PROGRESS IN PEDIATRICS

DISTURBANCES OF NUTRITION OF THE ARTIFICIALLY FED INFANT WITH PRACTICAL APPLICATION OF THE CLASSIFICATION OF FINKELSTEIN

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In order to make the subject more comprehensible, I take the liberty of reviewing the etiology of nutritional disturbances as classified by Langstein and Meyer.

ETIOLOGY IN GENERAL OF NUTRITIONAL DISTURBANCES VARIETIES

1. Food qualitatively normal, but metabolism abnormal. Idiosyncrasy, as seen in (1) neuropathic infants; (2) exudative diathesis.
2. Milk infected with bacterial products due to the action on fats and sugars. (These products are not affected by sterilization.) Seen most in (a) summer, and (b) unhygienic milk.
3. Primary infection. Dysentery, typhoid, streptococcus enteritis, etc.
4. Secondary infection. Probably primary injury of the sensitive intestinal epithelium, a counterpart of the nephritis which is so frequently seen following colds, pharyngitis, otitis, pneumonia, pyelitis, cystitis, etc., in sick infants.
5. Inanition, as seen in (a) pyloric stenosis; (b) excessive starch feeding (*Mehl-nahrschaden*).
6. Overfeeding. Not necessarily based on an excessive fuel value of the ingested food, but frequently due to a preponderance of one or more food elements, either alone or in the presence of a vitiated digestive apparatus.

It is the latter problem, i. e., *overfeeding*, which we will discuss, referring to the previously named etiologic factors only in so far as they may directly have a bearing on the cases under discussion.

HISTORY

The history is of vast importance in all nutritional disturbances and worthy of the most careful consideration. The following should be carefully noted: the constitution of the child, with the presence of hereditary and developmental defects, a neuropathic history, or the presence of an exudative diathesis; lowered immunity, as evidenced by previous general, local, or gastro-intestinal infections; history of improper feeding; quality or quantity of individual feedings and total food taken, and the frequency and number of feedings, with notation of the resulting effect on weight and temperature curves. Briefly stated, in a normal child an increase of food results in increased weight, normal temperature, and normal

stools; while in nutritional disturbances increase of food results in decreased weight, increased temperature, and often abnormal stools, a paradoxical reaction.

The effects of the individual food elements in excess in nutritional disturbances are as follows: *Salts* cause a rise in temperature; *sugars* cause toxic symptoms, rise in temperature, and diarrhea; *fats* cause toxic symptoms, obstipation and usually fat stools (possibly diarrhea); *proteins* cause no corresponding findings. Results are not due to the individual ingredients, but to their relations to each other in a given food.

Based on the above etiologic factors and metabolic facts, Finkelstein has divided nutritional disturbances into the following types:

- I. Disturbed metabolic balance (Bilanz Störung).
- II. Dyspepsia.
- III. Intoxication.
- IV. Decomposition (acute atrophy).

Nutritional disturbances should not be considered as local intestinal lesions, but as general, deep-seated disturbances of the whole organism.

I. DISTURBED METABOLIC BALANCE (BILANZ-STÖRUNG — FINKELSTEIN;
MILCH-NÄHRSCHADEN — CZERNY-KELLER; ATROPHY
OF MODERATE DEGREE)

1. *Exogenic Factors*.—Improper nourishment (usually excess of fat in food). Under this head we will consider in detail the disturbances described by Czerny-Keller under the name of *Milch-Nährschaden* (nutritional disturbances due to excessive milk feeding), which usually arise during the course of feeding with ordinary milk mixtures, the clinical picture usually being due to the fat content, which may be relatively or absolutely in excess.

2. *Endogenic Factor*.—Congenital idiosyncrasy toward milk digestion. This is clinically characterized by

(1) *Weight*: Notwithstanding proper or even excessive caloric intake, there may be no gain in weight or an irregular increase, however, under the normal.

(2) *Temperature*: Varies as to the causative factor. Usually we find a daily oscillation from 1 to 2 degrees, with a tendency toward subnormal. However, in the presence of associated excessive carbohydrates there is an early hyperpyrexia due to the development of dyspepsia. This is especially true of infants under six months.

(3) *Stools*: In excessive milk feeding (*Milch-Nährschaden*) the common type is the fat soap stool, which is dry, light in color (gray to white), friable, and does not stick to the napkin. In the presence of excessive carbohydrates this stool may be lacking, due to the presence of a slight dyspepsia.

(4) Regurgitation and vomiting are frequent.

(5) Skin is pale, with loss of elasticity.

(6) Muscles are soft and flabby.

(7) Abdomen tympanitic.

(8) Child restless.

(9) Sleep disturbed.

(10) Immunity lessened, with resulting furunculosis, eczema and intertrigo.

(11) Urine usually shows no marked changes.

Metabolism.—A careful study of the relative intake and the excretion of the individual constituents of milk shows us that

(1) Protein digestion and retention are normal.

(2) Fat absorption, according to Freund, is normal. Other substances, which result in the formation of an alkaline reaction within the intestines, will lead to soap stool formation. A substance which does this is casein. This is indirectly shown by the feeding of casein to breast-fed children with dyspeptic stools, which become less frequent and alkaline. The watery stools seen in artificially fed children receiving whey are often quickly improved by adding casein to the diet (Finkelstein); and in well children by adding casein even in moderate quantities an increase in the alkali salts and calcium are seen in the stools.

Fat only in the presence of certain products of food, therefore, leads to the formation of fat soap stools. The presence of fat soap stools is of importance in showing that an excess of fatty acids have been combined with Ca and Mg, but it does not prove that the presence of these stools indicates at all times a pathologic loss of Ca and Mg, as it may only be a relative, and not an absolute, loss. The above illustrates the error of Czerny's conclusions that fats *per se* are the cause of fat soap stools, which Finkelstein claims are due to loss of salt balance. Therefore, the conclusion of Czerny that fat soap stools constitute a symptom of poor fat absorption, with loss of body fat as an end result, is an error. However, an analysis of the stool fat constituents, i. e., neutral fat, fatty acids and soaps, shows an abnormal relation which is of importance. Normally 18 per cent. of the total fat constituent in the stools consists of the insoluble Ca and Mg soaps, of which, in disturbed metabolic balance, approximately 48 per cent. are insoluble Ca and Mg soaps; therefore, it is evident that there is an excessive calcium and magnesium waste through the stools.

Paradox Weight Phenomenon.—Salts other than the calcium and magnesium do not seem to be markedly affected by this negative balance, evidenced by the presence of soap stools. Fat soap stools can only be viewed as of importance in disturbances of nutrition when the Ca balance is pathologically changed. The reversing of the Ca balance results in stationary weight. Nutritional disturbances due to excessive milk feeding are the result of too much earthy alkali being thrown off through the bowel and the organism being robbed of its acid-binding radical, and, therefore, the organism must call on the ammonia radical to neutralize the same. This is evidenced by the presence of the increase of the ammonia coefficient in the urine, i. e., the relation between the ammonia and the total nitrogen products. This phenomenon, discovered by the Czerny school, is especially marked in intoxication and decomposition. The above findings apply to disturbed metabolic balance described by Czerny under the title of *Milch-Nährschaden*. Much less is known of the metabolic changes in the nutritional disturbances due to excessive flour feeding (*Mehl-Nährschaden*). Weigert believed in the latter, that the organism becomes richer in water and salts and poorer in the dry substances. Of the metabolic changes due to infection nothing is known.

DIAGNOSIS

The diagnosis of disturbed metabolic balance is based on the history of

1. Food of sufficient caloric value.
2. No gain in weight.
3. Slight variations in temperature.
4. Absence of acute symptoms, but a general loss of turgor.

To condense descriptions of stools and urine, the following characters are used:

STOOLS: | = normal; / = soft; — = quite soft; m = watery; [] = fat soap; x = mucus; c = curds; bl. = Blood.

URINE: A = albumen; S = sugar; Ac. = acetone; D = diazo; I = indican; C = casts; P = pus; Bl = Blood.

PROGNOSIS

The *prognosis* is usually good, even in the artificially fed.

TREATMENT

Change the causative diet. Reduce the quantity of milk and add carbohydrates, or substitute milk low in fats, usually skim- or buttermilk, and add malt or other easily assimilable sugars. In young infants human milk is by far the best form of feeding. The good results of treatment are due to the fact that the tolerance for carbohydrates is not reduced.

1. *Diet With Human Milk.*—This is by all means the best treatment, especially in young infants. Weight increase may be slow at first, probably due to low salt and protein content of human milk. A loss of more than 6 to 10 ounces over a period of three or four days is frequent. More than this should lead one to suspect an error in diagnosis. This loss may be due, as stated, to stopping of a food rich in proteins and salts, and substituting one low in the same with the continued excretion of water through the kidneys, bowels, etc. This stage is passed in about four days and the system adapts itself to the new ingredients. Other symptoms which must not lead to error are paleness, loss of turgor and sinking cheeks which soon disappear with beginning weight increase. Temperature and pulse do not change, and the stools assume a breast-milk character. If the stage of reparation is slow, and the child does not gain in weight, the substitution of one meal of food rich in protein and salts daily will frequently help (buttermilk or skim-milk). Mother's milk also helps to increase immunity.

2. *Diet with Artificial Foods.*—In the pathogenesis of feeding of milk, fat plays the most important rôle, and this is best counteracted by replacing it by the well-tolerated carbohydrates. In simple cases reduce the quantity of milk and add carbohydrates, estimating the form of sugar and starches, and if this does not answer, or if the quantity of milk in the previous diet was not too great, then change the kind of sugar. The following answer in most cases: (1) Soxhlet's *Nahrzucker* (nutritional sugar). (2) Cane sugar. (3) Mellin's food. (4) Horlick's malt food.

SEVERER CASES

1. Malt soup (Keller) (laxative), made as follows, is exceedingly valuable:

(1) Milk 11 ounces warmed gradually; add flour 1½ ounces; stir constantly, then pour through gauze filter; (2) malt extract (Trommer's, etc., will answer)

3 ounces; boiled water, 22 ounces; potassium carbonate solution, 10 per cent., $\frac{1}{8}$ ounce; (3) mix both solutions. Put on slow fire, stir continually, and boil for two or three minutes. Caloric value 800 to the liter. Formula: fat, 0.5 per cent.; protein, 2 per cent.; carbohydrate, 12 per cent. This can, at first, be diluted, if indicated. This food should usually be changed after two to four weeks. Loefflund's and Borchardt's Malt-Soup Extracts do not require the addition of the potassium carbonate solution.

It is indicated in the presence of fat soap stools which soon become pasty and of a mahogany brown color; best between 3 and 6 months; after 6 months add more milk to increase the protein. It is contraindicated below 3 months, if the stools are loose.

2. Buttermilk and skim-milk plus sugar and flour (two carbohydrates). (Antilaxative; action both the same.)

- (1) Indicated especially in infants under 3 months.
- (2) Valuable over 6 months because of high albumin content.
- (3) In convalescence they fill the needs of albumin hunger.
- (4) Antilaxative because proteins cause a rich intestinal secretion, which results in putrefaction, which tends to constipation.
- (5) Contraindicated in fat soap stools (less valuable than malt-soup in clearing them up).

Human milk diet may cause an early loss of weight because of low salt and protein. Cow's milk diet causes an early, rapid gain in weight due to fixing water in the tissues by increased salts and carbohydrates. Fat rich food causes loss of earthy alkali salts through bowel, resulting in soap building and loss of weight by loss of body salts. This must be counteracted by carbohydrates which increase weight by allowing the organism to use more salts. The early rapid increase is later equalized by the organism and soon becomes less rapid.

General symptoms following change of diet are, better sleep; improved turgor; skin less pale; less variation in temperature. Stools change from soap stools to (1) buttermilk, yellow-brown, alkaline, and fair consistency; (2) malt-soup, acid, softer, mahogany-brown color.

The above changes are due to the protein and carbohydrate constituents.

The results of treatment are due to the fact that the tolerance for carbohydrates is not lessened. Study each case to see if excess of carbohydrates is not being given which is indicated by (a) restlessness; (b) stopping of weight increase after early rise; (c) increase in number, acidity, and amount of mucus in stools; (d) alimentary fever (irregular). If there is irregular fever, human milk must be substituted.

The above mixtures should be gradually replaced by milk-mixtures after two to eight weeks.

OTHER THAN ORDINARY TYPES OF DISTURBED METABOLIC BALANCE

1. The ordinary type, already described (*Milch-Nährschaden*), due to excess of fat.

2. Infections, as otitis, pharyngitis, cystitis, etc. This form readily yields to the same treatment as 1 after cessation of infection.

3. Excessive carbohydrate nourishment (*Mehl-Nährschaden*) is only seen in older children with broader carbohydrate tolerance; in young infants dyspepsia is an early result with excessive carbohydrate feeding.

Treatment consists in administering milk mixtures, i. e., foods high in fat.

II. DYSPEPSIA (STADIUM DYSPEPTICUM OR STAGE DYSPEPSIA)

In practice, especially in young infants, frequently we do not see the stage of disturbed metabolic balance, but see dyspepsia directly, due to an excess of sugar in the food. This can be likened to an excess in whey components when milk is the food used. Fats, unless in excess, probably only play a secondary rôle due to the presence of sugar fermentation. This is characterized clinically by acute gastro-intestinal symptoms, the most marked of which are the stools, which are increased in number and of an abnormal quality. The organism does not show signs of any deep-seated general changes; weight loss is moderate or remains constant, temperature is moderately increased and repair is rapid with the withdrawal of the improper food.

The diagnosis can only be made by a careful consideration of the history, the clinical and functional symptoms, and only that picture of gastro-intestinal findings which developed primarily in a healthy infant or follows directly the stage of disturbed metabolic balance can be diagnosed as dyspepsia and treated as such; otherwise cases of intoxication and decomposition may be neglected.

ETIOLOGY

1. Congenital lowered tolerance to cow's milk.
2. Errors in diet with milk of good quality. (a) Overfeeding with diet of normal proportions (too frequent and too much). (b) Feeding with a diet of improper proportions (excess of sugar, etc.).
3. Feeding with infected milk.
4. Infection of the gastro-intestinal tract (external infections).
5. Systemic infections (otitis, pharyngitis, etc.) associated constantly with a lessened tolerance for food.

SYMPTOMS

Symptoms in General.—Severe symptoms are absent. The mind is clear. The heart action is not rapid. Respirations are not greatly increased. The skin remains unaltered.

Weight.—If there is an excessive sugar feeding in the treatment of a preceding disturbed metabolic balance, we at first get an increase in weight. This ceases later, and dyspepsia develops with moderate weight loss.

The temperature is irregular and moderately increased, and, when it follows diets rich in sugar and salts, is of the alimentary type.

Gastro-Intestinal Symptoms.—The appetite is poor. The mucous membrane of the mouth is red and may be the seat of thrush (due to decreased immunity). Vomiting may be present and usually occurs long after feeding. The stomach may contain food seven to eight hours after feeding, which contains volatile fatty acids (odor), but no free HCl. The abdomen is distended and peristalsis increased (auscultation). Restlessness is marked, probably due to abdominal findings.

Stools.—The clinical diagnosis is usually made from the stools. They are increased in number and watery, and the reaction is usually acid.

The color is green, mucus is present, and curds composed of fatty acids, not casein, are present. The number is more important than the quality, as microscopic examination gives us little etiologic or therapeutical enlightenment, and we cannot get a very definite idea of the quantitative chemical contents.

Bacteria specific for dyspepsia are unknown.

Urine.—Sugar, albumin and casts are absent, unless there is a tendency toward intoxication.

Fig. 2.—Chart showing hospital record of an infant with dyspepsia.

METABOLISM

Changes in part are due to (1) peristalsis; (2) to increased intestinal secretion, which latter accounts for the moderate increase in the protein matter in the stools, and not, as formerly thought, to decreased digestion of the food proteins (casein, etc.). The remainder of the curds is due to fatty-acid salts and bacteria. Fat absorption is usually only slightly reduced. Fat diarrhea is not frequent and only seen in fat-rich foods.

Soaps and neutral fats are in about normal relation to the total fat content. Volatile fatty acids formed from carbohydrates and fats by bacterial action are increased. Sugar is absent. Starch varies directly with the peristalsis. Salts in stools are increased. Green color is usually due to oxidation of bile constituents.

In general, protein retention need not suffer. Ash balance may remain normal. The water in the stools is increased, and in the urine is decreased.

PATHOGENESIS

Sugar and salts, which in the normal child cause no symptoms, in the impaired organism, or in excess, can cause dyspepsia, as evidenced by diarrhea and fever.

Pyrogenic Action of Food Constituents. Action of Salts.—Sodium is the most important salt in raising temperature. Meyer gave a normal child 100 c.c. 10 per cent. NaCl solution per mouth, with usually no result, but in infants with gastro-intestinal symptoms the temperature was raised. Potassium is less important in raising temperature. Calcium rather tends to lower temperature. The mode of action is unknown.

Sugar.—Milk-sugar is more active than malt-sugar in raising temperature, but sugars are less constant experimentally than salts. Whether due to sugar itself or products of fermentation is also unknown. *Proteins* cause no rise in temperature and tend to improve stools. *Fats* have an uncertain and probably indirect action. (They seem to add to the bad effect in the presence of sugar and salt disturbances which, however, cease when the latter are withdrawn.)

The dyspeptic picture is due to (1) slight intestinal epithelial injury following various causes; (2) the administration of certain foods: (a) whey and sugar, which are the most important, and (b) fats, of less importance. These must be administered in quantities large enough to affect the injured epithelium and further impair its function, but not sufficient to cause intoxication.

PROGNOSIS

This is good with proper diet. It is bad with improper diet, with tendency to intoxication and decomposition, varying indirectly with the age of the infant and directly with the previous number of attacks and tendency to recurrence.

TREATMENT

Human Milk.—The younger the infant, the more indication there is for human milk.

Artificial Food.—Remember that primarily the sugar and whey content of the food, and, secondarily, the fats are the irritants.

1. In mild cases reduce the milk to a minimum, and especially the sugar content.

2. In the severer cases reduce the sugar, whey and fats. It is best temporarily to withdraw food entirely as follows:

(1) Starvation (or hunger) for one day (tea and saccharin).

(2) Indifferent diet for second day (low in food value and salts); i. e., Young, one-third milk (best boiled) plus two-thirds thin oatmeal gruel, without sugar. In severe cases, one-third skim milk plus two-thirds thin oatmeal gruel without sugar. Buttermilk may be used in place of milk. The quantity on the second day should not exceed 6 to 8 ounces, as follows: five feedings of 1 to 1½ ounces of food, and 20 to 25 ounces of tea, plus saccharin, given *ad libitum*, giving a total of about one quart of fluids. This will usually answer. Further treatment depends on the reaction to the above; if the general appearance improves, also the disposition, etc., and the weight loss ceases in two or three days (which is the case even in under-feeding), then the treatment was correct, otherwise decomposition must be thought of.

(3) When the weight-curve is constant, the quantity should be increased, but the quality left unchanged; 5×2 ounces, etc., of the same mixture, plus water or

tea to the necessary quantity between the feedings. Weight increase should not be expected because of the low sugar content, but a decrease in weight should always be considered serious.

The stools are, at first, small and contain mucus; later less frequent, and often on milk mixtures without sugar there are fat soap stools which are a good indication.

3. If the temperature and stools are normal after three to five days, carbohydrates can again be added, best in the form of malt preparations (avoid milk-sugar because of its rapid fermentation), beginning with 1 per cent., and gradually increasing to 5 per cent. At first, there is a rapid increase in weight; later a slower one.

Avoid under-feeding too long, even if the stools look bad, if the temperature and weight curves improve, because of the danger of decomposition. Fats can be added in place of sugars, but this should be done with care. (Experiments with cod-liver oil have given me the best results.)

ALBUMIN MILK¹

The albumin milk of Finkelstein is distinctly indicated in these cases.

Direction for Making: To 1 quart of milk previously boiled for five minutes in a double boiler, add 1 tablespoonful of chymogen (elixir rennet, junket tablets, dessert-spoonful of pepsin or tablespoonful of essence of pepsin). The dish containing the milk is now placed in a water bath, and the milk is heated to 107° F.

1. Directions for making, recently obtained from the Kinder-Asyl diet kitchen, are as follows: To 1 quart of raw milk add 1 tablespoonful of essence of pepsin (elixir rennet, junket tablets, dessertspoonful of pepsin or chymogen) and stir for one-half minute. The mixture of milk and ferment is now kept at 100 to 107 F. and perfectly quiet for about fifteen minutes, when the clot is formed. The free whey is now poured off and the remaining curd is put into a thin muslin bag, or one made of four layers of gauze (if too thick it drips too slowly or vice versa, curds will pass through and some of the curd is lost), and left to drip for one hour. The curd is now placed in a fine hair sieve and rubbed through with a potato masher, adding buttermilk, one pint, to facilitate this process. The passing through the sieve should be repeated until the curd is minutely divided.

Now, as a final process, water to make a total mixture of 1 quart and 1 per cent. of malt-sugar are added.

This is now put into a receptacle resembling a small ice-cream freezer and put on the stove to boil for ten minutes. While boiling, the handle is vigorously turned, stirring the milk inside thoroughly and constantly (stirring with an egg-beater if the above is not obtainable will answer; the former is to be recommended as it also prevents evaporation. I find that stirring with a spoon does not cause agitation enough to prevent large curd formation.)

Churning is continuously kept up for two reasons: 1. To make the curd finer. 2. To get the suspension more homogeneous. After ten minutes of boiling, the kettle is placed in a bath of cold water to cool it off, still turning the crank for a few minutes until the milk is cool. Then it is put into bottles. Overheating for feeding ruins the product; 100 F. does not change it.

[NOTE: With all due courtesy to the author we wish to state that the first method of preparation was adopted in our diet kitchen, because of the indefiniteness of the details in its preparation as first published and the consequent difficulties encountered in attempting to get a fine curd.]

for thirty to thirty-five minutes. It is now put aside for fifteen minutes, then the excess of whey is poured off and the remaining curd is put into a thin muslin bag, or one made of four layers of gauze (if too thick, it drips too slowly, or if vice versa, curds will pass through and some of the curd is lost), and left to drip for two hours. The curd is now placed in a fine hair sieve, any further free whey drained off and then rubbed through with a potato masher, adding buttermilk 1 pint to facilitate this process. (The buttermilk used is made from whole milk heated to 180 F. for forty minutes, then cooled to 80 F. The scartaline is now added, and after standing from eighteen to twenty hours at the latter temperature it is churned to remove the butter-fat.) The passing through the sieve should be repeated until the curd is minutely divided. Now, as a final process, boiled water to 1 quart is added, and 1 per cent. of malt sugar is added. Sohxlet's Nährzucker is used for my cases, although Mellin's food or Horlick's malt food answers the purpose. Therefore, the finished product contains the curd from 1 quart of boiled milk and 1 pint of boiled buttermilk, 1 pint of boiled water, and 1 per cent. malt sugar.

I have found that rubbing the curd through the sieve with buttermilk prevents the freeing of fat from the curd, so frequently seen when water is used. Boiling the milk, although freeing somewhat less whey, makes only a moderate difference in the quality of the end product, and in our cases has been much better taken by the infants than that made from whole raw milk with its heavier curd which requires shaking during feeding.

Albumin milk prevents abnormal fermentation in the intestinal tract on account of (1) the low content of easily fermentable milk sugar; (2) the low content of whey salts (the latter favor fermentation), and (3) the comparatively high protein content (the latter counteracts fermentation). On standing, a precipitate is likely to form in the bottle containing the albumin milk. Before feeding, the bottle should be well shaken. The quantity to be fed should be warmed to the body temperature and again shaken. Overheating is objectionable. The milk should be kept in a cool place. If thus taken care of, it preserves its quality well, for at least five weeks.

Albumin milk contains 3 per cent. protein, 2.5 per cent. fat, from 1 to 1.5 per cent. of milk sugar, 0.5 per cent. of mineral salts. The mixture contains 450 calories to each liter (Finkelstein). My own tests show the fat content somewhat lower, averaging about 2.2 per cent.

Directions for Use.—Albumin milk is indicated in all infantile disorders which are associated with diarrheas, especially in those cases in which breast milk is not obtainable. It is also valuable in cases of dyspepsia, entero-catarrh, cholera infantum (intoxication, Finkelstein), acute atrophy (decomposition, Finkelstein).

In cases even where the bowel movements continue thin and frequent, or contain mucus, one should proceed to make a daily increase in the quantity of albumin-milk, and after a lapse of eight days begin to add more sugar, or even sooner if the stools warrant it, gradually increasing the dextrin-maltose to a total of 5 per cent. If the weight still remains at a standstill, 2 per cent. of flour may be added. It sometimes happens during the period of convalescence, after improvement has once set in, that an apparently unfavorable condition occurs, characterized by frequent and thin stools. In these cases it may be said with great emphasis that one should not diminish the quantity of the albumin-milk or the sugar, but should continue with the same dosage and quietly wait. After

a short time, if this suggestion is followed, improvement usually sets in; however, when a profuse diarrhea is associated with a marked drop in weight, the sugar must be reduced.

In some of these cases the disturbance is not primarily a digestive one, but it seems to be associated with an infection in some other part of the body. Thus, for example, an intercurrent rhinitis, or bronchitis, will almost invariably cause a standstill or loss in weight, with more or less pronounced digestive disturbance.

Management in Detail.—In cases of dyspepsia, feed the baby tea sweetened with saccharin for six hours. Begin by feeding 300 gm. (10 ounces) of albumin milk, divided into five or six feedings. Tea and saccharin may be given between feedings, as seems indicated. This can be gradually increased by adding 50 to 100 gm. daily until 180 to 200 gm. per kilogram body weight are given. Carbohydrates should be added as previously stated. Children will usually require from four to eight weeks of treatment with albumin-milk. After this time appropriate milk mixtures should be tried. The change from albumin-milk to other mixtures must be made quickly and completely, all bottles being stopped at one time. Relapses are an indication for a return to albumin-milk.

(For further details for its use see intoxication and decomposition.)

Other Treatment.—1. Avoid laxatives (castor oil, calomel, etc.), except for an initial dose. 2. Avoid gastric lavage, at least in early cases. 3. Warm compresses to abdomen aid in removing flatulence, etc. 4. Astringents (tannigen, tannalbin, bismuth subnitrate) are indicated after the weight-curve is steady, temperature normal, and the child generally improved, but the stools still frequent.

III. INTOXICATION (ENTERO-CATARRH; CHOLERA INFANTUM)

This is characterized by violent gastro-intestinal symptoms, rapid loss in weight and other symptoms of deep-seated alterations in the functions of the organism. Collapse and nervous symptoms outweigh the intestinal symptoms. On these findings Finkelstein bases his nomenclature rather than on the intestinal findings, as in previous classifications.

ETIOLOGY

The causative factors are the same as for dyspepsia, i. e., excess of sugar and whey content in the food, but here we have a more severe picture. All factors which cause nutritional disturbances can cause intoxication, but the important point is, which moment in a nutritional disturbance leads to the phase of intoxication?

Factors in a Nutritional Disturbance Which Tend to Intoxication.—

1. *Food.* (a) Combination of food elements which individually would be insufficient to cause a disturbance. (b) Infected food, i. e., spoiled milk. 2. *Infection.* (a) Gastro-intestinal infection. (b) Systemic infections, as otitis, cystitis, pharyngitis, etc., by disturbing the digestive function. 3. *Heat.* Heat can cause a chain of symptoms in itself resembling intoxication. All depends on the individual resistance, a more severe irritant being necessary in the disturbed metabolic balance, and dyspepsia, than in atrophy to develop the intoxication stage.

Carefully following nutritional disturbances teaches us that a definite constituency in the food leads to the various phases, e. g.:

1. Intoxication follows diet rich in sugar and whey content.
2. Decomposition (atrophy) follows diet rich in fat and relatively poor in sugar. These facts are so definite that one can prophesy which variety will prevail in a clinic by knowing the diet commonly in use (Finkelstein).

To develop intoxication there must be an injured intestinal epithelium or preexisting nutritional disturbance. Enteric and systemic infectious processes, as well as heat, give rise to intoxication through their action on the cells of the digestive apparatus in the intestines and the secretory digestive organs. Czerny and Keller believe much trouble is due to infected milk, not to the bacteria themselves, but their acids (toxins formed from fats and sugars by them).

SYMPTOMS

1. High temperature.
2. Rapid loss in weight, even 1 to 2 pounds in a few days, mainly due to loss of water. The skin becomes dry and inelastic.
3. The stools are liquid, are usually numerous and contain mucus and occasionally blood.
4. Collapse. The skin is gray in hue, eyes sunken with distant stare; pinched nose; also frequently incoördinate movements of the extremities, slow and often limited to one extremity; also catatonia of the extremities, and aimless movements, as of chewing, etc.
5. Typical respirations (deep, rapid without pause); described as "toxic respirations."
6. Glycosuria is almost constant, and the sugar is of the same variety as that in food. The phenyl-hydrazin test is the best as the copper sulphate tests require long boiling with lactose, etc., and the reaction may be overlooked.
7. The urine contains albumin and casts. The amount of urine is small, even to anuria.
8. Leukocytosis is present up to 30,000.
9. The heart action is weak, the pulse small and irregular.
10. Vomiting is frequent and the vomitus may contain blood.
11. Nervous symptoms may be pronounced and cause confusion with meningitis. The sensorium is disturbed (with an occasional cry as if in pain). The hydrocephaloid state may be present with strabismus, convulsions, etc. Whether uremia is a factor in this condition is very questionable.
12. Sclerema is constantly seen in the severer types, a very bad sign, due to a coagulation of tissue fluids of an unknown nature (Czerny-Keller).

METABOLISM

Clinical findings lead one to expect marked metabolic changes.

Protein.—Feces.—Nitrogen quota increased, probably due to increased peristalsis and intestinal secretion. Experiments are insufficient.

Urine.—There is a marked increase over the normal under same conditions as to food. During instituted hunger-day the albumin metabolism still progresses and is probably due to toxic processes. Changes from the normal end-products of protein metabolism are present. The ammonia coefficient is increased from 10 to 40 or 50 per cent. (Langstein and Meyer).

1. Change in amino-acids to urea (experiments with glycocoll-amino acetic acid) are interfered with and some excreted as glycocoll in the urine, probably due to a liver insufficiency (disturbance in synthetic production of urea) (Pfaundler). This theory of Pfaundler can also apply to the increased ammonia

coefficient other factors which probably positively cause increased ammonia coefficient.

2. Removal through the intestinal tract of Ca and Mg to neutralize the temporary fatty acids, and necessitating the utilizing of ammonia to neutralize the normally formed acids in the urine.

3. The acids (acetone and aceto-acetic acid) formed during the starvation period (toxic) must be neutralized in the urine.

4. Due to interference with carbohydrate metabolism, the fats are not completely burned up in the system by sugar, and as a consequence the acids are combined with alkalies Na and K, and as these are not sufficient the excess is combined with ammonia in place of being thrown out as urea (Rosenstern). This is a typical diabetic reaction (Rosenstern).

Fat.—Meyer found absorption reduced from 97 per cent. to 60 per cent. This improves early in convalescence. Splitting of fat is bad and we get an acidosis with the presence of acetone and aceto-acetic acids (butyric acids, etc., Rosenstern).

Carbohydrate.—Sugars of the type ingested are seen in the urine long before the end of the proper assimilation period. This ceases when the sugar is withdrawn from the diet.

1. With milk sugar in the diet we get milk sugar and galactose in the urine, while other components, as glucose, are missing, on account of the fact that part of the milk sugar is absorbed unchanged, and as it cannot be used as such in the system, it is again excreted, probably due to the injured epithelium allowing it to pass through, and not to the absence of the lactose ferment which Langstein and Steinitz always found present.

The presence of galactose is due to a different cause, and indicates that there is an insufficiency in the sugar oxidation function, and the system is only able to burn up the glucose. This is a liver insufficiency. Other disaccharids have the same ending. Maltose differs from these only in that it can be burned in the system as such without previous splitting in the bowel; therefore, its presence in the urine is double evidence of a lowered power of oxidization by the organism; that is, in its intermediary metabolism.

Water.—Great loss in weight is probably due to the loss of water. Water is lost in the urine and stools which, however, together have been found to equal about the normal amount, only the proportion is inverted, the bowels excreting the greater portion. Loss in body weight is, therefore, in all probability, due to excessive loss (pathologic) through the skin and lungs. Respiratory experiments must prove or repudiate this.

Salts.—(No good experiments to date). Excesses of salts are excreted in the stool, especially the chlorids. There is a negative balance of Cl. It is questionable whether the salt loss is the primary cause of the water loss, or whether the loss of water by the system has its own pathogenesis; indeed, it would require a great salt loss to account for the great loss of water, and it is quite possible that a toxic influence results in a disturbance of the water-binding property.

RELATION OF INTOXICATION TO FOOD ELEMENTS (FINKELSTEIN)

We are able with regularity in dyspepsias to produce intoxication by continuance of the carbohydrate and whey-rich foods; also in light cases

to improve by withdrawing carbohydrates alone; in severer cases by withdrawing both carbohydrates and whey elements.

INTOXICATION IN COURSE OF INFECTIONS

Pathogenesis is not due to a single factor. We know that some are influenced by food withdrawal; others are not. 1. We must believe that the infection injures the intestinal wall and other digestive organs. 2. From this point pathogenesis is the same as in alimentary intoxication. The toxic influences of different infections are as different as are the infections.

PATHOLOGY

In the small intestine there are usually no marked changes. Hyperemia of the mucous membrane and enlarged follicles, especially Peyer's patches, are usually present. Liver, cloudy swelling and fatty degeneration (probable causes of insufficiency). Rott and Tugendreich have believed that an acidity (*Saurung*) of the liver and muscle tissue is present.

DIAGNOSIS

The diagnosis is based on the above symptoms, with improvement on withdrawal of food for twenty-four to forty-eight hours.

Fig. 4.—Infant with intoxication whose hospital record is shown in the chart Fig. 3.

PROGNOSIS

This depends much on reaction to hunger diet, as very severe symptoms disappear often in twenty-four to forty-eight hours. If the same do not disappear in twenty-four to forty-eight hours, in the absence of infection the prognosis is bad. Infections add to the severity. They are bad as a complication of chronic nutritional disturbances.

TREATMENT

1. Removal of all food with sufficient water administration.
2. In severer types, subcutaneous salt infusions twice daily, 100 to 150 c.c., even though there is danger of salt fever in the severer types.
3. Salines per rectum, best administered by the drop method. One drop per second for four hours is 400 c.c. One-half strength of the following solution is good:

	gm. or c.c.
NaCl	7.5
KCl	0.1
CaCl	0.2
Water	1000.0

4. One lavage, if food has been given shortly before.
5. Avoid all laxatives, as the bowels empty themselves; also avoid astringents.
6. Analeptics. Give a mustard bath; reddening of the skin is a good sign.
7. Antipyretics. Use cold packs, and leave the infant undressed.
8. Stimulants. Caffein sodium benzoate, gr. $\frac{1}{10}$ to $\frac{1}{2}$, four or five times a day; camphorated oil, 1 c.c. every two hours, if indicated; epinephrin solution, 0.6 in 1,000 c.c. of salt infusion; veronal, gr. $\frac{1}{10}$ per dose, is less depressing than chloral.
9. Sedatives for convulsions, etc.
10. Diet: Hunger diet should be employed not longer than twenty-four to forty-eight hours. Occasional administration of dilute saline solutions (NaCl 5.0, NaHCO_3 5.0, water 1,000), per mouth, also vegetable soups, Moro's carrot soup,² Eichel kaffe or thin soya-bean gruel, may soon be used with care, to supplement an occasional tea feeding.

Human Milk.—Human milk is by all means the best food. Feed often and in small amounts, ten times daily, 5 c.c. from bottle or spoon. The first milk should be fat-free. Increase when the temperature, etc., do not react to food, and then not more than 50 c.c. daily.

A sustaining diet should be reached in eight to ten days (32 calories per pound), after which the child can be put to the breast five times daily. If the elevation of temperature returns, except in the presence of infection, cut down the food. The gain in weight is very slow in the stage of repair on human milk, due to the low protein and salt content.

Cow's Milk.—For the first few days after the hunger diet, a food low in fat and sugar should be fed because of the lowered tolerance. One-half skim-milk or buttermilk mixture, without sugar, will answer, but should be fed in small quantities, 10×5 c.c. daily, then 10×10 c.c. On this low diet weight loss and temperature will usually stop. After ten to fourteen days, 32 calories per pound may be fed. Less rapid increase than in human-milk feeding should be the rule. Older infants can be given gruels in their milk. Recurrence of dyspepsia is an indication for return to indifferent food. With a second recurrence breast milk is absolutely indicated. Return to carbohydrates should be made with great care. Albumin milk is indicated in this condition, and the following is a good working rule for its use in these cases: First day, tea. Second day, 10×5 c.c. albumin-milk, with tea *ad libitum*. Then increase 50 c.c. daily until stools are good, then increase 100 c.c. daily until 180 to 200 c.c. is given daily for each kilo weight. After the stools are solid, add 1 per cent. of sugar to the food, and increase gradually to 4 per cent. In intoxication I have obtained better results by at first feeding albumin milk without sugar additions. In infants over 6 months 1 per cent. of flour can also be added. After six to eight weeks, an ordinary milk mixture can be fed. Feedings can now be reduced to five or six daily. Never feed over 1,000 c.c. a day of albumin milk.

2. *Moro's Carrot Soup.*—One pound carrots are peeled, sliced and cooked one to two hours, and after mashing, poured through a sieve and mixed with the bouillon made from a pound of beef and 1 liter water. To the latter add one teaspoonful of salt.

IV. DECOMPOSITION (ATROPHY: SEVERE TYPE; MILCH-NÄHRSCHADEN: SEVERE TYPE)

Bilanz-Störung may be the direct forerunner of decomposition if the dietetic error is not corrected.

Dyspepsia leads to decomposition if fat predominates in the food and sugar is not present in excess; otherwise we get intoxication. Czerny's internal hunger or cell hunger is the cause of decomposition, in contradistinction to hunger as usually thought of, which is due to lack of food to appease the appetite. It is characterized by general severe changes: (1) Rapid fall in weight; average 20 to 100 gm. daily loss, somewhat slower than in intoxication. (2) Lack of ability to assimilate food. (3) Subnormal temperature with irregular daily curve, with tendency toward collapse. (4) Slow and small pulse, with weak heart-tones. (5) Rapid respirations; prolonged expiration, and irregular breathing, even to the Cheyne-Stokes type. (6) The sensorium is not involved. (7) Urine contains no albumin or sugar. (8) Stools from normal to very bad, depending for their character on the previous diet. Dark brown stools usually indicate duodenal hemorrhages (Helmholtz). (9) The hunger is often very great and cannot be satisfied. (10) Vomiting is frequent.

Only a determined effort to limit the diagnosis decomposition to such conditions as are associated with acute rapid loss of body weight (lowered temperature) and general disturbance of the normal body functions will lead us to get good results in our therapeutic applications. It should follow nutritional disturbances in whose pathogenesis alimentary toxins play the chief rôle, and should not be confused with the subacute and chronic atrophies, as *tabes mesenterica* (tuberculosis), etc.; neither is it to be confused with chronic disturbed metabolic balance, which can be differentiated by a functional diagnosis; that is, a test meal, as described later.

ETIOLOGY

All factors leading to dyspepsia and intoxication may be forerunners of decomposition. At what moment this change takes place can only be answered by the fact that deep-seated organic changes are necessary to its development; these changes which produce such an intolerance toward nourishment may have developed previously to the preceding illness or during its course. Premature infants are especially predisposed, also young infants with previous dietetic errors, and diarrheal attacks; also those fed on one-sided foods, especially carbohydrates, as cereal diets; and also from too long-continued hunger diet. Especially in the young does this apply to cereal waters. All of the preceding reduce the tolerance toward normal food and its assimilation. The tendency to decomposition and, therefore, to narrowing of the nutritional sphere increases with each dyspeptic attack. Death may also be caused by secondary infections, but be unrecognized because of a low temperature, cystitis, etc. (Alimentary sickness, with infectious death, Pfaundler.) Death may also follow quite normal stools without preceding symptoms of dissolution, but this is usually following some dietetic error with cardiac or respiratory failure. This is often the cause of sudden death in our service in malnutrition cases.

Fig. 5.—Chart showing hospital record of infant in Fig. 6 with decomposition.

DIAGNOSIS

We must ascertain the degree of nutritional disturbance, which is accomplished by noting the history from the present attack backward. If the case be a decomposition, we find an infant who has had a history of repeated diarrheal attacks, with a rapid loss in weight; improvement on hunger diet, with recurrence on refeeding, and a gradual decreasing of the assimilative powers to a point where supportive diet becomes impossible. This difficulty in the stage of repair with proper diet is absent in disturbed metabolic balance and dyspepsia. If this history fails, our only recourse is a very careful test for the reaction to hunger diet and food.

Fig. 6.—Infant with decomposition whose hospital record is given in Fig. 5.

1. *Reaction to hunger diet* is always dangerous. (a) Temperature falls even to collapse. (b) Pulse also slows and weakens. (c) Weight always falls, but varies with the severity of the case. (d) Stools become more nearly normal.

2. *Reaction to food*: This gives a clew to the stage of which Finkelstein recognizes three. The first stage is very much like that of dyspepsia, except that there is a loss in weight with supportive diet. With careful diet the weight loss ceases in from three to six days. In the second stage, with the stopping of food, the weight drop ceases as in the first, but slower; recurrences are likely with the re-administration of food. Rarely can they be saved by the bottle. In the third stage, very little food increases the symptoms. The breast is the only hope. Functional diagnosis with (1) hunger diet, and (2) test meals is therefore equally dangerous.

Weight.—Food administration causes weight drop, and stopping of food is equally as dangerous, a more paradoxical reaction is hard to imagine. It is important to inquire whether shortly before seeing the patient a rich salt and sugar diet had been changed to a poor one, as this might cause symptoms resembling decomposition.

METABOLISM AND PATHOGENESIS

Decomposition is due to mineral loss. In the fat soap stool stage especially, earthy alkalies are lost, and, with diarrhea stools, leads to much more alkali loss. The increased ammonia in the urine is due to the fact that because of alkali losses ammonia is called on to neutralize the acids in the urine. The increased nitrogen waste results from cell destruction following the withdrawal of salts necessary to their vitality. It is a question whether the salt loss is a primary one, or only secondary to the formation of the acids and a call on them to neutralize the acids. The abnormal acid formation in the intestines is the result of bacterial fermentation of the food stuffs (fat and sugar) through bacterial action. This can only occur where the intestinal epithelium has lost its function, or, at least, been impaired. Heubner first pointed this out.

In the condition of the epithelial cells lies the basis of the difference between decomposition and the simple nutritional disturbances, repair of these cells being difficult or impossible in the former, while recovery is rapid in the simple forms. Pfaundler lays the seat of trouble in the intermediary metabolism, and believes that there is a deep-seated involvement of the organ cells throughout the whole organism.

It remains to be proven in how far the loss of salts is the cause of the clinical picture.

1. Loss of alkalies causes loss of water, as they are necessary to the osmotic equilibrium: the water loss through the skin and lungs is very important, as the loss through urine and bowels equalize one another, and the former losses (skin and lungs) probably account for the weight loss.

2. Loss of alkalies causes destruction of cells.

3. Loss of sodium probably accounts for the subnormal temperature.

4. Loss of sodium probably accounts for the slow heart action. Ringer and Loeb have both shown that sodium stimulates and calcium slows the heart's action.

5. Loss of alkalies results in further acidity of the organism (relative acidosis, according to Steinitz; alkalipenie, according to Pfaundler), of whose rule we know as yet very little in relation to the end result.

The healing quality of mother's milk over cow's milk is undoubtedly due, as shown by the experiments of Meyer, to the difference in the whey content, which has a very good influence in the former, and a deleterious one in the latter, on the impaired epithelium.

PROGNOSIS

The prognosis depends on the following:

1. The stage of decomposition.
2. Feeding of human milk. Even light forms offer little hope with artificial foods.
3. Lowered immunity, which adds danger of infection with colds, bronchitis, etc.

THERAPY

Avoid hunger day and feed human milk, about 200 c.c. daily to start.

1. *Feeding with Human Milk*.—Must be given in moderate quantity, best guarded by drawing off and feeding, as they drink too rapidly (always hungry), and do not stand large amounts. About 200 c.c. daily is enough to sustain the infants temporarily (60 calories per kilo is sustaining, Rosenstern). (*Erhaltungsnahrung*). Feed often; ten feedings may be given, one every two hours (10×20 c.c.); weak tea or saccharin water *ad libitum* between feedings. Expect improvement with the above unless intoxication results from the low salt diet, with resulting loss of water through the stools. If no bad results are seen, increase the food in two or three days. Favorable cases can be fed 100 calories per kilo in ten to fourteen days, and putting to breast may be tried, but the dangers of over-nursing must not be overlooked. Weight may still not improve for some time. (This Keller calls the reparation stage.) This may be shortened by giving one feeding daily of buttermilk or skim-milk, 100 c.c., which is rich in salts and protein. This is not to be done until after the third or fourth week of treatment and with close observation of the result.

Carbohydrates should be added with even greater care. In older children a small amount of farina soup or zwiebach in tea may be tried later.

2. *Artificial Feeding*.—Change from human to artificial food should always be preceded by a hunger day. After one day of hunger diet small quantities of low fat and sugar diet as buttermilk or skim milk without flour or sugar, or, if not at hand, whey whole or diluted, may be given, using 10×10 c.c., and with it saccharin water or tea. Increases must be more careful than with human milk. If results are good, we may increase in two weeks to 70 calories. Add carbohydrates and fats with great care. Carbohydrates increase weight more than fats. Maltose is less likely to affect the bowels and is more assimilable. One per cent. may be added at first, increasing very slowly to 5 per cent. In infants older than 3 months, in addition flour may be used. Always expect a setback with new food additions.

3. *Albumin Milk* is of value in decomposition where human milk is not available; however, my results would lead me to make every effort in the future to feed this class of infants with human milk. Stimulants, artificial heat, and, in severer types, transfusions are indicated.

V. MIXED FORMS OF NUTRITIONAL DISTURBANCES

Bilanz-Störung (disturbed metabolic balance) is frequently associated with dyspeptic symptoms. Again, these may show signs of intoxication. Decomposition is the form most subject to complication.

THERAPY

Disturbed metabolic balance (*Bilanz-Störung*) with dyspeptic symptoms, treat as a dyspepsia by reducing diet. In dyspepsias with signs of intoxication, employ hunger diet, etc.

Decomposition complicated by intoxication is the severest combination, but can be recognized, if careful consideration is given to the history and the infant's general condition. The greatest danger lies in the fact

that in the former we must nourish; in the latter starve. Without a hunger day either human or albumin milk should be given in small quantities, 10×5 c.c., and increased 50 c.c. daily under control of the clinical symptoms. After eight to ten days of the above the infant can usually be put to the breast five times daily. The weight may remain stationary, and one must judge by the stools and general condition as to the addition of further foods. With albumin milk, after three or four days, the weight becomes stationary and the stools of fat soap character; then gradually some malt sugar can be added.

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METABOLISM AND NUTRITION

Importance of Proper Balance of Fat and Lime in Infant Feeding

(K. Stolle: *Jahrb. f. Kinderh.*, 1911, lxxiv, No. 4, p. 367)

Stolte comments on the conflicting statements of various pediatricists in regard to infant feeding, some obtaining excellent results with one food-mixture, while others have found this mixture harmful: some denouncing the very elements in the food to which others ascribe its efficiency. Looking over the entire field from a higher standpoint shows, he says, that one feature is common to all the cases in which the infants thrived, namely, that the stools became less fluid. His research indicates that this change in the consistency of the feces is the result of soap production and absence of fermentation. The aim in feeding, therefore, should be to insure soap production and avoid fermentations. From this standpoint the benefit from the most varied systems of infant feeding is easily understood; each by different routes arrives at the same goal. The essential point is the proper balance of fat and lime in the bowel to insure soap production. If too much or too little of either is present, the desired end is not attained. Fermentations induce the production of fatty acids, which irritate the intestines and interfere with saponification. These views explain the advantages of albumin-milk, as the intestinal flora with this induce putrefaction rather than fermentation. They explain further the tolerance of carbohydrates with buttermilk, which contains especially large proportions of lactic acid and albumin. They also explain how diarrhea may be arrested by albumin-milk, by buttermilk, by diluted milk, or by milk and lime-water. With cow's milk more lime is needed to saponify the fatty acids generated in the intestine by splitting of the fat. Stolte found it possible to check diarrhea almost unfailingly when he gave the infants lime-water with the diluted milk, without carbohydrates. The more complex the structure of the carbohydrate given, the longer it takes to be broken down into its ultimate elements, and thus the less material to ferment in a given period of time, and hence the less opportunity for the fermentation producers to get in their work. These more complex polysaccharids include dextrin, flours and malt. This difference between the various carbohydrates was instructively shown in one of his cases in which the infant had diarrhea on albumin-milk and cane-sugar, while the stools became formed when flour was substituted for the sugar, and the stools became actually hard when nothing but a cereal gruel was given with the albumin-milk. It is possible that the property of promoting putrefaction rather than fermentation is the chief advantage of the albumin-milk. Infants thrive on breast milk after serious illness, although breast milk is poor in albumin, showing that the protein content alone is not the essential factor.

W. T. C.

ACUTE INFECTIOUS DISEASES

Acute Rheumatism Among Children

(F. Langmead: *Lancet*, London, 1911, *clxxxi*, 1133)

Altogether 2,556 children were examined by Langmead. Of these, 844 were boys and 838 girls in the senior departments, i. e., from 6½ to 14 years old; 874 were attending the infants' department, and consisted of nearly equal numbers of boys and girls from 3 to 6½ years old. Fifty-nine boys and 74 girls could be classified as definitely rheumatic, a total of 133. This is equivalent to a percentage incidence of 5.20 for children of all ages attending school, or of 6.83 for children in the senior departments only, those attending the infants' department being excluded from the estimation. In the majority of cases attention was drawn to the rheumatic child by the condition of the heart, sometimes by definite valvular disease, at others by dilatation accompanied by such alteration in sounds and rhythm as one is accustomed to hear in rheumatic children. More and more children become rheumatic as they grow older. There is, however, an apparent accession of rheumatism at the age of 10 which is not shown in subsequent years. Langmead gives no explanation for this and regards it as an inaccuracy which would have disappeared if a large enough number of children had been examined.

Out of these 133 children all except eighteen showed some evidence of cardiac disorder at the time of examination. Thus rheumatic heart affections occurred in 4.49 per cent. of the children of all ages attending school, or in 5.94 per cent. of those in the senior departments only. Obvious valvular disease was found in 2.93 per cent. of children at all ages, or 3.92 per cent. of those in the senior departments. Mitral regurgitation was noted forty-four times; aortic regurgitation and mitral stenosis only once; dilatation with a systolic bruit at the apex, possibly mitral regurgitation, twelve times; dilatation with a short presystolic rumble, or so-called reduplicated second sound (mid-diastolic bruit), possibly early mitral stenosis, eight times; dilatation without bruits, twenty times. The comparative rarity of aortic regurgitation in children is emphasized by the fact that only one case was discovered among the 2,556 children examined. Chorea did not appear among the children or in the histories of their previous illnesses as often as might have been expected. There were eighteen cases only, which amounted to 0.70 per cent. of the children of all school ages or 1.06 per cent. of the children in the senior departments. The comparative incidence in boys and girls followed the usual rule, there being twice as many girls affected as boys. Thus 0.71 per cent. of the boys were at some time choreic and 1.43 per cent. of the girls. The relation of enlarged tonsils and adenoid vegetations to rheumatism in childhood is well illustrated by the figures obtained. Taking the 133 rheumatic cases, in seventy-five there were no signs of abnormality in the throat, in twenty-one the tonsils were slightly enlarged, in eleven considerably enlarged, in sixteen greatly enlarged and in three tonsillotomy had been performed before the patients were examined. Adenoids were responsible for producing signs of post-nasal obstruction and mouth-breathing in seven. In other words, there was an abnormality of the tonsils or pharyngeal mucosa or both in fifty-eight cases, or in 43.6 per cent. of the rheumatic cases. This was sufficient to warrant operative interference in thirty-seven cases, or 27.8 per cent. of the rheumatic cases.

F. C. ZAPFFE.

Diphtheritic Paralysis

(Andrew Love: *Glasgow Med. Jour.*, 1911, *x*, 245)

The cases on which this article is based were under Love's care in Belvidere Fever Hospital, Glasgow, during the years 1900-1906, inclusive, and in Gateside Fever Hospital, Greenock, during the years 1908-1911, inclusive. The number of cases of diphtheria admitted during the period selected was 1,313, and of these eighty-five or roughly 6 per cent. became paralyzed. The years 1905 and 1906, and more especially 1906, show an enormous increase in the number of cases of

paralysis. This was principally due to the greater severity of the disease during these years. Of the total number of cases observed who suffered from paralysis, thirty-four were males and fifty-one females, their ages ranging between 14 months and 40 years. Paralysis was more common at those ages when the incidence of the disease is greatest. Generally speaking, the later the day of illness before antitoxin is administered the greater is the toxemia, and naturally, the greater the danger of paralysis supervening. Of these eighty-five patients, sixty-five, or 77 per cent., recovered, and twenty, or 23 per cent., died. In no one case could death be attributed solely or directly to paralysis, although in all cases it was contributory. In ten of the cases that terminated fatally the disease was of the malignant type, while in the others it was of a severe character. Eight had nasal implication, seven a combination of faucial and laryngeal diphtheria with tracheotomy in three instances, and twenty-seven had greatly enlarged cervical glands. Of the latter, two went on to suppuration. Of the eighty-five cases, fifty-three, or almost two-thirds, had paralysis of the palate alone; fifteen of the eyes and palate; three of the eyes alone (strabismus). Of the fifteen who were afflicted with paralysis of the eyes and palate combined, three had paralysis of accommodation as well as strabismus. Paralysis of the ocular muscles alone is uncommon, and only two of the sixty-eight patients recorded were affected in that way. Of the twelve patients noted as having intercostal and cardiac paralysis, nine died and three recovered, the main symptoms being great difficulty in inspiration with marked feebleness and irregularity of the heart. In the one case in which the lower limbs only were affected the loss of power lasted only for a few days. The average duration of the paralysis was eighteen days. Two of the cases had paralysis of the palate on admission to the hospital, and the condition persisted until death occurred. In the cases in which death occurred the average number of days of residence in hospital was twenty-one, while the average number of days spent in hospital by those who recovered was 70.5. It is interesting also to note the persistence of the membrane on the throat after the administration of antitoxin. In no instance did the membrane disappear before the third day after admission to hospital, and only then in the milder cases. The average number of days that elapsed in the cases here recorded before the throat was free from membrane was 7.2; the longest period noted during which the membrane persisted was fourteen days. Vomiting in diphtheria is always regarded with apprehension, as it is frequently associated with cardiac failure. In the cases here recorded vomiting occurred in fourteen, and nine of these had a fatal termination. It is noteworthy that the vomiting occurred simultaneously with the onset of the paralysis. The urine varied greatly as regards albumin. Of the eighty-five cases, twenty-seven had a large quantity of albumin in the urine, eighteen a moderate quantity, seventeen a mere trace, and twenty-three no albumin at all. With one exception there was a large quantity of albumin in the urine of all the fatal cases. In the cases under consideration fifty-six received a single dose of antitoxin, the smallest being 6,000 units and the largest 75,000 units, while the average amount administered was 17,000 units. Second doses were given in twenty-four cases, the average in these being 17,500 units. In five cases only were third doses resorted to, the average in these instances being 9,500 units. The over-all average, that is, the average aimed at by taking all the doses administered to all the patients, is represented by the somewhat large amount of 22,000 units. In connection with the administration of antitoxin, it is interesting to note that out of the eighty-five cases dealt with, thirty-eight were afflicted with serum rashes. In twenty-eight cases the rash was urticarial in character and general in distribution; in six it was scarlatiniform, and in four morbilliform. Two of the rashes, which on their first appearance were urticarial, became morbilliform in about twenty-four hours, while one, which was in the first instance scarlatiniform, became morbilliform in the same time. The serum rash when urticarial in character appeared usually on the ninth day after the administration of serum, and lasted on an average 4.2 days. The scarlatiniform rash also appeared on the ninth day, but usually lasted only two days,

while the morbilliform rash came out on the twelfth day after the injection of serum, and as a rule lasted four days. All important as is the early administration of antitoxin, it is a mistake to suppose that in the late cases antitoxin is of no avail. Twenty-seven of the patients were admitted after the fifth day, and all received antitoxin. Of these, five died, a mortality of 18.5 per cent.

F. C. ZAPFFE.

Blood-Pressure in Diphtheria

(*J. D. Rolleston: Brit. Jour. Child. Dis., 1911, x, 433*)

This paper is based on observations made on 179 cases of diphtheria admitted to the Grove Hospital from July, 1909, to December, 1910. The blood-pressure in each case was taken with C. J. Martin's modification of Riva-Rocci's sphygmomanometer. The systolic pressure, as measured by disappearance of the radial pulse, was alone taken into consideration. As far as possible the measurements were taken at the same time each day between 10:30 a. m. and noon; i. e., from one and one-half to five hours after food; in many cases an evening record between 6:30 p. m. and 7 p. m. was also made. The blood-pressure was taken daily in each case until the patient was allowed to sit up; i. e., for a period ranging from three weeks in the mild cases to six or eight weeks in the severe. Comparative observations were subsequently made on the different readings in the recumbent and erect positions. Apart from the early fatal cases the total period of observation in each case ranged from four to fourteen weeks. All the patients, except fifteen who were above the age of 15 years, were children. Ninety were males, eighty-nine were females. There was no appreciable difference in the blood-pressure of any two individuals of different sexes but of the same age and suffering from attacks of diphtheria of equal severity. Of the 179 cases, sixty-three, or 35.1 per cent., according to Cook and Brigg's estimate, and forty-five cases, or 25.1 per cent., as judged by Seiler's standard, showed for varying periods a pressure inferior to the normal. Owing to the well-known depressive effects of diphtheria toxins these figures appear surprisingly low, but are explained by the inclusion of a large number of mild cases, in which the blood-pressure was not lowered in the course of their illness. The varying degrees of depression bore a direct relation to the severity of the attack, being pronounced and persistent in the severe and slight and transitory in the mild cases. Of those whose blood-pressure was not lowered, thirty-six cases, 20.1 per cent., according to Seiler's standard, showed a reading which always exceeded the normal. Judged, however, by the more liberal estimate of Cook and Briggs, the pressure of these cases did not persistently rise above the normal limits. In ninety-seven of the 130 cases admitted in the first week of disease and in ten of the fifteen cases admitted in the second week, the highest reading was the first taken. The occurrence of the comparatively high pressures in the first week is attributed partly to the febrile disturbance of the acute stage before the diphtheria toxins had had time to produce their characteristic effect and partly to the psychical action caused by the application of an unfamiliar instrument, the latter fact accounting for the highest reading being usually obtained on the first measurement. The preponderance of the lowest readings in the second week accords with the clinical fact that this is the time of predilection for the so-called "cardiac paralysis," which should more accurately be called vasomotor paralysis, the heart failure being secondary to the vasomotor affection. Apart from one case of diaphragmatic paralysis, all the deaths in the present series were due to this cause. The lowest reading in a patient who recovered was 60 mm., which was registered on one occasion in a girl aged 3 years. In only ten other patients who recovered did the pressure fall below 80 mm. in patients above the age of 2 years. The pressure in forty cases returned to the normal by the end of the seventh week. Forty-two cases, or 23.4 per cent., showed various degrees of laryngeal involvement. In thirteen severe cases tracheotomy was performed; in eleven moderate cases the symptoms, though fairly well marked, did not require surgical interference; in eighteen mild cases the laryngeal symptoms consisted merely of a croupy cough and husky

voice, thus representing the first degree of croup of earlier writers. The effect of laryngeal obstruction on the blood-pressure was most marked in the severe cases. Increase in dyspnea was followed by a rise of pressure and the relief of the obstruction on opening the trachea was immediately followed by a considerable fall. The rise of blood-pressure in the moderate laryngeal cases was less than in the severe. The blood-pressure in the mild laryngeal cases during the acute stage was not any higher than in the purely faucial cases; in fact, it may be said to have been dominated by the character of the concomitant faucial attack. In the great majority of early serum rashes, erythematous or urticarial, no change occurred in the pressure, while in a large percentage of late eruptions, which are usually associated with febrile disturbance and often with pains in the joints, muscles and fasciæ, the pressure was raised. Thus of seventy-seven cases at the time of the early eruption, sixty-two patients, or 80.5 per cent., showed no change in the pressure, in nine it was raised and in six it was lowered. On the other hand, out of fifty with late rashes, in twenty cases, or 40 per cent., the pressure was raised, in twenty-eight, or 56 per cent., there was no change, and in only two cases, or 4 per cent., it was lowered. The rarity of any rise in pressure at the time of the early rash is in keeping with the fact that during the second week of the disease, in which most of these rashes occur, the pressure is usually at its lowest. In the third week, in the course of which the late serum phenomena occur, the pressure as a rule is no longer falling, while on the other hand the fever and joint pains tend to raise it. As arterial hypotension and albuminuria are both phenomena of diphtheritic intoxication, any change in the blood-pressure coexistent with the occurrence of albuminuria was almost invariably in a downward direction. Thus among fifty-one cases in whom albuminuria was not present on admission, its onset was accompanied by a fall in the pressure in twenty-six and in twenty-five there was no change; in another eight cases increase of preexistent albuminuria was accompanied by a fall in the pressure. The solitary exception to the rule occurred in a girl aged 6 years, in whom severe faucial diphtheria was complicated on the twenty-ninth day by uremic convulsions, one of the rarest events in diphtheria. On the two following days the blood-pressure, which up till then had not been examined, stood at the disproportionately high figure of 120 mm., falling on the thirty-second day to 80 mm. Death took place on the thirty-third day. Albuminuria in diphtheria thus offers a striking contrast to scarlatinal albuminuria, during which it is usual for the blood-pressure to be raised. Thus in eleven such cases Rolleston found a rise in all but two. In early paralysis the blood-pressure tended to fall. In late paralysis, even when extensive, it was usually not affected. Rolleston concludes that sphygmomanometry in diphtheria, as in other acute diseases, though of considerable theoretical interest, has little practical significance.

F. C. ZAPFFE.

TUBERCULOSIS AND SYPHILIS

Consumption and Order of Birth

(W. C. Rivers: *Lancet*, London, 1911, *clxxxi*, 987)

Rivers observed in patients' case-sheets which showed ordinal position in the family, a large number of entries of eldest-born children, and from this observation conceived the idea that clinical pulmonary tuberculosis might affect such persons more than others. Brehmer's and Riffel's reports show that both first- and second-born offspring appear subject to consumption at a very much higher rate than younger members of a family. Rivers' own observations in 263 cases confirm those of other clinicians. Mitchell, among 443 idiots and imbeciles, found 138 first-born, or 31.1 per cent. Langdon Down claims liability to idiocy among primiparous children because 24 per cent. of the 2,000 idiots (illegitimate largely excluded) examined by him were first-born children. Among Wilbar's 675 there were 191 first-born; among 100 of Beach's, twenty. Thus, respecting

the exact amount of increased risk of developing phthisis that an eldest child runs, no direct data exist from which to calculate. As matters stand, one may say that whereas in 1,228 consumptives (494 upper middle-class Germans; 353 middle- and lower-class inhabitants of Lancashire, London, and Northumberland, and 381 mainly lower-class Lancastrians) one should find 196 eldest children, one actually does find 281, i. e., eldest-born consumptives are in excess, say, 40 per cent.

Of 141 of Rivers' last cases there is one patient with diabetes mellitus, three who suffer from hay fever, and three the subjects of congenital xerodermia. The diabetes patient is an eldest child; two of the hay fever patients are likewise, while the remaining one is second-born, and two of the ichthyotics are eldest-born. Yet the first- and second-born of these 141 only number fifty-eight and the first-born alone only twenty-eight. This, in Rivers' opinion, would seem to point to the conclusion that the tendency of the eldest-born to become consumptive is an innate tendency and the hypothesis is strengthened, he says, if we appeal to the literature of the subject. Lunatics and idiots are certainly very liable to consumption, and, as has been seen, it is exceedingly likely that they are disproportionately often eldest-born. Again, if faulty upbringing by inexperienced parents is to be made responsible for elder-born criminals, lunatics, idiots and perhaps men of genius, this would render ordinary human responsibility sufficiently formidable. The existing evidence negatives the view that infection is the sole cause.

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TRANSMISSION OF DISEASE BY MEANS OF BOOKS

Request for Data

I am preparing a paper on "Books as a Source of Disease," to be read before the next International Congress on Hygiene, and in order to obtain data, I respectfully request the readers of this note to send me an account of any cases, the sources of which have been traced to books or papers, or where the evidence seemed to make books or papers the offender. I further request information concerning cases in which illness or even death has been caused by the poisons used in book-making.

All the information possible is wanted in order to present as complete a paper as possible. As in the case of insects which we now know to be "carriers of disease," it is first necessary to collect the scattered evidence in order to show that there is real danger in books; and this will compel better care to be taken of libraries and books and improve the health of mankind.

The Free Library of Philadelphia, Department of Public Documents.

WILLIAM R. REINICK,
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BOOK REVIEW

Case Histories in Pediatrics. A Collection of Histories of Actual Patients Selected to Illustrate the Diagnosis, Prognosis and Treatment of the Most Important Diseases of Infancy and Childhood. By John Lovett Morse, A.M., M.D., Assistant Professor of Pediatrics, Harvard Medical School; Associate Visiting Physician Infant's Hospital and Children's Hospital, Boston. W. M. Leonard, Boston, Mass., 1911.

The title of this volume describes its purpose and mission. Teaching medical students by means of actual case histories originated at Harvard several years ago. The author states in his preface that this method of teaching is far superior to recitations, quizzes and conferences. It compels the student to think for himself. It is almost as valuable as the clinical lecture, in which the patient is shown, and is more instructive than the didactic lecture.

This book should inspire every physician who teaches any branch of clinical medicine to follow out the same method with his own cases. The description of

each case is so clear and concise and the essential points in the examination and diagnosis so forcibly brought out that this little volume could well serve as a pattern.

There are one hundred cases cited, which are grouped into Diseases of the New-Born, Gastro-Intestinal Tract, Nutrition, Infectious Diseases, etc. Each case illustrates a different disease, except in a few instances, wherein two examples which illustrate different phases of the same disease are included.

The value of this book would be greatly enhanced if more illustrative cases were included under certain sections. It is impossible to give a comprehensive picture of diseases such as tuberculous meningitis, bronchopneumonia, etc., by quoting one case.

The omission of cases illustrating chorea, tuberculosis, asthma, enuresis and the acute exanthemata, which are important and characteristic diseases of childhood, should be remedied in later editions.

The discussion of the differential diagnosis is full of practical points which the author has gleaned from his own wide personal experience. The treatment is based on what was needed and undoubtedly advised in each individual case.

There is nothing in this volume that savors of the text-book. The general practitioner will find many valuable suggestions and useful hints which are not found in other books. The index is complete and reference to the various diseases can readily be made.

The book would be more readable and satisfactory from a typographical point of view if the name of the disease were printed prominently as a heading to each case.