times, cauterized then the fifth time, saved the eye and some vision. In no case in the last seven years, in which he has used it constantly, has he had the least cause for regret, and there is never a time that he does not have some cases under treatment. To him it is as easy to use, equally convenient, better controlled, and infinitely more reliable, while fully as safe, if not more so, than any other method.

DR. G. C. SAVAGE, Nashville, Tenn., emphasized the value of dionin. It relieves tension, which gives great comfort, for the pain is largely due to the increase of tension. He believes the very best agent for the local treatment of corneal ulcers is acetic acid. It will destroy the germs; it has penetrating powers. It not only destroys the germs lying on the surface, but reaches those deeper in. The great advantage it has over any other application is that it does not destroy cell tissue. The epithelium is turned white for a moment, but soon clears up and the vitality of the tissue is greater than it was before. Iodin, the cautery and carbolic acid all destroy cell life as well as germ life. To his mind acetic acid has a decided advantage over any other application we can make to an ulcer. Besides making the application of the acid (1 part to 7 to 10 parts of water), with a cotton-wrapped toothpick, under cocain, he nearly always gives a weaker solution of the acid (5 to 10 drops to 1 ounce of water), to be used constantly.

DR. LEARTUS CONNOR, Detroit, said that one individual will find one agent better and another another. The thing is to destroy the malignant germs without harming the cell tissue. He has used all the remedies mentioned, with excellent results. At one time he used to fill the eye with a solution of bichlorid of mercury and then with a scoop curette the ulcer and obtained very good results. He thought the use of dionin important because the drug reduces the tension considerably. Dry cupping of the temples he finds helpful in certain conditions. In some cases he never omits a vigorous calomel and bicarbonate of soda purge and always regulates the diet with the idea of keeping the tension low. Generally patients get the greatest good as well as comfort from the local use of hot water. It is very effective and can not do any harm. He uses a tumbler, applied touching the nose and edge of bone below the socket and then raised until the eye is in the water. It is the next best remedy to atropin.

DR. S. L. LEDBETTER, Birmingham, Ala., said he had used hot water in the manner referred to by Dr. Connor, using a teacup and telling the patient to turn it over the eye and open and close the eye, having them use it frequently and for five or ten minutes at a time.

DR. H. B. ELLIS, Los Angeles, said that, for the last four or five years, he did not think he had ever had a case but what he had used dionin, in conjunction with atropin or eserine; sometimes separate from either of these, and as a promoter of absorption and for the relief of pain he knows of nothing better.

ALIMENTARY INTOXICATION.

WILLIAM J. BUTLER, M.D.

Assistant Professor of Pediatrics, Rush Medical College, in Affiliation with University of Chicago; Attending Physician, Cook County Hospital.
CHICAGO.

A series of toxic symptoms are frequently observed during the course of nutritional disturbances in infancy which Finklestein has termed alimentary intoxication. The clinical picture made up of these symptoms is better known under the appellation entero-colonic catarrh, milk poisoning, bowel infection, etc., and in the severer cases the term cholera morbus is applied to it.

The attack does not occur suddenly in a previously healthy infant, but, on the contrary, in one in whom a distinct deviation from the normal has existed for some time, which can only be ascertained by a close study of the daily temperature and weight charts. Clinically, such children may not, at this time, present any promi-

nent symptoms. They may or may not be irritable and restless during sleep. The stools often continue normal in appearance and number, or they may be increased to three or four in the twenty-four hours, and be of thin consistency. Occasionally, regurgitation of food after feeding is observed. There is a daily fluctuation of temperature, which is never marked, varying from 1 to 2 degrees. The weight curve shows that the infant, instead of gaining, is standing still or losing slightly.

This condition is always found to be preceded either by a quantitative or qualitative increase in the diet or the substitution of some other diet, which has carried with it an equivalent effect. This period is spoken of as the dyspeptic stage. It illustrates how close a relation exists under certain conditions between disturbances in the infant and food changes. This can readily be demonstrated, and, in fact, is occurring constantly in practice, by increasing the diet to intolerable points, uncontrolled by accurate temperature and weight records. In this manner this stage can be diagnosed intelligently, and the climax of intoxication may be aborted by taking appropriate measures. The dyspeptic stage may continue for days or weeks before the onset of the severe toxic symptoms. The latter may occur suddenly, at any time, during the dyspeptic period or after an additional increase in the diet.

The onset of the intoxication is announced by marked nervous symptoms, which may take on one of two forms. After a varying period of irritability, the infant is seized with convulsions, followed by a condition of sopor or coma, or he may become apathetic, very drowsy, not easily awakened, soporose and in some instances comatose. Occasionally, the temperature remains afebrile, but, as a rule, it rises sharply.

The number of stools are often increased, but not infrequently seem normal in appearance and number at the beginning of the attack. Considerable depression is evident, and in the severe forms there is marked collapse, with a low blood pressure and livid skin. Prior to, coincident with, or subsequent to the rise of temperature, there is a sudden decline in the weight curve, the loss often reaching from 400 to 700 grams in from 24 to 48 hours. The breathing is accelerated, deep and may be misinterpreted as a pulmonary symptom.

A striking feature at the height of intoxication is the alimentary glycosuria, dependent on lactose and galactose, and due to their absorption and failure to undergo oxidation. At the same time, an albuminuria and cylindruria may be found. A leucocytosis is invariably present during the acute stage, but not exceeding 30,000. Skin manifestations, such as vasomotor exanthemata, scleroderma, etc., are not uncommon.

A study of the physiognomy is most important in the diagnosis of this condition. A comparison of the expression and facial movements of the infant suffering from alimentary intoxication with that of the normal healthy child will readily show the finer grades of disturbances of consciousness, etc., and enable one in the first glance to recognize the infant suffering from intoxication. The face is usually relaxed, or drawn, as if in pain. The eyes may appear fixed, staring into space, or move slowly on attempts to attract attention.

The entire clinical picture varies in severity in different cases. In some cases all the symptoms will be found present in classical form; in others, certain symptoms will be so pronounced as to overshadow the rest. There are certain familiar conditions which Finklestein has styled forms of alimentary intoxication.

1. The choleric form, with great loss of weight and extreme collapse, and only slight evidence of cerebro-spinal symptoms.

2. Hydrocephaloid form, in which the nervous symptoms predominate, simulating a tubercular meningitis.

3. Soporose form, in which, from the beginning, there is deep sleep, which may deepen into coma, while all the other symptoms may be but slightly marked.

4. Asthma dyspepticum, in which the peculiar type of breathing is the most marked symptom.

The symptoms of intoxication do not always form the climax of a period of dyspepsia. A chronic intoxication may develop, resulting in what Finklestein calls decomposition, better known as marasmus or infantile atrophy. The symptom-complex characteristic of alimentary intoxication has usually been referred to bacterial decomposition of the food and absorption of toxins, or to bacterial infection, notwithstanding the frequent want of pathologic proof for the latter in many fatal cases.

Before discussing this feature, it may be well to establish tenable grounds for the assumption of an alimentary intoxication.

This condition is never seen developing in the healthy infant at the breast whose nursing is properly regulated. It is, however, not infrequently seen developing in a poorly nourished infant at the breast whose feeding is not properly regulated. Its usual subject is the artificially fed infant suffering with some disturbance of nutrition.

The status of nutrition of the infant plays the important rôle in this matter, and the more seriously it is affected the closer it approaches the stage of decomposition, the more serious and irreparable is the damage caused by intoxication. It is this nutritional disturbance of the child which renders possible the precipitation of a period of intoxication by dietetic errors, the latter acting merely as the exciting cause.

The action of the food on the infant with relation to development of toxic symptoms can readily be followed in artificially fed infants, when under complete control for observation and study. Intolerant increases in the quantity of diet or of individual constituents will be found to commence a period of dyspepsia, which latter, as above stated, can only be followed accurately by daily weighing and observation of temperature. The temperature will show daily subfebrile fluctuations, which, though slight, are significant, and the child, instead of gaining normally, will stand still or even lose.

This dyspeptic stage may be terminated by lessening certain constituents of the food, such as carbohydrates or fats, and in other instances by lessening the total food quantity. Lack of interference in the matter of an increase in food at this time will precipitate, sooner or later, the intoxication. And exactly at the time when the catastrophe of intoxication develops, the most convincing argument of the influence of food in the production of alimentary intoxication becomes evident. If food is withdrawn entirely, and water alone is allowed, within twenty-four hours, the temperature drops to normal, the weight loss ceases, the mental condition clears up, glycosuria disappears, and the most desperate clinical picture is converted into one of hopefulness. It might be contended, however, that this is not necessarily convincing, as it might be assumed that ridding the intestinal tract of its contents and not the withdrawal of food, is responsible for the drop in temperature. I think it will be conceded that if the temperature remains normal under institution of proper treatment with relation to food from the time of this first drop,

and this can be demonstrated, it, at least, could not have been an inflammatory process that gave rise to the first jump in temperature.

If a bacterial decomposition of the food were responsible, this would have occurred in the albumin of the food, and we would have an alkaline stool, while the stool in the intoxication is acid; on the other hand, were it alkaline, this decomposition could just as well have occurred in the bowel secretion.

It is well, here, to consider the reaction of the infant to food and food constituents that can be noted at this time.

As already stated, if, at the height of intoxication, the food is entirely withdrawn and water substituted, the temperature will drop to normal and can be maintained there if proper treatment, which will subsequently be considered, is followed. If, after the temperature has been normal, whether it be for a few hours, twenty-four hours or longer, the former diet is resumed, even in small quantities (barring certain instances that will later be mentioned), shortly thereafter the temperature will begin to rise, and if the diet is continued, in a day or two a relapse of the intoxication has developed with all its symptoms, including glycosuria. If, now, the food is withdrawn, the same sudden drop in temperature and improvement of symptoms will be observed. It might be claimed that there was a reinfection, but this change occurs under the most careful precautions for sterilizing the food, and the reaction in the infant occurs too rapidly to have been referred to bacterial infection or decomposition of food. On the other hand, no one has been able to demonstrate poisonous toxins in the bowel contents in these cases, and if a reinfection had occurred, it would not subside on withdrawal of food.

The relation of the food constituents to the infant's condition during the stage of de-intoxication is interesting. Quite contrary to expectations, the casein seems to act as an inert food during this period. It has been found that casein, precipitated from fat-free milk, and broken up, can be given mixed with the water the infant receives and his convalescence will proceed as though he were receiving only water. Quite the opposite is observed with the fats and carbohydrates.

Fat given during the attack is disastrous, causing the condition to grow worse at once. Fats are not only deleterious at this time, but also prior to intoxication, when an increase in this constituent is not infrequently responsible for precipitating the acute attack.

The rôle of the carbohydrates is especially important in starting the disturbance leading up to intoxication, and during the intoxication, if given, they will cause a persistence of the symptoms, often leading to a fatal termination.

It is a matter of experience that some children, with so-called "bowel infections," do not tolerate whey well after they have been tided over an apparently severe attack by the withdrawal of food. This formerly was attributed to the residual proteids in the whey, following the Biedert teaching of attributing almost everything harmful in milk to proteids. One can observe this same result when the infant is given milk sugar solution instead of whey, indicating that the carbohydrates are in all probability the responsible factor. Such misleading views concerning proteids have been responsible for the long discussions on intoxications, which have been inadvertently referred to as infections and reinfections, but which might have been cut short by proper diet.

It was long ago observed by Wiederhoffer that some of these children, when placed on the breast, instead of im-

proving, became worse and died. On the other hand, there are a certain number of infants who will slowly improve on the breast, and it is here that the question of the previous condition of the child plays such an important rôle in causation and in convalescence from alimentary intoxication. If the infant has suffered seriously before the development of the intoxication he may not show the sudden favorable reaction on the withdrawal of food, and only after a few days show a normal temperature and freedom from serious symptoms. Again, a certain number of cases are so overwhelmed that they proceed immediately to a fatal termination.

This condition of the infant is a very important factor and depending on it all shades of reaction of the organism to intoxication may be observed. While one infant may develop a relapse in convalescence from slight errors in dietetic management, another may, after the subsidence of intoxication symptoms, be able to take almost the same quota of nourishment as before the attack. In some cases there may be present a severe infectious complication, causing fever and preventing convalescence.

From the above considerations it would seem evident that we must seek the cause of this condition not in bacterial infections or food decompositions, not in toxins supposed to be contained in milk, as this condition may occur under the most careful preparation of food, but in an intolerable quota of nourishment, either quantitatively, qualitatively or both, supplied to an infant already suffering from nutritional disturbance, resulting in an abnormal destiny of the nourishment in intermediary metabolism. That some grave perversion of metabolism exists would appear from the presence of glycosuria at the height of intoxication.

While it presents clinically, in its severest form, a picture similar to that of the uremia or diabetic coma, there are some objections to the acidosis theory, which I will not discuss now.

Before considering the treatment I wish to report a case in my service at the Cook County Hospital:

This child of four months was brought into the hospital without any history, except that it had been fed on condensed milk, and had been seized with a convulsion the day it was received. The infant was soporose and in a state of collapse, with livid skin; emaciated; temperature 103 F.; pulse rapid; breathing accelerated; bowel movements brownish in color and of a thin, batter-like consistence.

The treatment employed in this case was as follows: Salt solution hypodermically, to combat collapse and low blood pressure. Water only by mouth. There was a drop in the temperature by the following morning, and the temperature remained at this level until the child was placed on a diet composed of milk, whey and water, when a relapse occurred with all the symptoms of intoxication repeated. Withdrawal of the milk given in the mixture resulted in a considerable drop in the temperature, although it still continued above normal, and symptoms of intoxication persisted. Suspension of the diet, and substitution of barley water, caused an immediate drop to normal and disappearance of symptoms.

The indications for treatment in alimentary intoxication consist in combating the collapse, cutting short the intoxication, and slowly bringing the child back to a diet commensurate with its caloric requirements. The first indication may be met by hypodermoclysis of salt solution, using 50 to 100 c.c., and repeating as is deemed necessary. The second indication is met by withdrawing the food entirely, and allowing water only for twenty-four hours. With the subsidence of all symptoms, begin feeding as follows: As exceedingly small quantities of fat are injurious, begin with centrifugated breast milk, if possible, given in teaspoonful doses, five times a day,

or an equal quantity of buttermilk, to which neither sugar nor meal has been added; or if this is not at hand, fat-free milk may be used. As a diluent, and to supply the liquid necessary, water and a thin cereal gruel may be used or given separately.

After an interval of twenty-four hours, provided the above has been well tolerated, without any evidence of return of the intoxication, the separate quantities of milk may be increased to two teaspoonfuls, which, after two or three days may, under the circumstances mentioned, be doubled, and so on, until they are gradually brought to a diet (free of fat in artificially fed infants) that will temporarily meet their needs from a caloric standpoint, having added, in the meantime, with their increased tolerance of the buttermilk (where employed), 30 grams of sugar, preferably maltose, and 15 grams of flour to each liter of buttermilk while boiling.

The child should not be kept on the buttermilk diet longer than six weeks. After this time whole milk dilutions will be necessary to meet its needs. In case the infant was at the breast, after four or five days of careful feeding, with centrifugated breast milk, it may be returned to the breast, the amount of nurse being carefully controlled.

During the treatment slight relapses are common where the food toleration has been passed. More effective and permanent results are accomplished by proceeding slowly, allowing forty-eight hours for judging the effect of a food increase before again adding to it.

Cathartics and intestinal antiseptics are unnecessary, unless constipation exists, as sometimes occurs in the soporose form, when a laxative is indicated.

There is little doubt that injudicious dieting and constant or frequent administration of laxatives in convalescence are responsible for the persistence of varying temperature rises in the first instance, and slimy green stools in the second instance, which are wrongly interpreted as manifestations of an entero-colonic catarrh, or a bowel infection with repeated reinfections, etc.

RESTORATION OF THE PELVIC FLOOR.*

HOWARD HILL, M.D.

KANSAS CITY, MO.

The lesions resulting from labor involve, primarily, the pelvic floor and the sphincter apparatus to a varying degree, while, secondarily, there result certain displacements of the pelvic organs directly dependent on the weakened pelvic floor. The manifold operations for repair of these injuries of the pelvic floor have depended largely on the originators' varying conceptions of the anatomy and physiology of these structures. The reason for so many different types of operation undoubtedly depends on insufficient knowledge of the anatomy. One naturally would suppose at the present time that this subject had been completely worked out, but a careful examination of the works of various anatomists shows that there is very little agreement as to the exact structure of the female pelvic floor. In a general way the works of the English and American anatomists have been characterized by insufficiency of detail in the treatment of the female pelvic floor, notwithstanding the fact that their descriptions of this region in the male is usually very complete and satisfactory. To this general statement there are, of course, notable exceptions, such as the article in Deaver's "Surgical Anatomy" and Peter

* Read before the Medical Association of the Southwest, Oct. 9, 1907.