DIGESTIVE DISTURBANCES OF ARTIFICIALLY FED BABIES *

TRANSLATED BY

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During the last twenty years there has been much change in the interpretation of symptoms occurring in the digestive diseases of babies. The older physicians regarded acute and chronic gastrointestinal disorders as being always due to diseases of the stomach and intestines. Fermentation dyspepsia, fermentation catarrh, gastro-enteritis, cholera infantum, dysentery, ileocolitis. These were compared with similar conditions in adults, allowance being made for the slighter resistance in babies which favored a severe course of the disease.

Widerhofer made a classification on anatomic grounds, dividing the various symptom-complexes into:

1. Purely functional.
2. Catarrhal.
3. Ulcerative-inflammatory lesions of the intestines.

These were clinically distinguished by the character of the stools and by the constitutional symptoms; thus, he speaks of acute and chronic dyspepsia, enterocolitis and enteritis with inflammatory lesions in the large intestine. A fourth form was the murderous cholera infantum.

This description is most valuable. Yet there were many intermediate and transitional forms and but few clear-cut typical cases.

Although severe inflammatory infections of the intestine do exist, yet they are less frequent in importance and frequency than purely functional disorders. It is also true that persistent functional disturbance may cause anatomic changes.

In all varieties of cases a uniform and normal condition of the intestines may exist where the old teaching would suggest severe organic lesions. With some digestive disorders the symptoms and course are more dependent on general visceral changes than intestinal inflammation.

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* This article is a condensed translation of Finkelstein and Meyers' treatise on diseases of the digestive tract in Feer's new book on Diseases of Children.

Thus, bacterial infection does not play the important rôle formerly supposed. Many symptoms are due more to alimentary food-intoxication than to bacterial toxins. The vomiting, fever, diarrhea are usually due to disturbed metabolism; hence the term introduced by Czerny "Disturbance of Nutrition."

Formerly great importance was ascribed the bacteria and bacterial products in polluted milk, yet this factor is of really slight importance compared with improper dosage or character of food. The maintenance of health and development is intimately associated with good functional energy and normal tolerance. A child with good tolerance may be injured by under-nourishment or over-nourishment; with correct feeding a baby may become ill through primary weakness of digestion, a misproportion between the demands of appetite and the process of assimilation.

Inanition also plays a serious rôle in infantile pathology. The food may be too highly diluted or lack important food element, as in gruels and proprietary foods. There are also disturbances due to over-nourishment; the digestive functions are overworked and weakened. The tolerance has been exceeded even if the quantity and character of the food recipe be theoretically correct. Overfeeding may cause sickness in a feeble baby. The diet may not exceed the physiologic need, yet it may make great demands on an especial function, leading to its exhaustion.

A breast-fed baby with careless handling thrives better than a bottle baby with the most correct regimen.

GENERAL PATHOGENESIS

Most children are easily fed. A few show uncertain digestion and have a stormy infancy. Overfeeding leads to delayed assimilation, food-stagnation and bacterial decomposition. Sickness from relative underfeeding is seen in weakly children of the neuropathic exudative diathesis. With artificially-fed babies tolerance is easily exhausted, and cow's milk mixture with the exact chemical formula of human milk has a radically different effect and often injuriously affects a baby. The difficult digestion of casein is a myth, shown by the analysis of stools, but there is no doubt of the occasional injuries by fats and sugars. This reaction is not primary, but is secondary to fermentation of the whey. The whey of human milk, on the contrary, acts as a stimulant to digestion. The fat and albumen of cow's milk in whey will cause sickness, while if the same fat and albumen be mixed in human whey, the child will thrive.

The poor digestion of fats and sugars is due to pathologic fermentative processes, resulting in acid formation causing a local reaction on the intestinal mucosa, increased peristalsis and secretion of mucus. The child's health deteriorates and the protective processes of metabolism are lessened and poisonous substances formed.

Bacterial poison causes cell injury of the internal organs as well as of the intestinal tube.

Excessive atmospheric heat hinders heat evaporation and injures the working capacity of the cells; symptoms like sunstroke often appear, coma and hyperpyrexia, which latter is relieved by hydrotherapy. Artificially fed children do not resist heat well.

Contaminated milk was formerly held to be the most important and frequent cause of diarrheal disease, but intestinal fermentation is usually endogenous and will develop in a sterile food from primary digestive incapacity; the present theory is that within certain limits heat has a damaging effect on the child, reducing digestive tolerance so that food injuries easily occur. A bottle baby with disturbed nutrition has less heat resistance than a child which is nursed.

GENERAL SYMPTOMATOLOGY

More variety exists than was formerly supposed. The symptoms affect not only assimilation and tissue-building, but also internal metabolism and heat production and regulation, therefore the description should not be limited to vomiting and diarrhea. We observe albuminuria, cerebrospinal irritation, anomalies of heart and lungs, which often may be relieved by a change in diet.

All of us admire the fine color and beautiful contour of a healthy baby. It has normal stools and by the continual formation of antibodies effectively resists infections, which, if they do occur, run a benign course. It also has a breadth of tolerance for food, thriving on all sorts of mixtures and passes easily through the extreme heat of summer.

A baby with disturbed digestion shows emaciation, increased or diminished muscular tonicity, pallor, excitability or apathy, fluctuations in temperature and suffers a lowering of resistance against bacterial influence. Alimentary fever is often confused with infection; it may be distinguished by its course and its disappearance by a change in food.

Children with disturbed nutrition show a lessened tolerance for food. A healthy child with an increase in diet will react by a gain in weight. A sick child will show a paradoxical reaction. An increase of food aggravates its symptoms and causes a loss in weight.

Example.—Three children (1) healthy; (2) slightly disturbed; (3) severely disturbed. Thirty grams cane sugar added to twenty-

four-hour quantity of food for two or three days. Healthy child increased in weight. Second child slightly ill with mild fever, diarrhea and a stationary weight. Third child has a temperature of 100.5 F., violent diarrhea and lost several hundred grams.

This reaction to food is the measure of the reduced functional power. The more the patient is disturbed by small increases of food, the more serious is its condition. Such children are very sensitive to infections, heat, etc.

CLASSIFICATION: NUTRITIONAL DISTURBANCES

- Disturbances: Alimentation; various foods; injuries.
 Czerny: Disturbances: Infection; polluted milk.
 Disturbances: Congenital weakness of constitution.

Finkelstein:	Mild forms without destructive processes	{ (1) balance disturbance (2) dyspepsia
	Severe destructive processes	{ (3) decomposition (4) intoxication

Also, disturbances from inanition—feeding on gruels, proprietary foods; treatment by repeated periods of starvation.

Infection, heat, etc.

Whole question is of lessening of digestive tolerance.

Balance disturbance—lessened tolerance for fat in milk.

Symptoms: Insufficient gain in weight; firm stools.

Dyspepsia	Lowering of tolerance for fat of milk	{ Symptoms: Insufficient gain in weight; vomiting; diarrhea; sometimes constipation. Vomiting; diarrhea; colic; paradoxical reaction for food.
	Lowered tolerance for carbohydrates	

Decomposition Marasmus Atrophy	{ Severe damage for digestion of fats and carbohydrates	{ Abrupt loss in weight; subnormal temperature. Slow pulse; paradoxical reaction; strong reaction to heat and infection.
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Intoxication	{ A condition developing on the basis of dyspepsia and marasmus if large quantity of food containing whey and sugar is given.	{ Fever; cerebral disturbance; albuminuria; glycosuria, deep breathing, vomiting and diarrhea.
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BALANCE DISTURBANCE — CZERNY — SLIGHT MILK INJURY

Retarded development without marked symptoms of illness. The weight curve rises slowly, fluctuates and remains stationary or diminishes. The patient appears smaller than the normal child; has a poor color, flabby muscles, distended abdomen, irritable disposition; the stools are dry and gray (soap stools); it occasionally vomits; is easily infected.

Two types exist. 1. Fed chiefly milk.

2. Milk and carbohydrates.

The child may always show poor progress on milk mixture or the food may contain too much milk and too little carbohydrate.

The baby may do well for a while or may have a congenitally weak tolerance, or one reduced by an infectious illness.

Pathogenesis.—Cream is usually the injurious food element, and there is often rapid improvement on a diet poor in fat and rich in carbohydrate, which improvement is arrested by adding fat again. It is probable that the low digestive power for fats is secondary to weakening of intestinal function, through abnormal fermentation of sugar of milk; a rapid cure may often be effected by substituting another carbohydrate, dextri-maltose or a cereal gruel.

Sometimes an injured tolerance for carbohydrates causes the condition to advance to dyspepsia.

There is a change in metabolism, chiefly in fat absorption, the alkaline secretions of the intestine are increased and combined with fatty acids; there is increased secretion of mucus; the stools are of a whitish color; the bilirubin is changed to urobilin. To produce a soap stool the large intestine must be strongly alkaline—soap stools are not pathologic and may be observed with normal development.

Diagnosis.—If a baby with sufficient nourishment, 100 calories per kilo of weight per day, with no diarrhea or vomiting makes no progress, one may make a diagnosis of disturbed balance. The condition may be confused in children reduced in weight by illness, for convalescents have a greater need of nourishment, often taking 120 to 140 calories per kilo per day. One must also consider mild cases of decomposition in whom a change in food has stopped a diarrhea. These cases show a paradoxical reaction, dropping in weight if food is increased.

Lastly, there are anomalies of growth from unknown constitutional causes which defy the most skillful feeding.

Prognosis.—With appropriate dietetics the prognosis is very good. The number and quantity of meals should be regulated, the milk reduced and the carbohydrates increased. The type due to excess of milk is most easily treated by adding a little cereal gruel, a malt food or two carbohydrates in proportion of 2 to 5 per cent. of the total recipe. If one carbohydrate fails, try another; not more than five meals daily; quantity of food should be watched until the tolerance is known. Malt soup is often effective in obstinate cases.

Recipe for Malt Soup.—50.0 wheat flour, $\frac{1}{3}$ liter milk, malt extract 100.0, $\frac{2}{3}$ liter water. Mixed, cooked, strained.

Or buttermilk, clean and not too acid, mixed, with a flour-gruel and cane-sugar are excellent for younger babies.

Dextrimaltose may be considered constipating. From 2 to 3 per cent. of carbohydrates should be given. With proper dietetics, increased weight and good stools should soon occur. If malt-soup or buttermilk is given, after six or eight weeks an effort should be made to place the baby on a normal diet. A cure is established only when a suitable milk mixture agrees with the baby.

If the baby's condition still remains unsatisfactory in spite of carbohydrates, the outlook is less favorable; low carbohydrate toleration leads to fermentation. If all means of artificial feeding fail, breast milk is advised as in the treatment of dyspepsia.

DYSPEPSIA

Dyspepsia may be primary or a result of primary balance disturbance. The digestive capacity of the stomach is insufficient for the food given. There results pathologic fermentation, producing increased peristalsis and diarrhea — the chief symptoms of dyspepsia. The baby suffers from poor appetite, vomiting, slight motor insufficiency of the stomach, the free HCl is diminished, the volatile fatty acids increased, the abdomen is distended and there is colic. The stools are increased in number, are thin, contain mucus; the odor is foul and sometimes sour. The color of the stools is green; the bilirubin is oxydized to biliverdin. As a result there occurs the following: Fat soap stools; small white or yellow lumps, on warming, producing fat crystals; gruel (flour) stools are pasty and foamy; blue with iodine; red with erythrodermin. As to the question of undigested casein stool, little yellow lumps formerly supposed to be casein, are really fatty acids and bacteria.

Only after feeding raw milk will stiff cylinders of casein appear in the stools.

Formerly dyspepsia was divided into fat and starch (*Mehl*) dyspepsia. This classification is not justified for the appearance of undigested food elements in the stool does not prove that this element is the cause of the trouble. Thus, fat diarrhea does not come from primary insufficiency for fats, but from carbohydrate fermentation, causing increased peristalsis and fat in the stools. By withdrawing carbohydrates the fats can again be utilized.

Pathogenesis.—The local symptoms of dyspepsia are due to the increased production of fermentative acids from pathologic decomposition of carbohydrates, sugar and starch. Again, it may be emphasized that casein, so far from injuring the intestines, is most effective in combating an acid fermentation and exercises a curative influence.

The carbohydrates used in feeding have important differences as to fermentation. The sugar of milk ferments most easily; cane sugar

less so; dextrimaltose is slow to ferment, hence the apparent constipation following its use.

The tolerance of an intestine for carbohydrate as well as fats depends much on the fluid in which they are dissolved or suspended. The same quantity of sugar in undiluted whey will more quickly cause dyspepsia than the same amount in water or in diluted whey. As a cause of dyspepsia in bottle babies, whey, even if regarded as free from sugar, plays an important rôle.

We have an acute dyspepsia and chronic dyspepsia, a temporary or persistent lowering of digestive function.

Diagnosis.—The chief thing in diagnosis is to separate dyspepsia from mild enteral and paraenteral infections which may also cause an irritation of the intestines. It must be remembered that infections are often associated with disturbed nutrition. An infection is present when temperature and mucous stools continue in spite of reduction in carbohydrates.

The physician is cautioned not to regard intestinal irritation from an infection as being due to fermentation, and institute a long, unnecessary course of underfeeding.

A dyspepsia being diagnosed, the physician must consider whether he is dealing with a previously healthy child or with a marasmus patient. This is most important as to treatment and prognosis. The history should be reviewed; the first attack of diarrhea is probably dyspeptic; recurring diarrhea and loss of weight indicate that the digestive strength of the child has suffered, and point to marasmus.

Prognosis.—In a previously healthy child the prognosis is good; recurring attacks in very young babies, or in emaciated patients are most serious.

DIETETIC TREATMENT

The safest treatment in all forms of dyspepsia is human milk; it is most necessary to babies in the first weeks of life where artificial feeding is so dangerous. The dose of woman's milk should be generous, although at first caution is advisable.

In acute dyspepsia, in a fairly healthy patient where the cause of attack is a misproportion between the child's appetite and his digestive power, there should be a short period of fasting, with copious draughts of saccharin tea for six or twelve hours; only exceptionally for a longer period. Complete emptying of stomach and intestine is effected by lavage and irrigation, and laxatives. One-third of the usual amount of food, well diluted, should be given. The nourishment should be rapidly increased so that the period of underfeeding may be as short as possible.

As to the kind of food, the offending food elements, fats and carbohydrates, should be avoided; also the injurious effects of whey should be remembered. A thin gruel, mixed with skim milk, is advised, and later dextrimaltose may be added. No sugar of milk. Skim-milk and buttermilk are usually well digested.

This dietetic treatment causes a sudden fall in weight, later a stationary condition, normal temperature and fewer stools.

After a few days the child's tolerance for carbohydrates should be tested.

Many patients, however, do not respond to this treatment, and the diarrhea continues. These children either have an infection or are on the verge of marasmus. To continually underfeed these children is to kill them.

Finkelstein recommends the use of albumen milk for these cases.

Children with chronic dyspepsia do badly under continued under-feeding. There is no acute depression, but a chronic weakness of tolerance and the continual trauma of hunger is injurious. The best carbohydrate is 2 to 3 per cent. dextrimaltose. If no improvement in the stools occurs, use human milk or albumen milk.

DECOMPOSITION — ATROPHY — MARASMUS

The chief symptom is the loss in weight which sinks slowly, at times very suddenly. The baby is emaciated, has a senile appearance, the abdomen is distended, the complexion gray.

At first the child is excitable, crying and taking food greedily; later it is languid, apathetic. The inclination is very noticeable to slow, irregular heart action and subnormal temperature, which may be later associated with edema and cyanosis.

The urine contains no albumen or sugar. The stools are dyspeptic and frequent; sometimes fluid, sometimes solid. The firm stool indicates an arrest of the process. Often the feces contain a large amount of fat. Occasionally the feces are dark red or black from hemorrhage from a peptic duodenal ulcer.

Such patients are most sensitive, showing an immediate severe reaction to unsuitable food, infections, etc. A cold, bronchitis or hot weather may cause high fever and collapse; purulent infections of the skin, pyelitis and pneumonia indicate the diminished immunity of the child.

Decomposition in very young babies, if not quickly improved by diet, is soon fatal. With older children there are periods of remission and improvement. A not unusual ending is a fatal hemorrhage from a duodenal ulcer. Some children die with symptoms of alimentary intoxication. Death may be quite sudden, especially after a therapeutic

hunger period. A hopeful case may succumb quickly to an intercurrent infection.

Pathology, Metabolism.—It was formerly supposed that marasmus was an inanition due to destruction of the digestive glands from chronic inflammation, but the intestine of an atrophic child may show normal anatomic relations. Decomposition is probably due to extreme functional depression as shown by the paradoxical reaction in feeding. The more nourishment the baby swallows the more rapidly it emaciates. A sudden drop of a pound or two is due to loss of water and salt. The melting of tissues goes on more slowly.

The prognosis depends wholly on the dietetic treatment. If errors are avoided and the case is not seen too late, very bad cases may be cured; if one-third of the original weight is lost, the condition is hopeless.

Diagnosis.—We must distinguish marasmus from the symptomatic emaciation of tuberculosis and other cachexias and also from inanition. Mild forms of decomposition may be confused with balance disturbance or dyspepsia. The history of recurring diarrhea or rapid loss in weight is important. The final test is the behavior of the digestion in feeding. With a fairly strong food mixture a marasmus child will show fever, diarrhea and a drop in weight.

Treatment.—The cure of the decomposition is possible if the fermentation which causes the deterioration in digestion is removed and the tissues are supplied with water and salts. Often the physician commences treatment as he does in dyspepsia, with a hunger period and small and increasing doses of food, but a marasmus child cannot endure starvation and should, if possible, be nursed. The danger of hunger is exhaustively considered under "Inanition." If insufficient food is given, the digestive strength is ruined.

With human milk the subtle curative agent is in the whey, which is somewhat offset by the difficult digestion of fat and sugar. If one gives too much the baby is upset; if too little, it grows rapidly worse. Finkelstein gives the breast milk in a bottle, 200 to 300 grams daily in eight to ten feedings, with copious draughts of saccharin tea, without a hunger period, to avoid the fatigue of nursing. The milk is continually increased so that after a week the baby is taking from 100 to 130 or 150 calories per kilo of weight daily.

At first the child may grow paler and thinner, but not after the second week. Then follows a period when the general condition improves, but there is no gain in weight. No change in the wet-nurse should be made. We are dealing with a period of repair. Cells are constructed slowly with human milk, which is poor in albumen and salts.

In a month the improvement may be hastened by one meal daily of malt soup or of buttermilk, with a small amount of dextrimaltose.

A complete restoration to health will not occur earlier than two or three months; then ordinary food mixtures may be tried. It must be remembered that the child may have an idiosyncrasy against cow's milk.

If no human milk is available, mixtures of skim-milk or buttermilk diluted with a thin gruel may be used; often an increase in strength of the food will occasion a serious diarrhea. Finkelstein strongly recommends albumen milk in such cases.

The idea of albumen milk is to limit the dangerous acid fermentation by lessening the percentage of sugar of milk in the whey.

3.0 Albumen sweetened with saccharin. Maltose should be added in the proportion of not less than 3 per cent.

2.5 Fat; 450 calories per liter.

0.5 Sugar.

A great advantage of albumen milk is that sufficient food may be quickly given without danger of fermentation. The risk of underfeeding and inanition is thus avoided. The amount of albumen milk may be quickly increased to 180 or 200 gm. per kilo daily. Total daily quantity should not exceed a liter. After a day or two the stools are firm and fewer in number. The weight curve ceases to sink, becomes stationary and then increases.

With advanced marasmus, to evacuate the intestines quickly a six- to twelve-hour hunger period with tea may be advisable, followed by 200 to 300 grams a day of albumin milk in ten doses; the amount is quickly increased and the number of bottles diminished. If the fall in the weight curve is not arrested in eight days, dextrimaltose should be increased.

If this technic is followed, the number of failures is surprisingly small.

Lack of success is due to:

1. Too slow an increase, producing inanition.
2. Too late and insufficient carbohydrate.
3. Omitting carbohydrates if the stools do not improve.
4. Reducing or withdrawing carbohydrate if a recurrence of diarrhea, fever or of emaciation occur.

In only extreme conditions should these changes be made and only for a short time.

Feeding with albumen milk should continue in young babies ten weeks; older babies six to eight weeks. After this, ordinary food mixture may be digested. A cure may be considered complete when after a course of albumin milk, followed by a milk mixture, the child continues to thrive.

INTOXICATION

An alimentary intoxication is a general toxic condition in a child with disturbed digestion who is given food exceeding its digestive power. The lower its functional tolerance is, the smaller the amount of food is which will produce the intoxication.

Symptoms.—The first symptom is fever, whose alimentary nature is proven by the fact that the temperature quickly becomes normal if nourishment is withheld. With the mildest type there is a slight rise in temperature; with a severe attack the fever may be 104, 105, 106 F.

The alimentary fever may be the only symptom or may be associated with diarrhea; usually the patient loses weight, is languid and shows signs of kidney irritation. These early symptoms are immediately, or after one, two or three days, followed by extreme depression diarrhea, nervous symptoms, deep breathing, albumin and casts in the urine, and a serious drop in weight. At first the nervous condition is one of languor and somnolence. The child, if aroused, sinks immediately back into its lethargy. The face twitches, the eyes are sunken. In the worst cases the patient lies in a coma, or has general convulsions, transitory paralysis. Usually there is high fever, but a bad case of marasmus may show no feverish reaction.

The respiration is of a toxic type—deep, rapid, like a hunted animal. The stools are frequent; at first contain food débris, later are watery, greenish-yellow; are mixed with mucus. They become strongly alkaline from the abundant alkaline intestinal secretion.

Vomiting is often a serious symptom, recalling the clinical picture of the old-fashioned cholera infantum. The weight may sink from 500 to 1,000 grams in a few days. The skin is dry, shrunken, features drawn, fontanelles sunken; the muscles may be contracted and cramped.

The rapid loss of fluids quickly leads to collapse. The pulse is small, the heart weak, the extremities cold and cyanosed; the urine contains sugar from an alimentary glycosuria.

There may be a leukocytosis, reaching 30,000.

The great variety of symptoms show how many organs are involved. We may separate several different symptom-complexes—a comatose cholera form and cerebral type.

Pathologic anatomy.—The morbid changes are not extensive and do not correspond with the serious illness. In the stomach and intestines there is a serous or serohemorrhagic catarrh. Peyer's patches are swollen and hyperemic. Microscopically the wall of the gut shows a round cell infiltration.

Etiology.—The great similarity of an infantile alimentary intoxication to true cholera and cholera nostras of adults caused the older

physician to speak of cholera infantum and to ascribe the malady to an infectious origin, accusing contaminated milk. While such symptoms may occur in the course of parenteral and other infectious diseases, yet no pathogenic specific germ has been found to explain the symptom-complex. On the contrary, the most careful clinical observation shows that the condition is a food intoxication from exceeding the subject's digestive tolerance, and the origin of the disease is due to a decomposition of food. The use of polluted milk is therefore not essential, since the intoxication may originate from aseptic food.

The fact that food is the cause of the malady is proven by the effect of withdrawing food and giving water freely.

In all cases not complicated by an infection, the temperature at once falls; if food be taken again the fever returns. The fever-producing agents are the carbohydrates combined with whey constituents. If these substances are given in large quantities the fever is complicated by a toxemia. Large amounts of fat have also a toxic effect. This fat intoxication is, however, not primary, but is due to a sugar injury, which has severely damaged the function of metabolism.

Nature or Toxic Condition.—An intoxication is a result of an insufficient intermediate metabolism associated with an acidosis. The damage to carbohydrate metabolism shows itself by an alimentary glycosuria; this disturbance is increased by hunger and loss of alkalies by a diarrhea.

Formerly it was asserted that intestinal stagnation produced a toxic agent of bacterial origin, but experimental observations show that food crystalloids cause fever and poisoning by physical osmosis. The thickening of blood is also of great importance.

Diagnosis.—Alimentary fever is distinguished from infectious fever by a therapeutic test, withdrawing food or by reduction of the whey and sugar in the mixture.

In a food intoxication the temperature falls by crisis unless the system is fatally damaged. In marasmus the patient may die of hunger with no sign of detoxication. Pure infections or infectious complications are not freed from fever by hunger metabolism.

Finally, with a very well-nourished child the detoxication may be slow and incomplete. (No explanation.)

Prognosis.—The outlook depends less on the severity of the symptoms than on the duration of the toxemia and on the previous condition of the child.

Prompt and correct treatment will cure most patients quickly. Thus, a baby who sickens on sweetened buttermilk may have threatening symptoms and very rapidly improve. A long period of intoxication is naturally exhausting. A marasmus baby will often die without relief.

Treatment.—With bad toxic symptoms the complete withdrawal of food cannot be avoided. Fluids should be freely supplied in the form of saccharin tea. To relieve the loss of fluid, salt solution is recommended. It has the disadvantage of sometimes causing fever. (Denied.)

Lavage of the stomach and intestine is useful, but not indispensable.

Laxatives should not be used in diarrheal cases.

As stimulants, camphor, caffen, digitalin, adrenalin, cognac, are well spoken of. High temperatures are to be combated by hydrotherapy. With a cold skin, hot mustard baths are effective.

With favorable, uncomplicated cases the starvation will, in twenty-four to thirty-six hours, cause a complete disappearance of the intoxication. The stools are less frequent; the baby is thin but placid. Feeding must now be commenced, at first in small but increasing amounts. Fluids must literally be pushed into the patient.

Human milk is the best food. The earlier the child is rescued from inanition the better; on the other hand, over-feeding will quickly produce a relapse.

Human milk may at first be given in a bottle; later the baby may be nursed.

If artificial food be used, the injurious fats and sugars should be avoided. Skim-milk, buttermilk are useful. The first doses should be 5 gm., five to ten times a day. The dietetic treatment is similar to that in dyspepsia.

Albumin milk is much used by Finkelstein. It has no tendency to ferment and can be digested in large quantities.

An emphatic warning is given, that mixed forms of intoxication and marasmus babies do not endure starvation, and after a few hours on tea they should be fed either human milk or albumin milk.

NUTRITIONAL DISTURBANCES IN BABIES FROM LACK OF FOOD

We see frequently the clinical picture of inanition and its consequences in children who have been given insufficient food on account of primary intestinal disturbances. Healthy babies or those who are only slightly ill respond to a considerable reduction in food by constipation and a loss in weight. If the underfeeding is continued the patient will have subnormal temperature and slow pulse.

The similarity of this condition with decomposition is unmistakable. At first, hunger inanition is distinguished from decomposition in that an increase in food brings up the weight quite rapidly, but finally the organism is so weakened that digestive tolerance is much reduced. This is observed in the convalescence from severe spastic pyloric stenosis after vomiting has ceased.

If the patient is given large quantities of food, even of human milk, it may lose in weight—a severe paradoxical reaction. A child dying of pyloric spasm sinks into a terminal toxic coma as in marasmus.

More frequently one sees bad consequences of inanition in severe disturbance of nutrition. Here food has been diminished or withdrawn far too long a time and without knowledge of the great danger.

With such children hunger and underfeeding has a terrific effect—a drop in weight, slow pulse, subnormal temperature, collapse and even death.

There is often a permanent weakening of digestion, so that the amount of food that is assimilated before the hunger period will now cause serious symptoms. In a severe case a short period of starvation gives the patient a strong push downward. A baby grows steadily weaker with moderate underfeeding, while repeated hunger periods result in a hopeless condition.

It may, therefore, be laid down as a fundamental principle in the prophylaxis and therapeutics of disturbances of nutrition that starvation and underfeeding can indeed remove toxic symptoms and fermentation of alimentary origin, but they may also cause far-reaching damage, and that this starvation treatment must be reduced to the minimum of necessity.

QUALITATIVE INANITION

This is a form of malnutrition in which the nourishment lacks important food elements and has an insufficient caloric value; as a result, the vitality is seriously damaged and poor tissue is formed.

The most common type of inanition occurs in babies fed largely or wholly on starchy gruels. The gruel has been prescribed to relieve diarrhea and has been continued by the mother. For a while the body shows no signs of illness, and may gain in weight from the retention of water in the tissues by the carbohydrate diet. The flesh is soft and pasty and the patient may have a muscular hypertonia—latent tetany.

1. The atrophic type is seen where starch gruel has been given without salt. It is distinguished from starvation inanition by the dryness of the tissue and the hypertonicity of the muscles.

2. The hydremic form is observed where the gruel has been salted and the weight is increased by water retention. The face is pale and swollen, the flesh is flabby and often edematous, without kidney irritation. Occasionally great muscular rigidity may be seen.

The stools vary with the carbohydrates—are foamy, mucous, liquid or solid; sometimes the patient has a colitis.

Wide fluctuations in the weight curve are characteristic.

If the child has a slight infection the weight may drop from several grams to a kilo. Purulent skin infection, inflammation of the cornea, conjunctiva, pneumonia or pyelitis may show the lessened resistance to bacterial infection.

The disturbances from the exclusive use of starch gruels result from the lack of essential tissue-building food elements — fats, albumin, salts. A serious consequence is the lowered production of antibodies and reduced immunity.

Prognosis.—The younger the baby and the longer the incorrect feeding has been carried out, the worse is the outlook.

Prophylaxis.—Qualitative inanition is prevented by a sensible diet, when starchy diet is ordered for therapeutic reasons to relieve convulsive symptoms or stop a diarrhea. The danger of starch inanition should be remembered with spasmophilia. Albumin preparations, fats and salts may be used. With a diarrhea, gruels should be used for only a short time without adding milk.

Treatment.—With very young babies human milk is best at first in minute doses. In general, whole milk should be added to gruel. Finkelstein advises albumin milk. The gruel should be gradually reduced, lest a terrific drop in weight occur.

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