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The etiology of diarrhea in children

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THE ETIOLOGY OF DIARRHEA IN CHILDREN

Beyond a doubt, the primary intent of every medical practitioner is to cure or alleviate the symptoms of disease. His ability to accomplish this end is directly dependent upon his understanding of the nature of the cause and course of the disease processes. It is with this in mind that it has been undertaken to review the etiological factors of diarrhea in children.

Holt (28) states that "The term diarrhea is used to cover all conditions attended by frequent, loose evacuation of the bowels."

The diarrhea which is to be considered in this paper has been synonymously called "alimentary intoxication" by Finkelstein, "alimentary toxicosis" by Czerny, "anhydremic intoxication" by Marriott, "gastro-enteric intoxication" by Holt, and catarrhal enteritis, infectious diarrhea, cholera infantum and summer diarrhea. In an epidemic of diarrhea in Kansas City, Kansas, with a large number of deaths, the death certificates contained one-hundred and sixty-nine terms given as the cause of death, all due to summer diarrhea (28).

Diarrhea is the most frequent form of infant disturbance that exists and is a more frequent cause of death than all the acute infectious diseases combined. (5) (11) (12). During the last century the prolongation of the span of human life has been made possible through the reduction of infant mortality.

Among the earliest attempts to classify the diarrhea are the works of De Loré, 1837 and Veliker, 1862, who thought that the symptom of vomiting and diarrhea was the result of inflammatory condition in the digestive tract. Fifty years later, Wirdenhofer, at the Allgemeine
Krankenhaus in Vienna, suggested a classification based on the anatomical and pathological changes in the intestinal tract. Their classification marked the anatomico-pathological era. During this period the terms "gastric catarrh", "gastro-enteritis", "ilio-colitis" and "duodenitis" were prevalent. The classification was short lived because Hubner showed that there was a wide variety and inconstancy of pathological changes. Following this, some satisfactory progress was made through metabolic studies and the term "nutritional disturbances" was introduced by Czerny (4).

In order to approach the subject methodically it has been necessary to accept a classification of diarrhea. That which follows is by Dr. James W. Reed (10).

I. Non-infectious

   Caused by improper proportions of fatty carbohydrates and proteins

II. Infectious

   A. Primary or enteral

   B. Secondary or parenteral following acute infections of air passages, middle ear, mastoid, tonsils etc.

   A very acceptable classification of the etiological factors of diarrhea is given by Dr. John Van Cleve (15).

Etiology of Infantile Diarrhea.

I. Predisposing causes

   1- Age

   2- Season

   3- Teething
4- Defective hygiene
5- Bad feeding
6- Parenteral infection

II. Exciting Causes

A. Unknown causes

1. Bacterial infection of some portion of the intestinal tract.
2. Metabolic or physiologic derangements so that the intestine contains a large amount of undigested or unabsorbed food which is split by the stool organism into irritating end products.

B. Known causes

1. Dysentery bacillus of the Flexner group
2. " " " Shiga "
3. " " " Mount Desert
4. Bacillus typhosis
5. Amoeba dysenteria
6. Bacillus Tuberculosis

An attempt has been made to follow the above outline throughout this discussion.

AGE. Diarrhea may be present during any of the childhood years. It is most prevalent under two years of age. (1) (12) The highest mortality is during the second half of the first year of life, provided that period is in the summer season (1). The first function which a child must perform is to adapt itself to its environment in order to continue its existence. The young infant has not yet met and built
up sufficient resistance against invading micro-organisms. Nor has it properly adjusted itself to external temperature changes, thus the internal temperature of the child fluctuates somewhat with the external temperature. The heat regulatory center is unstable. As a result we have marked and rapid febrile changes with minor nutritional disturbances and infections.

In Baltimore Dr. Lawson Wilkins (19) had 623 normal white infants under two years under his observation from June 1 to Nov. 1, 1925. Forty per cent. of these children developed gastro-intestinal disorders during the five-month period.

SEASON. An understanding of the relationship between external temperature and the instance and severity of diarrhea is less than two-hundred years old. Benjamin Rush, 1745-1813, (5) signer of the Declaration of Independence, Treasurer of the United States Mint, founder of the first dispensary in America, the Philadelphia dispensary, is best remembered for his medical work and his account of summer diarrhea. He was the first to relate the high temperature of summer to the incidence of diarrhea. He observed that severity of the diarrhea was proportional to the external temperature. He also observed that a single cool day would sometimes abate the course of the disease. It is beside the point, but it is interesting to note that Dr. Rush also states "In some cases the disease became so severe in Philadelphia as to require bleeding to cure it. In some cases two and three bleedings were necessary for that purpose."

Repeated observations in recent years have shown that the disease is most prevalent in the summer months (1) (6) (19) and that the maximum is reached during July and August (9). Several authors have con-
sidered the summer heat as the most important factor in the etiology of diarrhea (8) (19).

Dr. Arnold (8) has made an excellent study of the relationship of infant mortality due to diarrhea and climatic changes in New York City.

The Chart for 1892 shows a sudden elevation of the percentage of diarrheal deaths, beginning in the fourth week in June, with a temperature above 85°. The high mortality was a result of improper refrigeration of food and the fact that the milk was not pasteurized and other sanitary measures were not stressed. It is also evident that the susceptible children died before the maximum temperature was reached in August. (Figure 1).

FIGURE 1. Weekly Diarrheal Deaths 0-4 Years, New York City, 1892, Compared With the Mean Weekly Effective Temperature. Black Line -- Diarrheal Deaths 0-4 Years in Ratio Per 100 Deaths from Diarrhea of Same Age Group. Red Line -- Effective Temperature.
In 1896 the relationship was very similar with a mortality above seven per cent. for a period of three weeks following a steady progressive elevation of temperature. (Fig. 2).

FIGURE II. Weekly Diarrheal Deaths 0-4 Years, New York City, 1896, Compared With the Mean Effective Temperature. Black Line -- Diarrheal Deaths 0-4 Years in Ratio per 100 Deaths from Diarrhea of Same Age Group. Red Line -- Effective Temperature.
In 1908 there was a delay of several weeks between the elevation of temperature and the increased mortality, and the mortality rate was much lower than that shown in the previous charts. It is quite evident that there were some definite steps being taken in the prevention and treatment of the condition. (Fig. 3).

FIGURE III. Weekly Diarrheal Deaths 0-4 Years, New York City, 1908, Compared with the Mean Effective Temperature. Black Line -- Diarrheal Deaths 0-4 Years in Ratio per 100 Deaths from Diarrhea of Same Age Group. Red Line -- Effective Temperature.
Figure III

Percent Mortality

Effective Temperature

1908
In 1920 there was again a lag in mortality behind the elevation of temperature. Compulsory pasteurization of all but certified milk was adopted in 1914 with a corresponding drop in infant mortality. Throughout these charts there is a gradually diminishing death rate and the onset of the higher mortality curve lags further and further behind the temperature curve. (Fig. 4)

**FIGURE IV.** Weekly Diarrheal Deaths 0-4 Years, New York City, 1920, Compared with the Mean Effective Temperature. **Black Line** -- Diarrheal Deaths 0 - 4 Years in Ratio per 100 Deaths from Diarrhea of the Same Age Group. **Red Line** -- Effective Temperature.
Figure IV

Percent Mortality

Effective Temperature

1940

May June July Aug Sept Oct
In 1924 there is the most satisfactory chart. The increased mortality did not occur until the seventh week of elevated temperature. This is partially due to the more widely used acidified whole milk as routine infant food during the summer months, as recommended by Drs. Marriott and Davidson. (9) (Fig. 5).

FIGURE V. Weekly Diarrheal Deaths 0-4 Years, New York City, 1924, Compared with the Mean Effective Temperature. Black Line -- Diarrheal Deaths 0-4 Years in Ratio per 100 Deaths from Diarrhea of Same Age Group. Red Line -- Effective Temperature.
The theoretical reasons for the relationship of temperature to diarrhea will be discussed a little further on.

TEETHING. It is very doubtful whether teething actually is a predisposing factor to diarrhea. It is mentioned as such by a few authors (16) (27). However it is more probable that it just occurs coincidental with the prevalence of the diarrheal diseases. Perhaps there is a lowered tolerance for food during the teething age. Because there is an increased salivation accompanying teething, it should be considered as a possible predisposing factor. It will be later shown that anything that increases salivation must be considered a predisposing cause.

DEFECTIVE HYGIENE. Diarrheal diseases are much more prevalent in the cities among the poorer classes of individuals (30) (31) in homes where proper ventilation is impossible, where no means of cooling the living quarters are available. The children are too frequently overdressed (3) not allowing proper evaporation from the skin. Whenever the temperature and humidity become sufficiently high so that evaporation from the body surface is no longer possible then the internal body temperature is elevated (3). Anything that causes an elevation of temperature of the body causes digestive and metabolic changes as will be later proven. In New York City (30) there have been experimental summer camps established, especially for tenement house children, where the diet and hygiene and clothing has been supervised throughout the summer months and there was a very satisfactory reduction in infant mortality.

The adoption of proper feeding bottles and nipples which are easily cleaned is a forward step.
Too frequently the poor homes are overcrowded and the children are given food which has not been properly refrigerated, due to absence of means and prevalence of ignorance of the parents, and the milk supply is neither certified nor pasteurized.

**IMPROPER FEEDING.** Many authors who have discussed the subject of diarrhea have enumerated the factors in etiology according to their importance. First, they have considered external temperature, and second in improper feeding or artificial feeding (11) (18). It is valuable to show the greater incidence of diarrhea in artificially fed infants.

There is prevalent in district of Japan an infectious diarrhea which is very similar to our summer diarrhea and is called "Ediri" (7). It is accompanied by high mortality. The disease has never been seen in a breast fed infants (7).

Most commonly the parenteral infections which are an important predisposing cause of diarrhea are seen in malnourished (18) and artificially fed infants (16).

In Dr. Wilkins' (19) series of 628 cases observed throughout an entire summer, one-fourth of the infants were exclusively breast fed. Of this group ten per cent. developed simple diarrhea and none developed dysentery. Of the remaining three-fourths, or the artificially fed group, thirty-three per cent. developed simple diarrhea, ten and one-tenth per cent. had typical dysentery and six and nine-tenth per cent. had probable dysentery. The group of artificially fed infants was subdivided into three groups. The first group was given butter milk; the second group, whole milk formula; and the third group was given protein milk. The incidence of diarrhea was about equal in each of the three
groups of artificially fed infants.

A lot is written about fermentative diarrhea in children on a formula with a high carbohydrate diet. These children may get along nicely for a period of several months (18) (22), then with the onset of hot weather or with the development of a parenteral infection, a diarrhea is started and of course it is a fermentative diarrhea because the predominant media in the intestine is a fermentable carbohydrate. This will be mentioned in greater detail in a discussion of metabolic disturbances.

PARENTERAL FACTORS. In the outline of Dr. Van Cleve (15) which is being used as a guide throughout this paper, parenteral infection has been placed with the predisposing causes of diarrhea. So we leave the topic in that position even though it seem more probable that it should be considered an exciting cause. The exact way in which parenteral infection acts to cause diarrhea cannot definitely be shown. Possibly the toxin absorbed from the site of infection is a definite irritant and stimulant to the gastro-intestinal tract. The best explanation is that it acts on the same basis as does an external temperature and causes an increase of the body temperature with a resulting change of secretion along the gastro-intestinal tract (2) and a greater alkalinity of the intestinal contents and a number of resulting metabolic changes to be discussed later.

The diarrhea in parenteral infection is not unlike the usual summer diarrhea. Whenever a patient with diarrhea is seen there is too common a tendency to attribute it to a primary gastro-intestinal upset. Every case should be thoroughly examined for a focus of infection outside
the intestinal tract. (13). Dr. Crawford (13) has been so bold as to state that ninety-nine per cent. of the diarrhea is due to parenteral infection, the most common causes being otitis media, mastoiditis, pneumonia, scarlet fever, tonsillitis, meningitis and pyelitis. (6). Chronic focal infections as inbedded tonsils, adenoids, arthritis, endocarditis may result in diarrhea which flares up at intervals of two or three weeks or over periods of months.

At Iowa City Dr. Jeans and Dr. Floyd (16) have studied the relationship of upper respiratory infection to diarrhea. In their report of cases the majority were artificially fed infants with an average age of five months. Most of their cases have occurred in the fall and winter and early spring when upper respiratory infections are most prevalent. In all of their cases of diarrhea, during the past several years, they have found a parenteral infection to which they have attributed the diarrhea. The majority have been infected mastoids and paranasal sinuses. Sometimes the focus, when in the mastoid, was not found except at autopsy.

Dr. Marriott (2) believes that sometimes the parenteral infection precedes the diarrhea and is the cause of it and at other times the parenteral infection is secondary to and the direct result of the gastro-intestinal upset. He has records of cases of otitis media from which bacillus coli has been isolated. His theory is that the colon bacillus has reached the stomach by the reversed peristalsis and then the patient has vomited and the organism has found its way into the eustachian tube. The resulting otitis media has set up a vicious cycle.

It seems quite safe to state that the diarrhea in winter and spring, rarely as they do occur, are the result of a parenteral in-
fection, except in instances in which poisoned or irritating food has been consumed.

**EXCITING CAUSES.** The first group that is to be discussed under this heading is of unproved value. In the specific dysenteries there is a known organism with definite pathologic lesions along the tract. These will be discussed later.

**UNKNOWN CAUSES.** In the non-specific group of diarrhea there have been found the normal stool organism which are sometimes found in increased numbers plus other organism which appear sometimes but not constantly in the normal stool.

In parenteral infection, as a Staphylococcus or Streptococcus otitis media or pharyngitis accompanied by diarrhea, the staphyloccoci and streptococci have been found in the stool cultures (2). It has not however been shown that these are intestinal lesions which can be proved the result of the pyogenic organisms.

In the majority of summer diarrhea the diarrhea is usually not the result of infection of the intestinal tract (15) but the result of changes in the reaction and position of the flora of the intestinal content.

In an analysis of one hundred-four diarrheal stools in children, Fothergill (17) found an atypical paratyphoid organism in forty-six stools or 44.2 per cent. These organisms are different than any known genus of gram negative intestinal bacilli. They are non-motile, gram negative, do not liquify gelatine, do not ferment lactose. Agglutination reactions within the group show that they are not a homologous group like the typhoid or paratyphoid groups. These organisms have as yet not been proven to have a definite role in the etiology of diarrhea.
Bacillus Welchii is sometimes present in the normal adult stool, rarely in the normal stool of children. Pounds (27) reports that in some epidemics of diarrhea this gas bacillus has been found in a large percentage of the stools.

METABOLIC AND PHYSIOLOGIC DERANGEMENTS. Before entering upon the disfunctions of the gastro-intestinal tract, the normal function and reactions will be considered.

It has been commonly taught that the reaction of the duodenal content is alkaline. The secretions of the duodenal mucosa and the liver are alkaline and in a fasting animal the duodenal content is alkaline, but duodenal analyses by several recent workers have shown that the content is acid in reaction, the greatest part of the time. Dr. Lloyd Arnold (8) states that normally the stomach, duodenum and jejunum are relatively acid, or at least contain acid buffered substances. His method of investigation is approved by several workers in the field and is commended by Mann (32). To obtain samples he brings a loop of the gut to be analyzed to lie just under the skin and his samples are secured by placing a needle into the gut. The method is not commonly used because only a few samples can be secured daily. Dr. Wilbur C. Davidson analyzed the duodenal content of twenty-five children, some normal, some with different types of diarrhea. In twenty-three specimens on six normal children he found the mean pH of the duodenal content to be 6.2. His method is not wholly satisfactory because he passed a stomach tube into the duodenum and it is possible that some of the stomach contents passed into the duodenum during the extraction of the sample. He discarded the first fluid extracted, however, in order
that he would not be aspirating any acid directly from the stomach. A table of the analyses will follow later. Drs. F. C. Mann and J. L. Bollman (32) at the Mayo Foundation, Department of Physiology, have done repeated determinations on dogs. A loop of intestine was brought out through the skin and aspirations were made through a tube. The dogs lived apparently normally for several years. The duodenal content of the normal fasting dog varied from a mean PH of 7.0 to 7.6. A few dogs with a hyperchlorhydria had a PH as low as 4.6. After feeding the dog a normal diet the acidity of the duodenum became immediately apparent becoming a PH of 4.2 usually and sometimes as low as PH 1.8. After two hours the acidity began to diminish and usually reached the point of neutrality in six hours.

Now, in a normal infant we are dealing with an individual far different from a fasting animal. So it seems logical to assume that in an infant, which is being fed at three to four hour interval, that the duodenal content would never reach the point of neutrality but would always remain markedly acid in reaction.

It has also been observed that the stomach, duodenum and jejunum of normal individuals is relatively free from the intestinal flora, and that this sterility is dependent upon the acidity of the contents. (2) (3) (22) (26). The same individuals have shown that in the diarrheal diseases of infancy and childhood the stomach and duodenum contain large amounts of the normal fecal flora. Moro, a German author, (8) points out that by alkalinizing the duodenal tract by injection of alkaline solutions directly into the duodenum that the fecal flora enters the duodenum. Davidson (22) shows that in patients with a heavily infected
duodenal content, the cultures of the content become sterile within one-half hour following administration of lactic acid milk feeding.

Now we have shown that the duodenal content is normally acid and relatively sterile due to the hydrochloric acid secreted by the stomach. Anything that diminishes the HCl secretion will tend to make the duodenum less acid and less sterile.

In Parenteral infections, especially of the upper respiratory tract (2), there is an increased salivation with a direct neutralization of a part of the gastric acidity. This may have a small role in predisposing to a diarrhea.

Anything that elevates the temperature of the individual, whether it be external temperature, fever from infection, or what not, causes a diminished gastric secretion. (2) (9) (25).

Davidson (25) has done gastric acidity determinations on normal children and those with a pyrexia. His results follow:

<table>
<thead>
<tr>
<th>Body Temperature of Infants</th>
<th>Number of Specimens</th>
<th>Number of Patients</th>
<th>Mean PH of Gastric Contents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>22</td>
<td>7</td>
<td>PH 4.1</td>
</tr>
<tr>
<td>100°-101°</td>
<td>14</td>
<td>4</td>
<td>PH 5.9</td>
</tr>
</tbody>
</table>

In acute illness in children it is common knowledge that a large amount of gastric acidity is lost by vomiting.

The question presents itself, "Just what causes the diarrhea?"
The answer is undetermined. Perhaps it is the result of the fecal flora having attained a higher than normal level in the intestinal tract, acting as a direct irritant to the intestinal mucosa, causing increased peristalsis. More probably, however, it is the result of an increased amount of fecal material, including skatol, indol in the intestine. The increased
amount is the result of the fecal flora coming into a great abundance of media before there has been time for intestinal absorption. It is known that there is a toxic substance absorbed from the intestine in diarrhea. In animals with a diarrhea, extracts from the intestinal mucosa have been injected into normal animals resulting in twitching, fever, convulsions, diarrhea and death. Systemic blood from a dog with a diarrhea has been injected into normal dogs with a mildly toxic reaction, while portal blood similarly injected gives a markedly toxic reaction. Dodd, Minot and Aspin (23) thinking that this might be due to hyperguanidinemia undertook the following experiments. Hyperguanidinemia was produced in dogs and convulsions, pyrexia, twitching, and sometimes death resulted. The normal blood guanidine of infants is 0.3 to 0.4 mgm. per cent. Determinations were made in five infants with diarrhea complicated by anhydremia. The results follow:

<table>
<thead>
<tr>
<th>Case</th>
<th>Guanidine (mgm. per cent.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0.59</td>
</tr>
<tr>
<td>II</td>
<td>1.00</td>
</tr>
<tr>
<td>III</td>
<td>0.86</td>
</tr>
<tr>
<td>IV</td>
<td>0.87</td>
</tr>
<tr>
<td>V</td>
<td>1.20</td>
</tr>
</tbody>
</table>

**KNOWN CAUSES OF DIARRHEA.** In this class are listed the organisms which are proved to be the cause of diarrhea and which are associated with definite intestinal pathology.

Typhoid fever is rarely seen at the present time.

The paratyphoid dysenteries are not uncommon. An atypical group of paratyphoid organisms was found in 44.2 per cent. of the stools analysed by Fothergill (17).
Amoebic dysentery caused by entamoeba histolytica is a chronic disease, characterized by recurrence, most common in the tropical countries but occasionally seen in the United States.

In 1898 and 1899 Shiga found an organism constantly present in stools in a Tokyo epidemic. This dysentery organism now bears the name of Shiga and is prevalent in the Far East and Western Hemisphere.

In 1900 Flexner isolated an organism which he determined to be the cause of an epidemic of dysentery in the Philippine Islands. In 1902 the same type of organism was isolated from the stools of children with diarrhea in America.

The Mount Desert dysentery bacillus is seen more frequently than the Shiga or Flexner groups but the course of the disease is less severe.

Dr. Wollstein (34) in stool analyses of eighty-six cases of diarrhea from July to September found three stools positive for the Shiga group, five positive for the Flexner group and twelve for the Mount Desert Group.

In the United States ten per cent. of the true dysenteries are the Shiga group and eighty per cent. are the Flexner or Mount Desert group. Sixty-six per cent. of the mortality occurs in the Shiga group, (15).

An analysis of 104 diarrheal stools by Fothergill (17) showed the presence of true dysentery bacilli in 5.7 per cent.

Dr. Lawson Wilkins, Baltimore (19), found that 7.8 per cent. of the 628 children under his observation from June 1 to November 1, 1925 developed typical dysentery with a mortality of
During the summer of 1926, forty per cent. of the gastrointestinal disturbances admitted to the Harriet Lane Home for Children were dysentery, with a mortality of 24.8 per cent. (19)

The dysentery organisms are transferred very similarly to those of typhoid fever by water, milk, food, fresh vegetables, by carriers and fomites.

CONCLUSIONS

1. The most potent factor in the etiology of summer diarrhea is external temperature.

2. The second factor in importance is artificial feeding.

3. Otitis media and mastoiditis are frequently associated with diarrhea.

4. The gastric secretion is diminished by anything which elevates the body temperature.

5. The duodenal content is acid for six hours following a feeding.

6. The stomach, duodenum and jejunum are normally comparatively sterile.

7. The stool flora inhabit the duodenum during a diarrheal disease.

8. In diarrheal diseases a toxic substance is absorbed from the small intestine.

9. There is probably a definite toxin associated with diarrhea but at present it is an undetermined factor.
CASE REPORTS
Case Report - No. I.

B. G. McF. #32514 - White, American female child 3½ years old entered the University Hospital on July 25, 1930 because of vomiting and diarrhea. Onset of vomiting was on the evening of July 23d. Diarrhea followed the same evening and persisted in spite of all medication. With the onset of abdominal pain on July 25 the child was brought into the hospital. Temperature on entrance 104.2°. The vomitus was bile stained. The stools were watery and frequent, 16 to 18 in 24 hours.

On July 25, a sister and the mother both developed a severe diarrhea.

PHYSICAL EXAMINATION: Head, neck and chest, negative. Abdomen soft, not distended, tympanitic, tender. Skin hot and dry.

Rectal examination shows anal dilatation, inflamed rectal mucosa, mucous discharge.

LABORATORY: Blood - Hemoglobin 80%, R. B. C. 5,350,000. W. B. C. 7,800. Differential 40% Lymphocytes, 49% Polymorphs, 4% Monocytes, 3% Basophiles, 1% Eosinophiles.

Blood Culture - Negative.

Widal - Negative.

Some of the stools examined shows dysentery bacilli.

During course in the Hospital there were 9-12 stools in 24 hours. The patient became markedly dehydrated. Fluid was given by proctoclysis, by mouth and subcutaneously. Glucose and barley water was also given by mouth.

On July 27 pulse became progressively weaker and more rapid, the face and extremities became cyanotic and the child died, a cardiac death.
AUTOPSY REPORT:

Liver shows advanced parenchymatous degeneration.

Stomach normal. Small intestine, lower jejunum and ilium shows accumulation of mucous and an intense green color to the intestinal content. Large intestine, rectal mucosa injected. Pelvis shows a fibrinous exudate in the cul de sac.

Diagnosis: Acute enteritis and colitis.
Case Report - No. II -

B. T., white American male child, 2½ years old, came into the University Hospital for the first time on Dec. 7, 1930, because of vomiting and frequent stools of one day duration. Patient was sent in by an outcall student with a tentative diagnosis of acute appendicitis or acute enteritis. On December 5 the child developed an acute upper respiratory infection. On December 7 he vomited anything that was given by mouth and the diarrhea was first manifested. During the day there were ten watery stools. The stools were green and contained mucous.

PHYSICAL EXAMINATION:

Nose - Mucous discharge
Ears - Negative
Pharynx - Injected
Neck - Palpable submaxillary and anterior cervical lymph nodes.
Chest - Negative
Abdomen - No rigidity, general tenderness with deep palpation

LABORATORY: R.B.C. 5,300,000, W.B.C. 15,600, Differential Lymphocytes, 26% - Polymorphs. 74%.
Urine - Acid, Sp. Gr. 1.031 - Alb 1 plus, Dicetic, positive.


Diagnosis: Acute upper respiratory infection with a secondary diarrhea.
Case Report, No. III -

J. H. - Age 8, white, American, female, was seen on out-
call from the University Hospital on March 21, because of fever and
soreness of four days duration. Medication previous to the first call
had been aspirin, grains V three times a day. In eliciting the his-
tory it was learned that on the second day of the illness the girl had
and watery bowel movements. On the third and fourth days the child
had about five watery stools.

On the evening of the fourth day when the child was examined
the complaint was pain in and around the left ear. The temperature was
104°, pulse 150, and the respiration rate 22. The pharynx was slightly
injected, the right ear was normal but the left ear showed tenderness
over the mastoid and the ear drum was injected and markedly distended.

A member of the University Staff was called and a paracent-
tesis was done and a thick creamy pus discharged. Following this the
ear was frequently wiped out and drops were instilled by the mother.
The following day the temperature was 99, the ear was discharging and
the child had two soft bowel movements. The second day the temperature
was normal, the ear continued to discharge and there was one formed stool.
The tenderness over the mastoid and the discharge from the ear persisted
until the outpatient service changed to another group.

This case is quite characteristic of the diarrhea of parent-
teral origin. Like most of these cases, the diarrhea ceases when the
focus of infection is adequately treated.
Case Report - No IV -

J. McF. - White, American, male, age 9, entered the Arnold Hospital early in August, 1931, because of abdominal pain and frequent watery stools. The diarrhea had been present for five days before a physician was called. Medication had consisted of frequently repeated doses of castor oil. There were 15 to 18 stools daily.

On admission the temperature was 104.8°, pulse 140 per min. The physical examination was negative except some evidence of dehydration and some tenderness over the abdomen.

The patient was given nothing except water and medicine by mouth for 24 hours. The medicine consisted of paregoric each four hours. The first day in the hospital there were four loose bowel movements. The second day the temperature was normal. A little cooked cereal was given. There were two partially formed stools. The patient was dismissed on the fourth day.

The summer heat was probably the greatest cause of the onset of the diarrhea and the castor oil in repeated doses caused a definite intestinal irritation and aggravated the diarrhea.
The two following cases were seen with a member of the Staff of the University and Methodist hospitals in November, 1929, and are reported here with his permission. The two children are brother and sister and became ill the same day, a week before their entrance into the hospital. A few days after their entrance the mother and another child of the family developed similar symptoms at their home.

Case Report No. V -

S. P. - A 15 months old female, white, American child entered the Methodist hospital in Omaha on November 6, 1929, following an illness of one week, complaining of frequent, loose bowel evacuations, convulsions, weakness and coma. Examination on entrance showed an undernourished, emaciated, dehydrated, comatose child. The eyes were sunken, glazed like and could not be closed because of fluid loss.

Ear, nose, throat, lungs - negative.

Heart sounds very faint.

Abdomen sunken, skin loose and stood in folds when picked up between the fingers.

Two stool examinations while in the hospital both showed soft yellow fecal matter which contained blood, pus and mucus in large quantities. The daily stools numbered from seven to twelve in twenty-four hours.

The urine showed albumen and pus cells.

The temperature varied between 103.8° and 106.4°. The patient died a cardiac death on the fifth day in the hospital. Treatment consisted of fluids, orange juice, five per cent. glucose, protein milk and paregoric by mouth; fluid intra-peritoneally and a
transfusion of 250 cc. of blood. Castor oil was used in the eyes.

Case Report No. VI -

E. P. - 3 years, white American male child entered the Methodist hospital in Omaha on Nov. 6, 1929, the complaints being convulsions, fever, emaciation and diarrhea of one weeks duration and unconsciousness of one days duration. Examination showed an emaciated, dehydrated anemic comatose child.

Eyes had a sunken glazed appearance.
Ears, nose, throat, chest and abdomen were negative.

During the twenty-one days in the hospital the white count varied between 17,000 and 35,000 cells. Urine repeatedly showed traces of albumen. Two stool examinations showed soft yellow consistently with blood and mucus. During the first eight days in the hospital the total number of stools per twenty-four hours varied between eleven and twenty-two.

The temperature on admission was 105.6°, which gradually declined to normal on the fifteenth day. The first day he received nothing but paregoric by mouth in doses of Gtt's. XV every three hours. The second day he was given glucose, five per cent. and orange juice. The third day, vegetable soup, cereal and tea. On the fifth day he was placed on a protein milk formula. On the fourteenth day some solid food was added. Patient left the hospital on the sixteenth day.

These two cases, Nos. V and VI, present typical picture of a dysentery bacillus type of infection. Unfortunately stool cultures were not made, so a bacteriological report is not available.
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