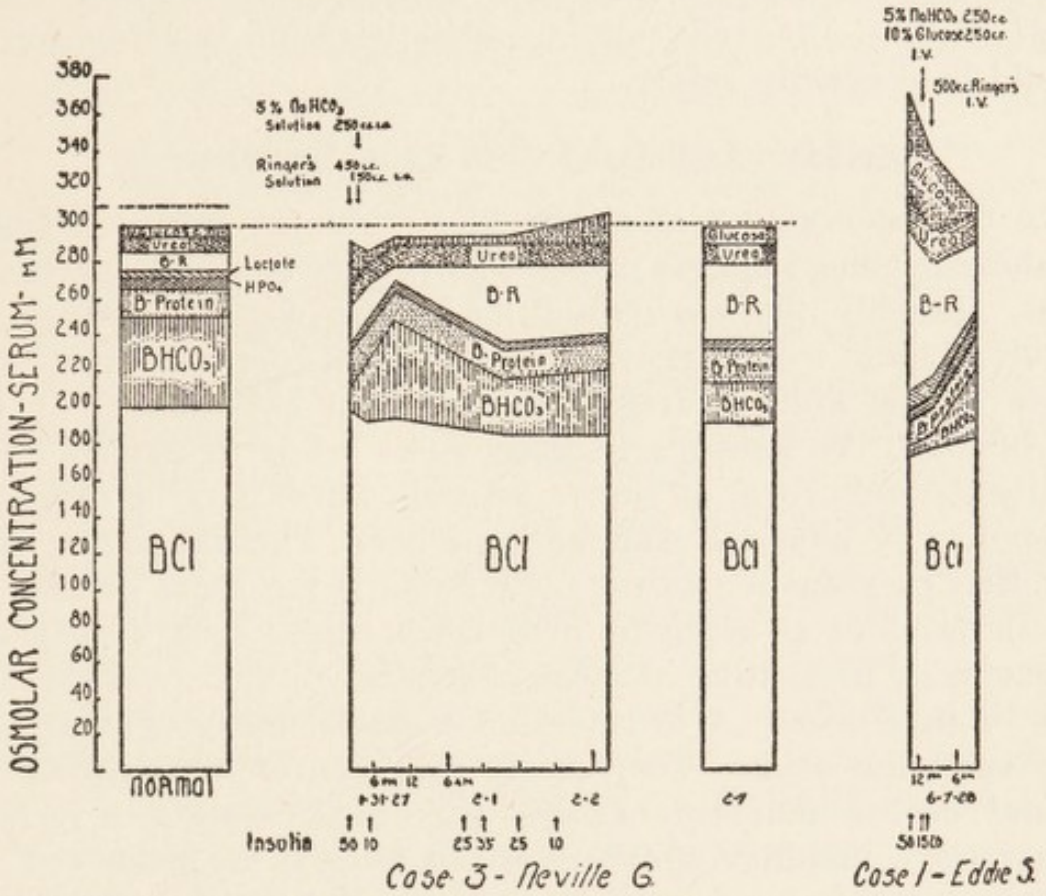


Fig. 47.—Rapid recovery from diabetic acidosis with aid of $NaHCO_3$. (Hartmann.)

appears. Ordinarily this is synonymous with disappearance of diacetic acid from the urine as tested for with 10 per cent ferric chloride solution. Rarely significant amounts of ketone bodies are present in the blood when absent in the urine. Since frequently excess glucose in the body disappears with even the production of hypoglycemia before ketosis is completely abolished, it is safer to combine glucose administration with insulin administration if frequent observations of the blood-sugar concentration are impossible. One-half to one gram of glucose for each unit of insulin should be more than sufficient, since invariably the glucose

content of the body is considerably elevated. Such glucose may be given by mouth or parenterally. When ketosis has been entirely relieved, glucose administration should be discontinued and insulin given in smaller dosage every six hours until glycosuria ceases and the blood sugar returns to normal. At this stage the average patient is ready for his regular diabetic diet.

We have had little opportunity as yet to note the effects of "combined" solution administration on diabetic acidosis. We should expect, however, that its administration would render $NaHCO_3$ and Ringer's solution administration unnecessary and would be distinctly safer.

Acidosis Associated With Renal Insufficiency

In the later stages of severe glomerular or diffuse nephritis acidosis is almost always present, frequently to a very marked degree. Exactly the same condition occurs during infancy as a result of renal insufficiency dependent upon congenital malformations of the kidney, ureteral or urethral obstruction, or pyonephrosis. The acidosis in these cases tends to persist and is really the only form of severe acidosis which may be difficult to recognize by physical examination alone. The underlying cause for $BHCO_3$ reduction in the body fluids is the renal insufficiency which develops as nephritis progresses. Such renal insufficiency operates by permitting abnormal retention of acids and excessive loss of fixed base. Acid retention consists chiefly of phosphoric and sulphuric acids. Very rarely retention of unidentified "organic" acid is indicated. Loss of fixed base occurs as a result of progressive inability of the damaged kidneys to make and substitute ammonia for fixed base and to secrete strongly acid urine. In addition, BCl continues to be excreted into the urine even though considerably reduced in the blood.

Intervening symptoms, such as vomiting, diarrhea, uremic convulsions and circulatory failure complicate the blood picture. Marked vomiting causes loss particularly of gastric juice and tends to elevate $BHCO_3$. Diarrhea would increase acidosis as described previously. Convulsions and circulatory failure would favor accumulation of lactic acid.

In considering the logical treatment of nephritic acidosis, it must first be stated that mild acidosis not only seems to do little harm, but actually may be of benefit in preventing symptoms of

tetany (favored by reduction of serum calcium) and hindering formation of edema. On the other hand, however, severe acidosis leads to excessive dehydration and contributes significantly to the clinical picture of chronic uremia and demands vigorous treatment. Since the underlying cause of the acidosis is the inability of the kidney to conserve sufficient fixed base during the excretion of acid, and since there is no satisfactory way of increasing kidney efficiency, treatment should consist of reducing to a minimum the amount of acid to be excreted and to increase fixed base intake relative to intake of fixed acid. A diet with a high alkaline ash is essential. Such a diet is one containing orange juice, green vegetables, fruits and milk, with but little cereals, bread, meat, or eggs. On this diet alone moderately severe acidosis may be relieved.

It is sometimes necessary, however, to add extra alkali to the diet, and during periods of very marked acidosis, alkali, Ringer's solution and dextrose may have to be given parenterally, as described previously in the treatment of acidosis associated with severe diarrhea and dehydration. It must be remembered, however, that the damaged kidneys have lost their ability to concentrate alkali also and that overalkalinization leads readily to severe alkalosis. The value of "combined" solution in the treatment of nephritic acidosis has not yet been fully determined.

In addition to the measures outlined above, oral administration of calcium acetate is of value in reducing inorganic blood phosphate and in raising blood calcium. A dosage of 15 to 30 grains (1 to 2 grams) three times daily is usually effective.

Other Forms of Acidosis

In addition to the chemical types of acidosis just described, more rarely other forms are encountered. Associated both with acute bacillary dysentery and certain types of respiratory infections, severe acidosis is sometimes seen, apparently due principally to ketosis plus dehydration. Administration of alkali to the very severe cases, Ringer's solution and glucose (with or without added insulin) individually, or "combined" solution by itself relieves this type of acidosis very promptly. Extensive skin burns are also frequently followed by marked acidosis. Products of tissue destruction plus shock with circulatory failure

seem responsible for the $BHCO_3$ reduction. The usual shock therapy, morphine plus Ringer's solution intravenously, is usually sufficient to relieve such acidosis. More rarely, severe acidosis is seen following ingestion of toxic quantities of methyl alcohol, or bichloride of mercury. Detailed information of resulting chemical changes in the blood in such conditions is lacking, but at present alkali, glucose and fluid administration (or administration of "combined" solution) seems indicated in the treatment of them. In the types of carbonic acid acidosis (Table XVIII) indications are to aid circulation and restore normal pulmonary ventilation.

Symptoms and Signs of Acidosis

The only symptom that may be considered pathognomonic of acidosis is hyperpnea or deep breathing, the so-called "air hunger" of Kussmaul, the cause of which has already been explained. The breathing of acidosis is deep, it is not usually increased in rate, and differs markedly from the shallow rapid respiration of pneumonia or the labored breathing of obstruction. It is pauseless and though the several inspirations may vary in depth, in general the excursions of the abdomen and thorax are nearly the same with succeeding respirations. The most striking feature is the amplitude of the respirations and the distinct effort with which they are accomplished. They are heaving, the chest rises and falls with each respiration, and often the accessory muscles of respiration are brought into play. There is no cyanosis except in the presence of cardiorespiratory disease. A cherry-red coloration of the lips is occasionally observed but is of little diagnostic value. Drowsiness may be seen as a frequent accompaniment of acidosis.

Although in well-marked cases of acidosis showing the characteristic respirations the diagnosis is unmistakable, there are other instances in which acidosis of a fairly severe degree exists without occasioning a great change in the character of the respirations. This is particularly true of the chronic acidosis associated with renal insufficiency and of the moribund athreptic young infant.

In order to confirm positively the diagnosis of acidosis and to determine its degree, it is necessary to resort to laboratory methods. The most generally used and most satisfactory method

for this purpose is the determination of the carbon dioxide content of the plasma by the Van Slyke method. The carbon dioxide content of the plasma of normal infants varies from 50 to 60 volumes per cent. A fall in the carbon dioxide content to below 40 volumes per cent is an indication of acidosis. The carbon dioxide is a measure of the bicarbonate content of the plasma.

Another test of value is the determination of the hydrogen-ion concentration (pH) of the blood. Various electrometric and colorimetric methods are available, the most serviceable of which is the colorimetric method of Hastings and Sendroy. The normal pH of the blood is from 7.35 to 7.45. A drop to below 7.30 is an indication that acidosis is present. The determination of the pH is seldom necessary, but occasionally hyperpnea and reduction of CO_2 content occur as a result of intracranial lesions instead of accumulation of acid. In such instances uncompensated *alkalosis* exists and not acidosis. It is only by pH determination that this can be recognized.

Acidosis cannot with certainty be diagnosed by the examination of a single specimen of urine. The reaction of the urine varies greatly in normal infants and may be acid, neutral, or alkaline. If the urine is alkaline or neutral when freshly passed, acidosis may be usually ruled out. An acid urine does not prove the presence of acidosis, but when the urine remains consistently acid after the administration of alkali, the finding is of significance.

In acidosis due to an overproduction of the acetone bodies, acetone and beta-oxybutyric and aceto-acetic acids appear in the urine in sufficient amounts to be detected by qualitative tests, with sodium nitroprussid or ferric chloride. Both of these are in reality tests for aceto-acetic acid, the nitroprussid test being much more delicate. For practical purposes, the ferric chloride reaction is sufficiently delicate. This test is carried out by adding a solution of ferric chloride to the urine until no further precipitate forms. This may require a volume equal to that of the urine. When acetone bodies are present, a deep reddish brown color is produced. Acetone bodies occur in the urine in only one type of acidosis, so that their absence is no indication that acidosis is not present. On the other hand fairly large amounts of acetone bodies may continue to be excreted in the urine after the acidosis has been fully compensated for by alkali administration.

Alkalosis

Alkalosis, the condition in which the ratio $\frac{BHCO_3}{H_2CO_3}$ is increased, or tends to be increased, may result from either increase in the numerator or decrease in the denominator. The conditions under which this may occur are outlined in Table XIX.

TABLE XIX

ALKALOSIS

(Tendency toward or actual increase of ratio $\frac{BHCO_3}{H_2CO_3}$)

I. *Increase of* $BHCO_3$
   A. *Accumulation* of administered alkali.
      1. Overdosage.
      2. Failure of urinary excretion.
         a) Pathologic: due to kidney damage.
         b) Physiologic: due to diminished plasma fixed base.
   B. *Replacement* of other anions by HCO_3.
      1. Cl: due to loss of gastric juice by vomiting, lavage, or fistula.
      2. Lactate, diacetate and beta-oxybutyrate during recovery from acidosis under certain circumstances.

II. *Decrease of* H_2CO_3*:* by hyperventilation.
   A. Voluntary.
   B. Involuntary: due to stimulation of the respiratory center by inflammation, pressure, or *fever*, or psychic disturbances, including hysteria.

Under ordinary circumstances, when alkali is given by mouth in the usual therapeutic dosage, accumulation in the body fluids does not occur, since the normal kidney is able to excrete sodium bicarbonate in a concentration of approximately 15 gm. per liter. Overdosage with accumulation occurs readily, however, when severe kidney damage is present or when the electrolyte content of the body fluids has been reduced. In the latter condition, retention of $BHCO_3$ by the kidney is to be looked upon as a normal physiologic reaction designed to keep the total fixed base or electrolyte content of the body fluids at its normal value. Such a condition is brought about chiefly by loss of gastric juice (through vomiting, lavage, or fistula) in which loss of the fixed anion Cl′, greatly exceeds B^+ which remains in the blood as $BHCO_3$. Prolonged loss of gastric juice, therefore, leads in itself to alkalosis with low total electrolyte concentration due to reduction of BCl (Fig. 48). The clinical conditions chiefly responsible for such loss of gastric juice are pyloric stenosis, gastric fistula, gastric dilatation, high intestinal obstruction and pyelitis.

During periods of alkalosis of this type, the urine is very poor

in fixed base, is distinctly acid (i.e. contains little or no $BHCO_3$), and is free from chloride. The acids excreted are either free or bound to ammonia. Occasionally diacetic acid is present. It can be seen, therefore, that except for the absence of chloride such urine is very similar to the urine secreted during periods of acidosis. If alkali is given to such patients, it will be retained in the body, increasing alkalosis, until the fixed base level has been restored to normal. If, on the other hand, fixed base is given in combination with chloride, as Ringer's solution or sodium chloride, for example, the fixed base level will be restored without increase

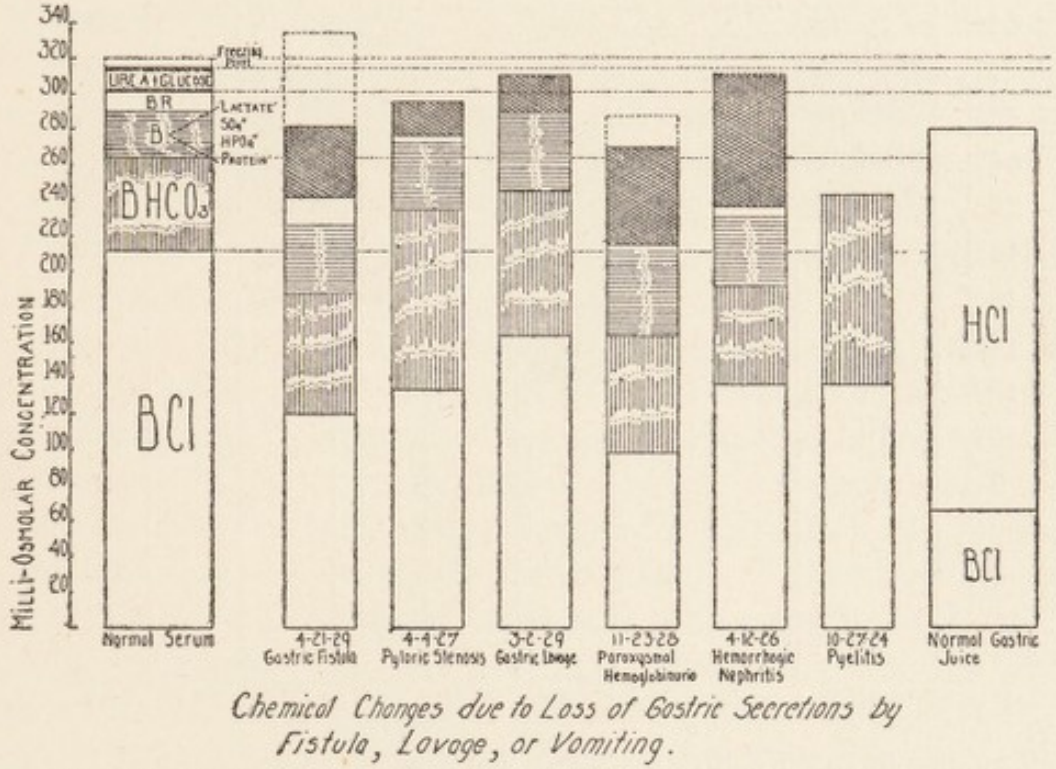


Fig. 48.—Type of alkalosis due to loss of gastric juice. (Hartmann.)

in alkalosis, and excretion of $BHCO_3$ will occur just as rapidly (Fig. 49). In addition physiologic saline or Ringer's solution restores the chlorine ion which is also reduced.

Frequently, as acidosis is being recovered from, $BHCO_3$ seems to "overshoot" the mark, with the development of some alkalosis. Such abnormal increase in $BHCO_3$ seems referable to reduction of total electrolyte content, the $\overline{HCO_3}$ ion being retained in combination with fixed base as long as there is a deficiency in the other anions (particularly Cl'). Such alkalosis does not tend to develop when administration of Ringer's solution or its equivalent is used

in conjunction with other measures for the relief of acidosis (e.g. insulin in the treatment of diabetic acidosis).

Excessive breathing leads to reduction of carbon dioxide tension in the blood and body fluids, and therefore tends toward alkalosis. Alkalosis severe enough to produce tetany may be readily produced by voluntary hyperventilation (Grant-Goldman). Involuntary hyperventilation producing similar changes is seen particularly associated with abnormal stimulation of the respiratory center by pressure or inflammation. Periods of excessive

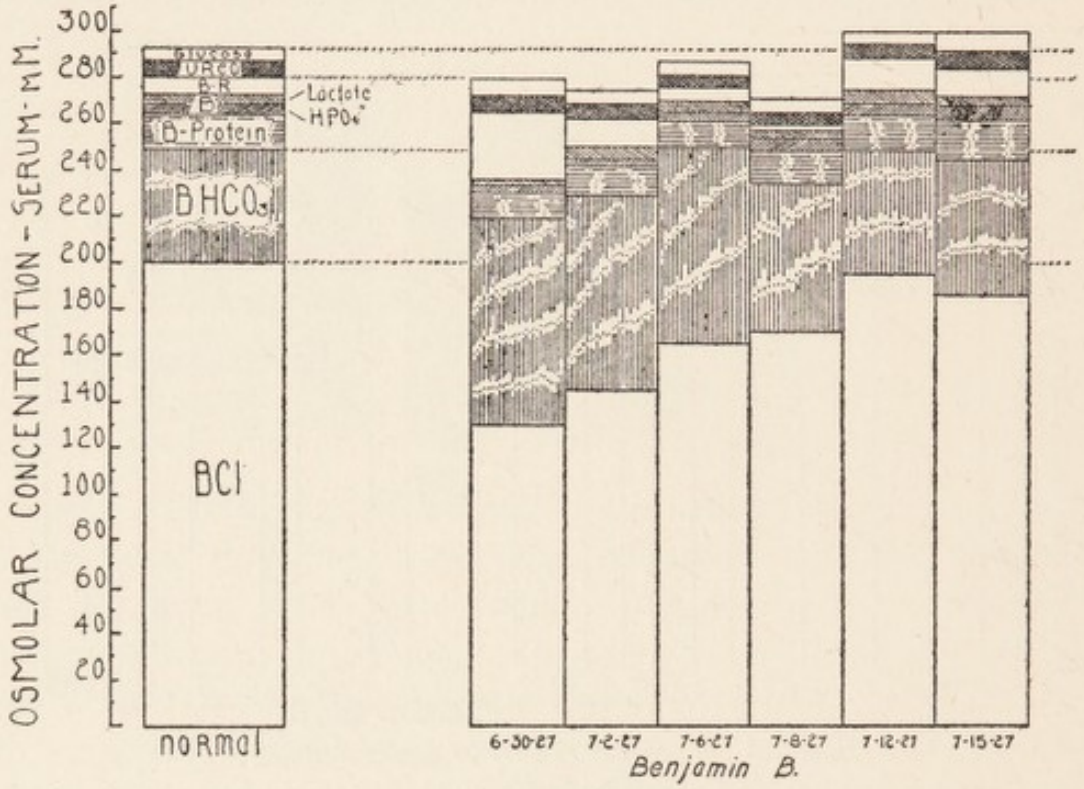


Fig. 49.—Effect of BCl in relieving alkalosis and dehydration associated with intestinal obstruction. (Hartmann.)

breathing are not uncommonly seen in hysteria and in post-encephalitic states. Anoxemia, the result of high altitude, leads to hyperventilation and alkalosis. In cardiac failure, circulatory failure usually offsets the effect of hyperventilation produced by anoxemia, and acidosis is more frequently encountered than alkalosis. Fever is almost invariably accompanied by hyperventilation. In addition, increase in temperature without change in the $BHCO_3/H_2CO_3$ ratio leads to increase in pH (alkalosis) because of the different effect of temperature on the degrees of dissociation of $BHCO_3$ and water.

Symptoms and Signs of Alkalosis

The symptoms of alkalosis are depressed irregular respirations and tetany. The depression of the respirations may be associated with cyanosis. (For symptoms of tetany, see Chapter XXX.)

The laboratory findings are a high carbon dioxide content of the plasma (except in those cases due to hyperventilation), and a high pH of the blood. As mentioned above, the urine may be strongly acid in those cases in which alkalosis is due to vomiting; in other types of alkalosis it may be alkaline. Hence, urinary examination is of but little value as an aid in the diagnosis of alkalosis.

The logical treatment of alkalosis should include (1) immediate relief of tetany, if present; (2) restoration of normal pH; (3) removal of excess $BHCO_3$; (4) restoration of electrolyte if diminished and (5) removal of the original cause for the development of alkalosis.

The relief of tetany is accomplished by the means described in Chapter XXX.

Reduction of pH follows administration CO_2, HCl, or the acid-producing salts NH_4Cl and $CaCl_2$. The latter three substances also reduce $BHCO_3$ by neutralization. Reduction of the latter may also be secured by excretion into the urine in response to increase in plasma electrolyte resulting from administration of physiologic saline or Ringer's solution. Hartmann's "combined" solution has a similar effect, despite the fact that because of its sodium lactate content it is a potential alkalinizer. Because of its BCl and water content, it enables the kidney to excrete $BHCO_3$ at a more rapid rate than it is formed from B lactate. In the presence of renal insufficiency with alkalosis its use would seem contraindicated, however.

In addition to the emergency measures outlined above, it is extremely important to relieve the original cause of the tendency toward development of alkalosis, if possible. This usually means preventing loss of gastric juice by surgical means, discontinuing administration of alkali, or preventing hyperventilation.

CHAPTER XXXIII

NUTRITION AND DEVELOPMENT OF THE TEETH

In the development of the teeth several factors are involved. There must first be the anlage and then an adequate supply of the materials from which teeth are made and the proper conditions in the circulating fluids to allow for the deposition of these materials. As the character of the anlage is determined chiefly by evolutional heredity, any alteration is beyond our control. We are, however, in a position to control, to a considerable extent, the supply of nutritive materials and the conditions which allow for their deposition. In accomplishing this, the most important means at our disposal is the proper regulation of the diet.

That the structure of the teeth may be influenced by the character of the diet, especially during the developmental period, has been demonstrated both on experimental animals and on human beings, and attempts have been made to evaluate the importance of the various elements composing the diet. The most clear-cut results that have been obtained are in the case of experimental animals, for in these it is possible to control the diet absolutely and to obtain teeth for examination at will. In the case of human beings, observations are necessarily limited to individuals who have unintentionally received deficient diets and who have later been given diets in which the deficiencies have been remedied. The observations on human beings have, furthermore, had to be limited to a considerable extent to inspection of the teeth in the mouth. Microscopic examination has been possible only in the case of shed deciduous teeth, extracted teeth, or the teeth of those who have died. A sufficient number of observations have, however, been made upon the teeth of infants and young children living under different dietary regimens to make it evident that the conclusions drawn from animal experiments are, for the most part, applicable to human beings.

The development of the teeth of infants begins early in intrauterine life with deposition of lime salts in the crowns of the temporary teeth as early as the sixth or seventh month of fetal life. At the time of birth all of the temporary teeth are calcified, and the

crowns of the permanent first molars are partially calcified; hence, it might be expected that the diet of the mother during pregnancy would exert an influence upon the development, especially of the temporary teeth. Embryonic tissues possess an extraordinary growth impulse which enables them to remove nutrient materials from fluids in which the amounts present are minimal; hence, the teeth of the developing fetus appropriate calcium, phosphates, and other minerals from the blood, even though this leads to a depletion of the mother's blood and fixed tissues. In this way the developing fetus grows at the expense of the mother. Deficiencies in the diet during pregnancy are, therefore, more likely to effect the mother than the developing fetus, but where the deficiencies are too great, both fetus and mother are affected. The mother can and does supply a great deal for the nutrition of the infant, even when her diet is insufficient. Calcium and phosphates come from her own teeth and bones and protein from her own muscles, but she cannot supply that which she herself lacks, consequently when the mother's diet is deficient in vitamins the developing fetus suffers from a deficiency of these same vitamins.

Vitamins A, D, and C are especially essential for the proper development of the teeth. These vitamins should, therefore, be supplied liberally in the mother's diet. The diet should also contain adequate amounts of calcium salts. When the diet of the mother is deficient in calcium and in the vitamins A and D, the alterations in the mineral composition of the teeth of the young are much greater than when the diet is deficient in calcium alone. E. Mellanby found that puppies born of mothers whose diets during pregnancy were deficient in vitamin D had deciduous teeth in which the enamel was poorly developed and the dentine contained interglobular spaces. Toverud obtained similar results in rats. Marshall found that when vitamin A was deficient in the diet of pregnant animals structural changes occurred in the teeth of the offspring which rendered them especially susceptible to caries in later life. Neff observed extensive hypoplasias in the deciduous teeth of infants and young children, which would lead one to suspect dietary deficiencies during the early development of the teeth in intrauterine life.

Even though the temporary teeth are fairly well formed though unerupted at the time of birth, their structure, as well as that of the developing permanent teeth, may be influenced profoundly by

the character of the diet during infancy. If the infant is breast-fed and the mother is in good health and taking an adequate diet, all of the necessary materials for the development and maintenance of the infant's teeth will be supplied. If, on the other hand, the mother is not in good condition or if her diet is a poor one or if the milk is scanty in amount, the nutritional demands of the infant may not be met. In such cases it becomes necessary to resort to artificial feeding which may or may not be adequate. The diet may be deficient in a variety of particulars, and the effects on the teeth will depend upon which factor is deficient. Simple underfeeding with a diet too low in caloric value, but containing adequate amounts of minerals and vitamins results in general undernutrition and slow growth of the body as a whole. The muscles become lax and hypotonic, so that the stimulus of the muscle contraction on the bones of the maxillae is diminished. In such underdeveloped jaws the teeth are irregularly placed.

When particular elements are deficient in the diet, nutritional disorders result, some of which influence the development of the teeth. One of the most common of these is rickets, a disease which, although a general one, affects especially the bones and the teeth. (Rickets is discussed in detail in Chapter XXIX.) One of the chief causes of rickets is a deficiency of vitamin D in the diet. In rickets, the teeth erupt late and are irregularly set in the jaws, the enamel is of poor quality, and the dentine of irregular structure. Not only are the temporary teeth affected, but also the permanent teeth, especially the first molars and incisors. Marked hypoplasia, with disintegration of the teeth at the biting edges, has been observed in rachitic children. The hypoplastic teeth of children who have suffered from active rickets are more susceptible to caries than are normal teeth.

A deficiency of vitamin A in the diet of the infant results in some retardation in the growth of the jaws, and consequently in irregularity of the teeth. According to Marshall, the pulp cavities become degenerated and inflamed, and the odontoblasts lay down bone instead of dentine. This results in a less dense and less resistant calcification of the tooth structure. Vitamin A deficiency also causes changes in the mucous membranes, including those of the mouth, which render them susceptible to infections. The flow of the saliva is decreased, and there is a tendency to the formation of salivary calculi.

The effects of deficiency in the antiscorbutic or C vitamin have been studied especially by Percy Howe, who has found that such deficiencies result in decalcification of the teeth and alveolar processes, changes in the pulp, and loosening of the periodontal attachments. Howe's experiments were performed on guinea pigs and monkeys. Hanke has noted the frequent occurrence of caries, pyorrhea, and mouth infections in human beings whose diets have been deficient in vitamin C. It has not so far been demonstrated that a temporary deficiency of vitamin C in the diet during infancy results in any permanent changes in the structure of the fully developed teeth. Indeed, it is unlikely that such is the case, for following the administration of sufficient amounts of orange juice or other vitamin C containing foods to individuals with scurvy, a prompt restoration of normal conditions occurs.

A deficiency of calcium salts in the diet of the infant is rarely the cause of changes in the teeth. Such changes as have been observed are essentially the same as those occurring in rickets.

When protein is deficient in the diet of the infant, development is slow and the teeth erupt late; but, so far as is known, there are no changes in their structure. The carbohydrate content of the diet in itself appears to have but little influence on the teeth. There is a popular idea that the eating of too much sugar causes damage to the teeth. The theory that fermentation of sugar in the mouth is the cause of dental decay, has not been substantiated. Carbohydrates are harmful only when they comprise such a large proportion of the diet that inadequate amounts of other foods containing minerals, vitamins, and proteins are taken. Infants fed on proprietary foods having higher carbohydrate contents and deficient in other particulars, often develop rickets, with the accompanying structural changes in the teeth.

The physical character of the diet has some influence upon the development and structure of the teeth. Hard and rough foods which require chewing cleanse and polish the teeth, favor normal development of the jaws, and lead to an increase in the flow of saliva. Consequently it is desirable that infants be given something to chew on as soon as the first teeth are erupted. Crackers, toast, hard bread, and occasionally a piece of bacon, are valuable foods in this respect.

To sum up, the development of the teeth may be influenced to some extent by the diet of the mother during intrauterine life.

The development and structure of both the temporary and the permanent teeth are largely influenced by the character of the diet during infancy. In order to provide for normal development and structure of the teeth the diet of the infant should contain sufficient of the vitamins A, D, and C. These may be supplied through the administration of cod liver oil, egg yolk, and orange juice. An adequate intake of calcium and phosphate is insured by the giving of at least two and one-half ounces of human milk or one and one-half ounces of cow's milk per pound of body weight, per day. As soon as the teeth are erupted the diet should include foods which require chewing. Even though the teeth may be well developed as a result of proper diet during infancy these well-developed teeth will not remain healthy unless the diet continues to be adequate throughout life. The teeth are live tissues which can be maintained in healthy condition only when the nutritional needs are met adequately. There is an increasing amount of evidence that a proper diet is more essential in preventing dental caries and in maintaining normal conditions in the mouth than is mere mechanical cleaning of the teeth.

CHAPTER XXXIV

MISCELLANEOUS TECHNIC

Collection of Urine

The method for collection of urine in infants will depend upon what is desired in the way of study. For the usual routine examination of approximate acidity, albumin and sugar content, sediment, etc., special precautions in collection need not be observed. All that is desired is the collection of a clean sample. In male infants this is done by strapping the proper sized test tube in place with a cloth T-binder. Adhesive tape may be used instead of the cloth, but if a number of specimens are required the frequent removal of the adhesive tape will cause excoriation of the skin. The cloth binder is provided with a hole, through which the test tube is placed, and if the test tube has a sufficiently large lip it will not slip through the hole after the binder is strapped (Fig. 50).

In the case of the female infant a similar test tube may be used, fastened accurately in place with a square piece of adhesive plaster. It is better, however, to use an ordinary canary bird feeding trough. The edges of the hole in the trough are covered with adhesive tape to prevent excoriation. The trough is placed in the proper position so that when the infant is lying on its back the hole will be uppermost (Fig. 51). The trough is anchored in place with a T-binder, two arms of which are placed around the abdomen, the third passing between the buttocks, and being pinned in front to the other two. In either case, after the test tube or trough has been placed, it is necessary to prevent the infant from turning over and spilling the collected urine. For this purpose restraints are used by means of which the arms and legs are anchored firmly to the mattress.

If urine is to be collected for bacteriologic study, it is best in the case of both male and female infants to obtain specimens by catheterization. If the urine is to be collected for chemical study which includes determination of pH, it is necessary to prevent loss of CO_2 from exposure to the air and to prevent bacterial growth after collection. In such instances the urine is made to pass

through a funnel to the bottom of a bottle which contains an inch-thick layer of mineral oil and a few c.c. of toluol.

Urine Cultures

A satisfactory method for determining the presence of such bacteria as colon or typhoid bacilli or staphylococci in the urine is to deposit a drop of the catheterized specimen of urine on a

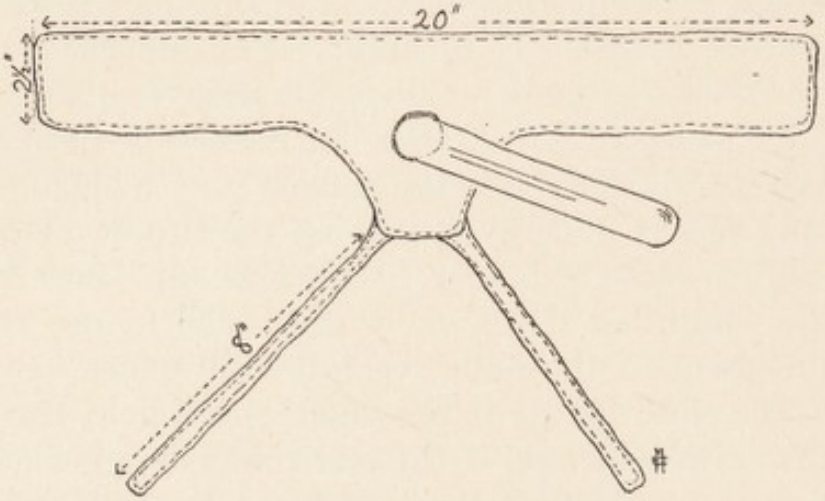


Fig. 50.—"T" binder with test tube attached for collection of urine from male infant.

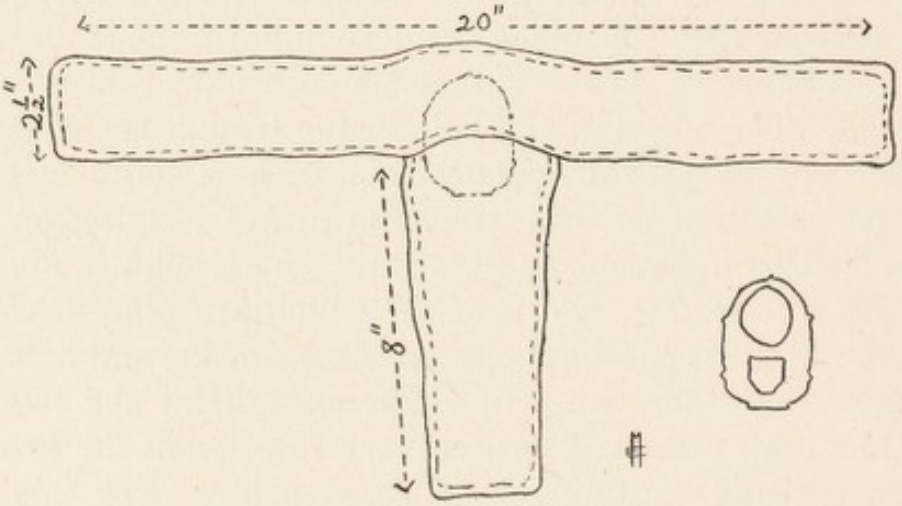


Fig. 51.—"T" binder with bird cup, arranged for collection of urine from female infant.

poured Petri plate of nutrient agar, and on a second plate five to ten drops. The surface of each is smeared over with a sterile glass rod, first spreading the single drop over its plate and then the second plate containing a larger amount of the urine. After twenty-four hours' incubation, isolated colonies when present can be picked, smeared for microscopic examination, and subcultured

for identification. For such organisms as pneumococci and streptococci, plates of blood agar are used.

Stool Cultures

In general, cultural examination of the stool is limited to a search for typhoid and dysentery bacilli. To isolate these from the colon bacilli normally present in the stool, special solid culture medium containing 1 per cent of lactose and an indicator is used. Lactose-litmus-agar or lactose-bromcresol-purple-agar plates are quite satisfactory. It is essential that the stool specimen should be *fresh*. It may be obtained by inserting a sterile catheter into the rectum and collecting the material caught in the "eye" of the catheter, or a fleck of bloody mucus from a freshly passed stool may be used. The material is emulsified in a small amount of sterile salt solution or broth and a drop of this emulsion placed on the dry surface of such a plate. This material is smeared over the surface of the medium with a sterile bent glass rod or wire. After incubation for twenty-four hours, the isolated colonies of bacteria which develop, if colon bacilli, produce acid from fermentation of lactose and change the color of the medium. If litmus is used, the acid changes the color to red, and if bromcresol purple is used, to yellow. Typhoid, paratyphoid and dysentery bacilli do not ferment lactose and, therefore, do not alter the color of the indicator. Such colonies may be identified by the usual bacteriologic methods.

Collection of Blood

The technic of collection of blood will also depend upon what is to be studied. For most bacteriologic, serologic, and chemical studies, the amount of blood necessary is such as to require venipuncture. In the small infant the most accessible vessel is usually the external jugular vein. As a rule, the veins elsewhere are too small to be seen or felt distinctly, and are not usually satisfactory for the collection of more than a few drops of blood. The method of puncturing the external jugular vein and aspirating it is of importance. A needle of a fairly large gauge (16-18 as a rule) is selected; it is desirable to have a needle with a fairly short bevel. Such needles can be made by merely cutting off the original point and grinding a new one on a small oil stone. Such a short bevel lessens the risk of transfixing the vein, which usually causes

extravasation of blood around the vein with resultant occlusion. The infant is wrapped in a sheet with its arms to its sides. It is held firmly on its back with its head hanging over the edge of the table. The head is turned so as to bring the external jugular vein uppermost, and with one hand pulling down on the uppermost shoulder and with the other hand holding the head, a single attendant is able by leaning gently on the infant to hold it firmly in place. The skin over the vein is cleansed with iodine and alcohol, and with the short beveled needle attached to a syringe, the vein is entered directly from above. After the needle has entered the vein for a distance of one-quarter to one-half inch, the plunger of the syringe is withdrawn slowly and the blood aspirated into the sterile syringe. The blood is discharged from the syringe into a test tube, and allowed to clot. If the blood is to be used for ordinary serologic examination, such as the Wassermann test, or for blood matching, no other precautions need be taken.

If blood is to be examined for pH, CO_2, protein or chloride content, etc., it is necessary to prevent exposure to the air. This can be done by first placing a small amount of mineral oil in the syringe, which must be tight fitting. Before it clots, the blood is discharged into a test tube filled with mineral oil, the excess oil being floated out of the tube as the blood settles. If the blood is to be cultured for bacteria, the syringe and needle in which it is to be obtained had best first be sterilized in the autoclave.

Grouping and Matching of Blood for Transfusion

For blood grouping or matching, both serum and cells must be obtained. For a satisfactory suspension of cells a few drops of the venous blood are dropped into about 5 c.c. of physiologic salt solution. The remainder of the blood is then placed in a test tube and allowed to clot for liberation of serum.

Occasionally in very small infants who have very few veins available it is desirable not to risk damaging the vein for transfusion by puncturing it to obtain blood for matching. Capillary blood may be obtained in sufficient quantity in the following manner: The heel is dipped in warm water and then cleansed carefully with iodine and alcohol, and punctured deeply enough to obtain several large drops of blood. The blood is then aspirated into a capillary glass tube, one end of which is later sealed in a Bunsen flame. The glass tube is then centrifuged and broken

at the proper place so as to yield one or two drops of serum. A saline suspension of cells may then be obtained from the clot.

Before transfusion of blood, it must be determined that the donor is healthy, has a negative Wassermann, and has blood which is compatible with the recipient's blood. A compatible donor is one whose serum will neither cause agglutination nor hemolysis of the recipient's cells, and whose cells will neither be agglutinated nor hemolyzed by the serum of the recipient. Bloods in the same group are usually compatible. However, since occasionally bloods which are apparently in the same group do not match perfectly the safest procedure is always to determine directly whether they "cross-match." To determine this, serum and cells from both the donor and the recipient are necessary. Two slide preparations are made: (1) a drop of the recipient's serum mixed with a drop of the donor's cell suspension, and (2) a drop of the donor's serum mixed with a drop of the recipient's cells. It is desirable to cover the preparation with a cover-slip which is sealed with vaseline to prevent drying. A satisfactory preparation, when first looked at, should show uniform distribution of the cells. The slides are then left to stand for thirty or forty-five minutes and again examined. After such an interval it can easily be determined whether agglutination or hemolysis has occurred.

It is well to select a donor whose blood matches so that neither agglutination nor hemolysis occurs in either combination. Never should blood be given if the donor's cells are agglutinated by the recipient's serum. In cases of emergency blood may be used without much likelihood of harm if only the recipient's cells are agglutinated by the donor's serum since, after dilution of the donor's blood with the recipient's, such agglutination is not so likely to occur as in the case of the less diluted slide preparation.

From the above it may be seen that preliminary grouping is unnecessary if the final cross-matching is done. Grouping, however, is often of much help in a preliminary selection of possible donors, especially when repeated transfusions are contemplated.

Blood Transfusion

After selection of the donor it must be decided whether a direct transfusion or an indirect citrate transfusion should be given. The direct seems to possess few, if any, advantages over the indirect method. When blood diseases are encountered in which

abnormal bleeding occurs because of a reduction or absence of platelets, the direct method seems superior to the citrate. On the other hand, the citrate method, particularly for the transfusion of infants, seems to possess certain advantages: (1) a sufficient quantity of blood may be obtained from the donor to provide for several transfusions; (2) the blood may be given to the patient without necessarily cutting down upon the vein; (3) the superior longitudinal sinus may be used if necessary; (4) the blood may be given much more slowly than by the direct method, and in the absence of the donor. In general the citrate method is to be preferred. This is carried out as follows:

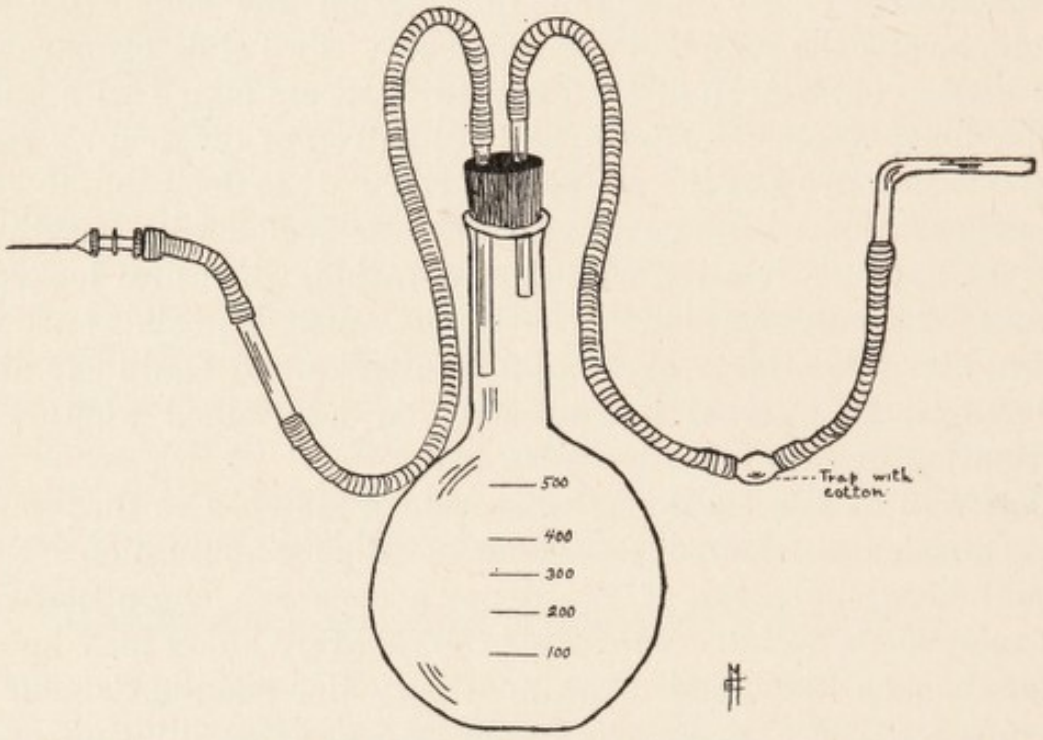


Fig. 52.—Apparatus for collection of blood for transfusion by the citrate method.

The apparatus necessary for collecting the blood is first assembled. This consists of a 500 c.c. Erlenmeyer or Florence flask which is marked in such a way that one can determine how much blood is present in the flask with a fair degree of accuracy (plus or minus 25 c.c.). The flask is fitted with a two-hole rubber stopper. Through one hole a piece of glass tubing is inserted which extends almost to the bottom of the flask. The glass tubing is connected with a piece of rubber tubing of sufficiently thick wall to withstand moderate suction (Fig. 52). Only a high grade of pure gum tubing should be used, and this should be especially treated before being used the first time. The tube should be soaked in a

5 per cent solution of sodium bicarbonate for three or four hours and then in 1 per cent hydrochloric acid; it should then be thoroughly washed and allowed to stand overnight in a jar of distilled water. The end of this rubber tube is fitted with an adapter which will fit the needle which is to enter the donor's vein. In the other hole of the stopper a shorter glass tube is inserted, which is also connected with rubber tubing at the end of which suction may be applied in some manner, usually by the mouth of the operator. To prevent contamination of the flask by saliva of the operator's mouth a trap is interposed between the end of the tubing on which the operator sucks and the flask into which the blood flows. A small bottle with a two-hole stopper with glass and rubber tubing connections, or simply a pipette stuffed with cotton, makes a satisfactory trap.

The whole collecting apparatus is sterilized by autoclaving at 15 pounds for thirty minutes. A sterile solution of 3 per cent sodium citrate is then prepared in an amount sufficient to equal one-tenth of the amount of blood to be taken and is then placed in the flask. It is best to do this by aspirating it through the needle and rubber tubing through which the blood must pass before it enters the flask. If, for instance, 300 c.c. of blood are to be taken, 30 c.c. of sodium citrate will be placed in the flask. The donor's vein is then punctured with as large a needle as possible, and blood is aspirated into the flask with suction. The flask is continuously agitated to assure a thorough mixture of the blood with the citrate solution. When the proper amount has been secured, the flask is stoppered with a sterile cotton plug. Blood so obtained may be kept in a refrigerator for as long as two or three days if necessary.

Before transfusing the blood it is filtered from the original flask through several layers of cotton gauze into another sterile flask (preferably a 500 c.c. Florence flask) which is also fitted with a two-hole stopper and glass and rubber tubing connections so that the blood may be given by gravity. It is very convenient to connect the end of the tube with a syringe with a side-arm. The flask of blood is warmed to body temperature by immersion in warm water. Air is then expelled from the rubber tubing and the syringe by inverting the flask, and at first holding the end of the rubber tubing and the syringe above the level of

blood in the flask, and then gradually lowering them until the blood flows out, expelling the air. With the plunger inserted fully, the syringe is attached to a needle such as that used for the collection of blood. The vein is entered in the same manner as previously described. The plunger is then withdrawn to a point beyond the side-arm, permitting the blood to flow into the recipient's vein. Usually little hydrostatic pressure (12-24 inches) is necessary to cause the blood to enter the donor's vein at a desirable rate. If the child is crying vigorously, however, its venous

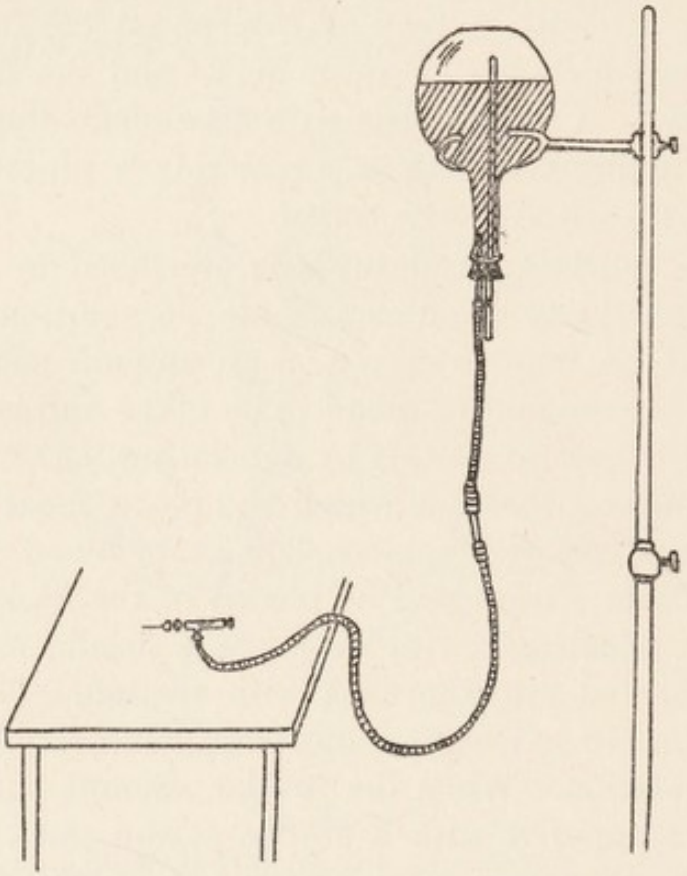


Fig. 53.—Arrangement of apparatus for blood transfusion by the citrate method.

pressure during the prolonged expiratory phase of respiration may increase to such a point that the blood will flow in the wrong direction. In such instances, and also when a very small needle is used, it may be necessary to use several feet of hydrostatic pressure. An adjustable standard with a ring holding the flask (Fig. 53) is convenient for this purpose.

In general the dosage of blood is 20-30 c.c. per kilogram of body weight, given at a rate of about 10-15 c.c. per minute. A convenient indicator for determining the speed at which the blood enters the recipient's vein can be fashioned by placing a small

amount of sterile saline solution or 70 per cent alcohol in the bottom of the glass tubing through which the air enters the flask as the blood leaves it. This solution is sucked up through the tubing at the same rate that the blood leaves it. As it is sucked up, it gradually coats the wall of the glass tubing, until finally no column of fluid remains. The fluid then gradually flows down the wall by gravity and collects again at the bottom of the tubing, whence it is sucked up as before. The speed at which the fluid column rises serves as an indicator of the speed at which the blood is leaving the flask. More blood than 20-30 c.c. per kilo of body weight may be given to patients who have diminished blood volumes, due to hemorrhage. Less blood should be given to patients with pneumonia or cardiac diseases.

In many instances no superficial veins which are large enough to permit transfusion can be seen in small athreptic or dehydrated infants. When the anterior fontanelle is open, the superior longitudinal sinus may be entered easily and serves excellently as a site for blood or fluid injection. Because, however, of its situation, care and skill must of necessity be exercised in puncturing this vein. The technic usually employed is as follows: The infant is wrapped securely and pinned in a sheet so that the assistant has no difficulty in keeping the infant flat on its back and holding its head steady. The head rests near the edge of the table. The person holding the head has both hands held firmly over the cheeks and temporal regions. The operator giving the blood is seated at the head of the patient. The scalp over the anterior fontanelle is shaved, and cleansed with iodine and alcohol. The operator then locates exactly the midline by noting the position of the anterior and posterior angles of the fontanelle and the nose of the patient. With the side-arm syringe connected with the flask of blood, and attached to a specially shortened needle, one-fourth to one-half inch in length, the longitudinal sinus is entered by firmly thrusting the needle at an angle of about 45 degrees, the syringe and needle being kept in the sagittal plane with the needle pointing posteriorly. The superior longitudinal sinus lies just beneath the inner table of the fontanelle at this point, and is exactly in the midline. By the use of the shortened needle the danger of going through both walls of the vessel is almost entirely removed. The plunger is withdrawn, and the blood is allowed to

flow into the sinus as in the case of the external jugular vein. At frequent intervals, however, the plunger should be replaced and withdrawn in order to ascertain whether the needle point is properly within the vessel. There should be no difficulty in inserting and removing the plunger past the side-arm. If, because of faulty position of the needle, blood cannot be withdrawn readily, the plunger will not be moved easily. In such an event a fresh puncture of the sinus should be made.

In addition to the external jugular vein and the posterior longitudinal sinus, there are other veins that may sometimes be utilized. Occasionally the cubital vein may be sufficiently large and visible to be entered readily. Occasionally also a fairly large vein may be seen on the medical aspect of the ankle.

When other veins are not available, the femoral is sometimes used. This is not visible at the surface but may be located by palpating the femoral artery just below Poupart's ligament. The vein lies just medial to the artery in the same sheath. With the finger on the artery the needle is inserted just to the inner side. It is sometimes surprisingly easy to strike the vein in this manner, even though it is not seen.

In infants of but one or two days of age, particularly when suffering from hemorrhagic disease of the newborn, the umbilical vein may not be firmly thrombosed, and may be entered quite readily after the removal of the ligature around the umbilical cord.

Saline Administration

Physiologic salt solutions frequently injected are: first, 0.85 per cent sodium chloride; second, Ringer's solution; and third, Hartmann's combined solution (see page 322). In the case of the first two solutions, the preparation is very simple. Tablets of the dry salts may be obtained from a number of pharmaceutic firms, and they simply need to be dissolved and diluted to the proper volume with distilled water. The latter solution is obtainable in ampule form and merely requires dilution with sterile water.

Saline solutions may be given intravenously into any of the veins which were mentioned above, and in the same manner as transfusions. Saline solutions may also be given subcutaneously or intraperitoneally.

Intraperitoneal Injection

The technic for the intraperitoneal administration of fluids is as follows. The sterile saline solution in a flask (such as was described above for the giving of blood) is warmed to body temperature. A needle of 16-18 gauge and one and one-half inches in length, not too pointed, is attached to the adapter at the end of the tubing. After the air is expelled from the system, the operator grasps the shank of the needle between the thumb and forefinger, at the same time clamping off the rubber tubing with the third finger and thumb. The infant is held flat on its back, the thighs immovable and extended. The skin midway between the symphysis pubis and umbilicus, previously cleansed with iodine and alcohol, is picked up with the left hand. The needle is thrust into the abdominal wall somewhere above the midpoint. The direction of the needle is an angle as acute as possible to the plane of the abdominal wall (i.e., almost parallel with the abdominal wall) and in the sagittal plane. After the needle has entered the peritoneal cavity, it is thrust up to the shank and held firmly in position, parallel to the abdominal wall. It usually then can be palpated very distinctly through the umbilicus. Saline solution is run in by gravity, until the abdomen begins to feel distended. Sometimes three to four hundred c.c. may be injected within ten to fifteen minutes into the intraperitoneal cavity of an infant weighing ten pounds.

Entrance more laterally into the abdomen through the rectus muscle is preferred by some who feel that by so doing there is less danger of puncturing the bladder, and that the muscle tends to seal up the needle hole more effectively. However, the midline has been used very satisfactorily on my service.

The chief contraindications to intraperitoneal injection are inflammation of abdominal organs, abdominal distention, and contemplated intraabdominal operations.

Dextrose (Glucose) Administration

The technic of the administration of dextrose is similar to that described for the administration of blood and saline solutions. The preparation of dextrose solution requires a few words. Chemically pure dextrose must be used and must be made up with freshly distilled water. The solution, if to be used immediately,

may be sufficiently sterilized by boiling in a flask for from three to five minutes. If the solution is to be kept for future use, it should be sterilized in an autoclave at a pressure of fifteen pounds for thirty minutes. Occasionally considerable titratable acidity in such solutions develops on standing, and may be partly responsible for such reactions as sometimes occur when dextrose solutions are injected. Usually, however, even though the pH of the solution drops to 5± there is so little titratable acid present that the solution is practically nonirritating. Grulee advises dry sterilization of the dextrose followed by solution in previously boiled water; such a solution is less irritating and is to be recommended when intraperitoneal injections are used. A very convenient method of preparing dextrose solutions is by the use of sterilized ampules of 50 per cent dextrose. These may be obtained from pharmaceutic houses, and the contents need only to be diluted with boiled distilled water.

The concentration, amount, and the route administration of dextrose solutions will depend upon the purpose for which the solutions are administered. If administration of water with quickly available carbohydrate is chiefly desired, isotonic (6 per cent) or slightly hypertonic (10 per cent) solutions are used and given slowly. If diuresis is particularly desired more concentrated solutions, 15-25 per cent, are given intravenously at a rather rapid rate (30 c.c. per minute). If the purpose is to provide as many calories as possible in the form of glucose, as for instance in the case of the athreptic infant who cannot take enough food by mouth to gain, 20 per cent dextrose, to which is added one unit of insulin for each 2 gm. dextrose is given intravenously as slowly as possible, so as to keep glycosuria at a minimum.

Gavage

The infant is wrapped in a sheet and held on its back. In the case of the small infant without teeth a rubber catheter, which is fairly stiff, is attached to the barrel of a sterile glass syringe, and thrust into the mouth and down the esophagus. In the case of older infants, the catheter is inserted through the nose. As the catheter enters the stomach the air bubble usually present there can be heard as it is expelled through the syringe. If the catheter has not entered the esophagus, but has entered the trachea instead, there will be no doubt of the fact, as the infant will become sud-

denly very dyspneic and restless. When the air bubble in the stomach has been expelled, the glass syringe is filled with the material to be gavaged, and the flow started by "milking" the catheter. Occasionally slight pressure with the plunger of the syringe is necessary to start the flow. The fluid to be gavaged is run as rapidly as possible by gravity. Before withdrawing the catheter, it is pinched tightly and then withdrawn as quickly as possible so as not to tickle the pharynx and provoke vomiting.

In the event that it is necessary to gavage thickened feedings, such as are used in the treatment of pyloric stenosis, some type of grease gun, as for instance an alemite grease gun, is very satisfactory. A catheter can be attached to the gun, and the thickened feeding forced in under pressure.

APPENDIX

St. Louis Children's Hospital

OUTLINE FOR HISTORY TAKING

Chief Complaint—

Family History.—Father. Age. Condition of health.
Mother. " " " "

Transmissible diseases. Lues. Tuberculosis in family or immediate associates, history of contact.

Other children, age and health. Causes of death. Miscarriages, month and cause.

Previous History.—Birth, premature or at term; spontaneous or instrumental. Weight. Condition at birth—cyanosis, snuffles, convulsions, jaundice, hemorrhage, malformations.

Development.—Age when first sat alone, walked, talked, dentition.

Feeding.—Breast or artificial. Reason for weaning. Amount and feeding intervals. Did well or not. Feeding after infancy. Intervals. Character. Tea, coffee, candy.

Habits.—Bowels. Hours of sleep. Appetite. Exercise.

Illnesses.—Measles, pertussis, scarlet fever, diphtheria. Severity and convalescence. Similar illness to present. Operations or injuries. Recent exposure to contagion.

Present Illness.—Onset (date). Sudden or insidious. First symptom. Fever. Appetite. Pain. Bowels. Cough. Negative history of value in differential diagnosis. If history suggests a particular disease, inquire as to all symptoms characteristic of this disease.

St. Louis Children's Hospital

OUTLINE OF PHYSICAL EXAMINATION

Age_____ Weight_____ Length_____ Pulse_____ Respiration_____

Aspect.—Nutrition and development. Posture in bed. Evidences of distress. Sensorium.

Skin.—Pallor or cyanosis. Eruptions. Edema. Turgor. Hair (coarse or fine).

Mucous Membranes.—Moist or dry. Pallor. Rhagades. Eruptions (Koplik spots). Inflammations.

Lymph Nodes.—Tonsils, cervical, axillary, epitrochlear, inguinal.

Head.—Circumference. Shape. Fontanelle (size, tension), cranio-tabes. Prominences.

Eyes.—Abnormality of palpebral fissure. Ptosis. Nystagmus. Pupillary reflexes. Strabismus. Inflammation.

Ears.—Discharge. Odor. Mastoid tenderness. Appearance of drum. Presence of furuncles in canal.

Nose.—Discharge (character). Obstruction.

Mouth.—Teeth (number, condition, abnormalities).

Gums.—(Spongy, bleeding, etc.)

Tongue.—Size. Tremor. Character of surface.

Fauces.—Inflammation. Paralysis of palate.

Pharynx.—Obstruction or inflammation.

Chest.—Size. Symmetry. Expansion.

Lungs.—Type of respiration. Fremitus. Percussion. Auscultation (character, pitch, intensity, duration of breath sounds). Adventitious sounds. Râles—character, location, persistence. Friction sounds. Rhonchi.

Heart.—Apex impulse. Location (interspace, cm. from midsternal line). Diffuse or well localized. Force. Thrills.

Percussion.—Outlines, right and left border. (Maximum distance in cm. from midsternal line).

Auscultation.—Rhythm. Force.

Sounds at apex and base, 1st and 2nd. Character. Intensity. Comparison between P_2 and A_2.

Murmurs.—Location, maximum intensity. Transmission. Time. Quality. Friction sounds.

Pulse.—Character. Rate.

Abdomen.—Distended, flat, or scaphoid. Tenderness or rigidity. Presence of masses or fluid.

Liver.—Cm. below costal margin. Soft, hard, or smooth.

Spleen.—Cm. below costal margin. Soft, hard, or smooth.

Kidneys.—Palpable or movable.

Spine.—Curvature. Angular deformity. Tenderness. Rigidity.

Extremities.—Paralyses. Swelling. Tenderness. Deformity.

Reflexes.—Facial. Biceps. Abdominal. Cremasteric. Patellar. Plantar. Clonus. Kernig. Brudzinski. Babinski, etc.

Genitalia—

Summary.—Chief positive features in history and physical findings. Impression as to diagnosis.

NAME OF EXAMINER.

The Clinical Chart

A satisfactory form of clinical chart for use with infants is shown on page 357. This gives on one sheet the essential information desired as to the progress of the patient. The chart is arranged for use with the metric system of weights and measures, and centigrade temperatures. It may be easily modified, however, for use with the English system. The temperatures are charted in black and the weights in red. The feeding formulas are written in the blank squares just under temperature-weight chart. In the column marked "Vomiting" the time that vomiting occurs, together with the approximate amount is recorded. In recording the bowel movements the number of stools passed during the day and night of each day are recorded separately, as this serves to give a better idea as to the progress of the patient.

Signs are used to indicate the number and character of the stools. The designations customarily used are as follows:

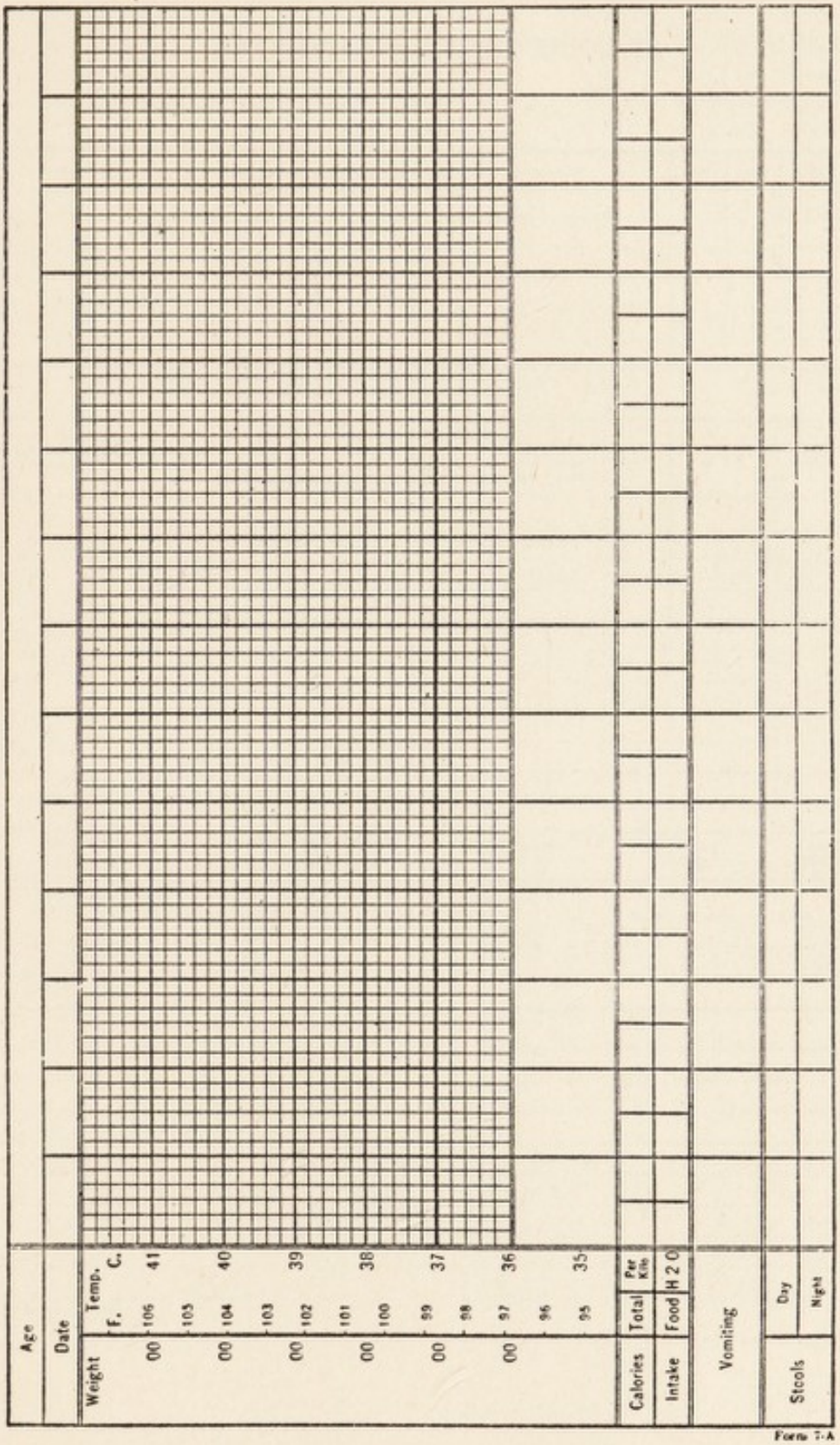
|	Normal stool
/	Moderately loose stool
—	Watery diarrheal stool
/\	Soft stool with mucus
/\	Soft stool with mucus and pus
/[b]	Soft stool with blood (the sign may also be made in red to indicate blood)
□	Hard, constipated stool

Laboratory Sheet

A convenient form of laboratory sheet to be attached to the history is shown on page 358. The space at the bottom of the sheet is used for recording special examinations such as spinal fluid and blood chemistry findings, and the results of blood cultures.

ST. LOUIS CHILDREN'S HOSPITAL

NAME_____ NO._____



Form 7-A

ST. LOUIS CHILDREN'S HOSPITAL

No. Name

BINDING LINE

URINE

Date	Reaction	Sugar	Alb.	Microscopical	Date	Reaction	Sugar	Alb.	Microscopical

BLOOD

Date	% Hb.	R. B. C.	W. B. C.	DIFFERENTIAL						No. Counted	Parasites	REMARKS (Record if reds are abnormal, if platlets seem increased or diminished, etc.)
				Neutrophiles	Eosinophiles	Basophiles	Lymphocytes	Large Mono. and Transit	Myelocytes			

THROAT CULTURE		VAGINAL SMEAR		PIRQUET TB TEST			SCHICK TEST			WASSERMANN REACTION			
Date	Result	Date	Result	Date	Result		Date	Result		Date	ANTIGEN		
					1d.	2d.		2d.	4d.				

Form No. S—536—12-23

Drugs

	DOSES		
	6 MONTHS	1 YEAR	
Acetyl Salicylic Acid (Aspirin)	½ gr. (0.03)	1 gr. (0.06)	Every four hours
Ammonium Chloride (To acidify urine)	5 gr. (0.3)	5 gr. (0.3)	Every four hours
Antipyrin	½ gr. (0.03)	1 gr. (0.06)	Every four hours
Atropine Sulphate (Mouth)	$\frac{2}{1000}$ gr. (0.0001)	$\frac{3}{1000}$ gr. (0.00015)	Every four hours
Bismuth Subcarbonate	10 gr. (0.06)	15 gr. (1.0)	Every four hours
Caffeine Sodio-benzoate (Hypo.)	1 gr. (0.6)	1½ gr. (0.1)	P. r. n.
Calcium Acetate or Lactate	20 gr. (1.25)	30 gr. (2.0)	Every four hours
Calcium Chloride (Mouth)	15 gr. (1.0)	15 gr. (1.0)	Every four hours
Calcium Chloride (Intravenous)	3 gr. (0.2)	5 gr. (0.3)	Once daily
Camphor in Oil (Hypo.)	5 min. (0.3)	7 min. (0.5)	P. r. n.
Caprokol (Hexylresorcinol in oil)	1 dram (4.0)	1 dram (4.0)	Three times daily
Castor Oil	2 drams (8.0)	3 drams (12.0)	One dose
Chloral Hydrate (By enema)	3 gr. (0.2)	5 gr. (0.3)	P. r. n.
Codeine Sulphate	$\frac{1}{16}$ gr. (0.004)	$\frac{1}{12}$ gr. (0.005)	P. r. n.
Ephedrin	$\frac{1}{12}$ gr. (0.005)	⅛ gr. (0.008)	P. r. n.
Epinephrin (Adrenalin 1:1000) (Intramuscular)	2 min. (0.125)	3 min. (0.2)	P. r. n.
Hyoscine Hydrobromide	$\frac{1}{480}$ gr. (0.00015)	$\frac{1}{320}$ gr. (0.0002)	P. r. n.
Iron Lactate	2 gr. (0.125)	3 gr. (0.2)	Three times daily
Magnesium Sulphate (Hypo.)	10 gr. (0.6)	15 gr. (1.0)	Twice daily
Magnesia, Milk of	1 dram (4.0)	2 drams (8.0)	Once daily
Methenamine (Urotropin)	5 gr. (0.3)	7½ gr. (0.5)	Four times daily
Mineral Oil	1 dram (4.0)	1½ drams (6.0)	Twice daily
Morphine Sulphate	$\frac{1}{48}$ gr. (0.0015)	$\frac{1}{32}$ gr. (0.002)	P. r. n.
Paregoric	5 min. (0.3)	8 min. (0.5)	Four times daily
Phenobarbital (Luminal)	¼ gr. (0.015)	½ gr. (0.03)	Three times daily
Pituitrin (Hypo.)	2 min. (0.125)	3 min. (0.2)	P. r. n.
Sodium Bicarbonate	30 gr. (2.0)	45 gr. (3.0)	Every four hours
Sodium Bromide	3 gr. (0.2)	5 gr. (0.3)	Every four hours
Sodium Citrate	45 gr. (2.5)	60 gr. (4.0)	Every four hours
Theobromin Sodio Salicylate (Diuretin)	2 gr. (0.125)	3 gr. (0.2)	Four times daily

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