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SOME OBSERVATIONS

on

ZYMOTIC ENTERITIS

with special reference

to

CHOLERA INFANTUM

by

D. L. MORRISON.

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It has for long been a source of wonder to me that while Infant Mortality has received much attention especially of late years, comparatively little has been done to enquire into and alleviate Zymotic enteritis which is by far and away the chief cause of this huge Mortality.

The Infant Mortality from causes other than diarrhoea has been reduced in late years, but the Mortality from Diarrhoeal Diseases, and these diseases account for almost one-third of the Mortality in infants, remains unaffected in spite of our undoubtedly improved modern sanitation.

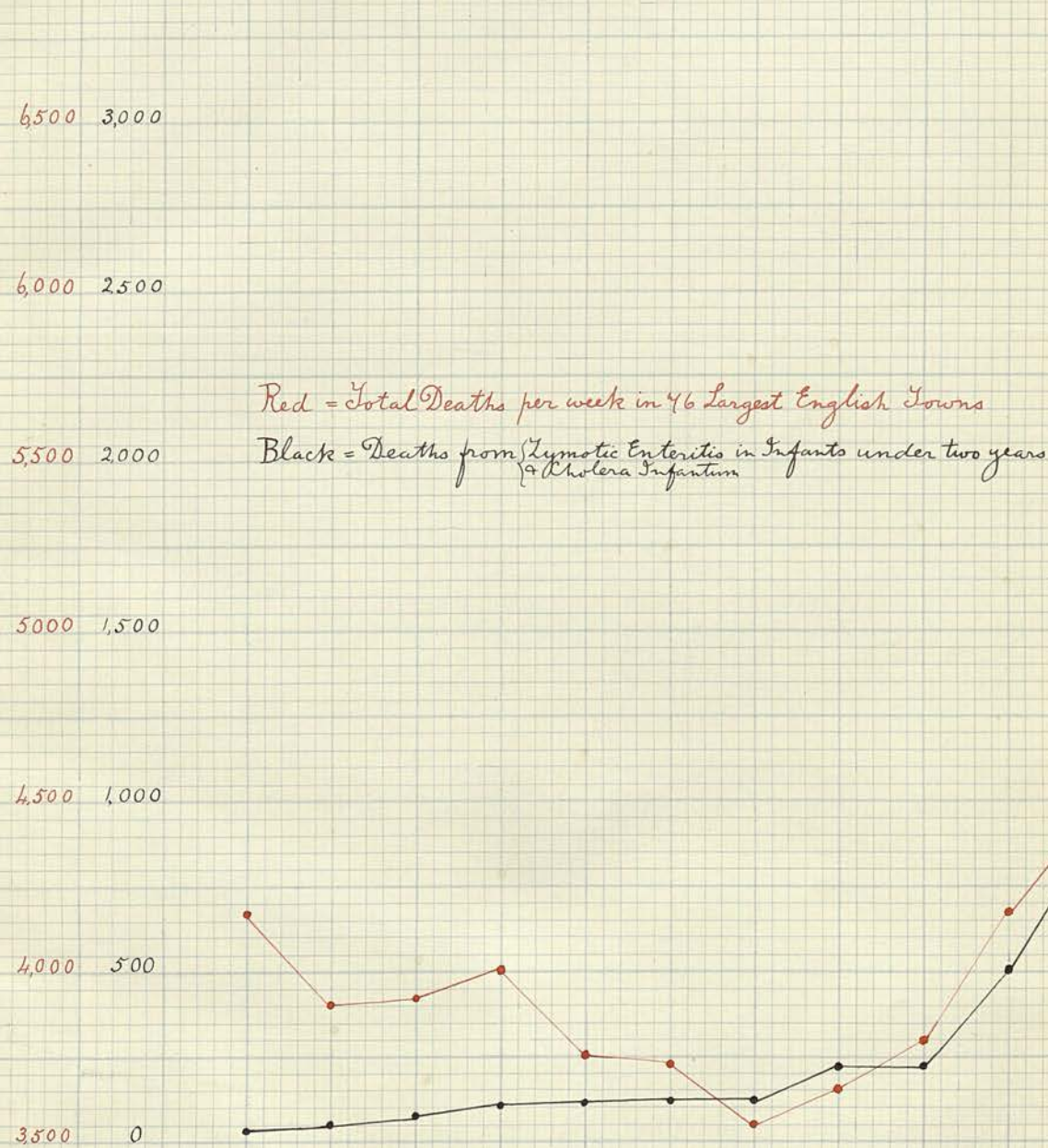
And again while the General Death Rate in England and Wales has declined in the past fifty years, the Infant Mortality has remained more or less stationary and has been even higher in the last ten years than in the first ten of the half century.

It has been pointed out that the Index of Deaths from Diarrhoeal Diseases is now practically the index of the Infant Mortality and also follows the curve of the Death Rate from General Causes in a remarkable manner as shown by the accompanying two charts.

A Chart to show what a striking relation the Mortality from the Diarrhoeal Diseases bears to the General Death Rate

For the year 1906. A year of high mortality from Diarrhoeal Diseases

Week ending: June 2nd 9th 16th 23rd 30th July 7th 14th 21st 28th Aug 4th 11th 18th 25th Sept 1st 8th 15th 22nd 29th Oct 6th 13th 20th



A similar Chart for the year 1900: a year of low mortality from the Diarrhoeal Diseases.

Week ending: May 26th June 2nd 9th 16th 23rd 30th July 7th 14th 21st 28th Aug 4th 11th 18th 25th Sept 1st 8th 15th 22nd 29th Oct 6th 13th

5,000 1,750

4,750 1,500

4,500 1,250

4,250 1,000

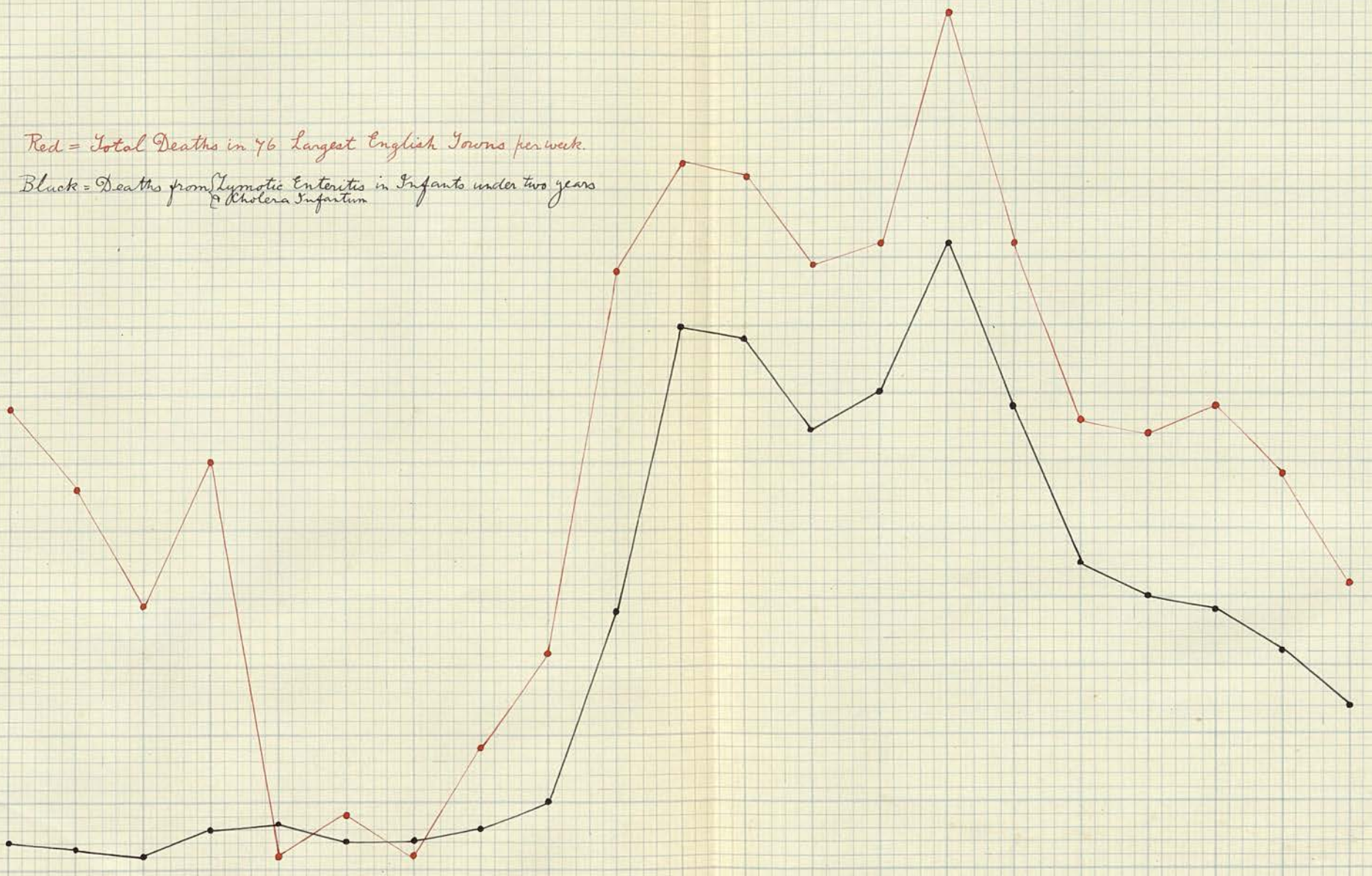
4,000 750

3,750 500

3,500 250

3,250 0

Red = Total Deaths in 76 Largest English Towns per week.
 Black = Deaths from Symptomatic Enteritis in Infants under two years
 & Cholera Infantum



Though the Diarrhoeal Diseases, with the special factors Zymotic enteritis and Cholera Infantum claim a Death Rate higher than Cancer and Phthisis put together they have not received one quarter of the attention that has been given to the last named.

I am inclined to think this is because Zymotic enteritis occurs in infants and in the great majority of cases in infants of the working classes.

But are not those lives of the greatest possible value? Are not the infants of to-day the citizens of to-morrow? And are not the working classes the very back bone of every nation?

It is down in the slums that this scourge rages, and its victims are helpless infants. This makes the disgrace to our civilisation all the greater that little should have so far been done to improve matters. The Medical Profession cannot be expected to cope with this scourge unaided. It is a blot on the nation in general and on those in public authority in particular. Considering these facts, and being fortunate in being, during the Summer of 1906, a Resident Officer to St. Mary's Hospital for Women and Children, Plaistow, London, E, in the Borough of West Ham, one of the most virulent hot beds of Zymotic enteritis, I determined to make a careful study of the disease, and

to take it as the subject of this Thesis.

Previous to the summer of 1906 I do not think wards in any Hospital had been provided specially and solely for cases of Zymotic enteritis. Patients suffering from this disease had been rather shunned by those responsible for the admission of cases into hospitals. For the 'true' cases had a reputation for dying, whatever treatment might be adopted, and besides it was pretty commonly recognised that these cases were infectious, and that all the infants in a ward might become infected from the admittance of one such case.

My senior, in residence at St. Mary's Hospital - Dr. J. S. Pearson and I were much impressed by the many conflicting and seemingly untrustworthy accounts of the disease in the standard text-books. Moreover the treatment seemed so ineffective, - for although almost every known drug had been tried, inquiries at many Children's Hospitals elicited the fact that the worst cases almost invariably died.

Amongst the local practitioners it was considered essential that the 'diarrhoea must be stopped at any cost.' The result was invariably fatal.

Dr. Pearson asked the Board of Management of our hospital for a Ward and nursing Staff exclusively for the Treatment and Study of Summer Diarrhoea. He was given practically entire control of his

suggested scheme.

A ward containing four beds on the first floor in a separate wing was provided and, for the last three weeks of the season, two extra beds were obtained in a small room off the ward.

I am very much indebted to Dr. Pearson for allowing me to take part in the scheme, and to make use of the large amount of clinical material amassed.

There were three special nurses who each did eight consecutive hours duty under the control of the Out-Patient Sister. The following Set of Rules was given to each nurse and hung about the Hospital. It helps to explain the manner of working the scheme.

RULES TO BE OBSERVED FOR THE SUMMER DIARRHOEA WARDS.

- I. No one of the three Nurses on Special Duty are to be allowed to enter the other wards of the Hospital, and no other member of the Nursing Staff, with the exception of the Out-Patient Sister, is to be allowed in the Diarrhoea Wards on any pretext whatever.

- II. The Nurses are to rinse their hands in 1-2000 Biniiodide of Mercury solution after feeding or otherwise attending one infant before going to attend on another case.

After handling the diapers the hands should be carefully washed, as it is believed that they are sources of re-infection.

- III. The soiled diapers are to be placed at once in 1-20 carbolic solution.

- IV. No food, except for immediate consumption, is to be kept in the Wards.

No feed must be given that has been allowed to stand in either of the Wards.

- V. The Albumin Water should be gradually heated with water to 70° or 80°.

This feed should not be less than 72° or more than 80° F. in temperature.

- VI. Milk feeds must be given at 100° F.
- VII. The Raw Beef Juice ordered is to be given at the time of exchange of duty, and brought over by the on-coming Nurse from the Dairy. N.B. Rule IV.
- VIII. The bottles, teats, thermometers, rectal tubes, etc., of each patient are to be kept quite separate and sterilised after each use.
- IX. Each child is to be weighed on admission, and again at 10 o'clock every morning. Every care must be taken with these weights and the weight of the clothes deducted.
- X. The character of the stools as regards consistence, color, offensiveness and re-action is to be recorded at the time of occurrence on the slate provided for the purpose.
- XI. Each Nurse is to fill in the Daily Record of the times of the feeds, stools, etc., and initial it before going off duty.

In the Out-patient Department of which I had entire control, I felt that considerable difficulty would present itself. For before the diarrhoea season began, the new cases I had to examine and treat amounted to over 400 per week. In addition there were the enormous number of old cases to be seen and

Epidemic Enteritis Chart.

TYPE.

Date

Register No.

Name

Age

Length of Illness in hours

Diarrhœa, how long

how often

Vomiting ,,

,,

Interval between 1st V. and 1st D.

Stools. *Character*

Temperature

Feeding

Breast fed until

old.

Fam. Hist.

Weight

Treatment adopted

Result

the general work of the hospital to be attended to. I wondered how I could possibly be able to cope with the work when the rush of cases of zymotic enteritis, not to mention the many cases of ordinary mild diarrhoea, began.

But I felt that here was a splendid field for the observation of the physical signs of the disease and the experimental use of drugs. I therefore did not let the opportunity slip and, in addition to filling up the ordinary Out-patient card which is supplied to every case, and brought by the patient on every visit to the hospital, I found time to fill up 120 charts such as the accompanying.

In a huge city where there are many Children's Hospitals it is impossible to keep an exact record of all the cases and note the ultimate result. I made the attempt but found it impossible to get precise figures as to our Death Rate.

Many of our cases probably went to other hospitals, dissatisfied that the diarrhoea had not been stopped, or else they may have called in a local doctor who stopped the diarrhoea and signed the Death certificate.

All I can say is that out of 665 cases of Zymotic enteritis and cholera infantum, we signed only 34 Death certificates.

Many of the worst cases I was happily able to follow up, to note the effects of treatment day by day, and ultimately to be able to pronounce cured.

It had become known in the East End that we had special wards for the disease and were giving it special attention and thus we had an unusual number of bad cases coming to our hospital, many moribund and having been refused admission elsewhere.

On reading over accounts of the disease in the standard text books I was much struck with the contradictory statements made as regards the character of the stools.

Thus I found that Dr. Holt in his 'Diseases of Children', 1903, page 377. says, "The stools are frequent, large and fluid of a pale green, yellow, or brownish color in the beginning, but as they become more frequent they often lose all their color and are almost entirely serous. The first stools are acid, later they are neutral and when serous they may be alkaline. In most cases they are odourless; in rare instances they are exceedingly offensive."

While Dr. John Thomson in his book on the Clinical Examination and Treatment of Sick Children (1898) page 279 says:

"At first, ordinary faeces are passed, along with much wind, and preceded by colicky pains.

"The motions succeed one another rapidly; their yellow color changes to pale green, grey or brown, and they are very offensive."

Again Dr. Osler in his 'Principles of the Practice of Medicine', 1901, page 510, says, "The stools are at first faecal in character, brown or yellow in color. The stools first passed are very offensive, subsequently they are odourless. The thin serous stools are alkaline."

And Dr. Waldo in the Milroy Lectures, 1900, says: The stools "are usually at first yellow and later become watery brown and offensive. In reaction they are acid."

Dr. Holt. Dr. Thomson. Dr. Osler. Dr. Waldo.

Faeces:

Color:

At first-pale
green, yellow
or brownish.

Ordinary

Brown or
yellow.

Yellow

Later - Lose all
color.

Pale green -
grey or
brown, then
reddish.

Serous

Brown

Reaction. Acid
then neutral,
then Alkaline.

Not stated.

Alkaline.

Acid.

Odour. Odourless,
in rare cases
offensive.

Very offen-
sive.

Very offen-
sive, then
odourless.

Offen-
sive.

Dr. Pearson and I therefore determined to attempt to clear up these seemingly contradictory statements and to find which description was the accurate one.

We determined to have records kept of every stool passed of each case admitted to the Diarrhoea wards.

On special charts were recorded the hour of occurrence, color, consistence, degree of offensiveness, and reaction of every stool immediately after it had been passed.

This we believe has never been done previously.

We quickly found that contradictory statements might easily arise unless a record of all the stools in an individual case was kept.

It was obvious that it would be useless to make a summarising dogmatic statement merely on the evidence of one or two stools from separate cases.

We also soon found that (a) stools on being kept often alter in re-action and color in a few hours, and (b) that stools non-offensive on being passed may quickly become most offensive on standing.

This might also possibly be in part the source of contradiction in the text books. It certainly accounted for apparently inaccurate statements of

the parents of infants under treatment.

Almost every case admitted was I think chosen from cases which had come up to the Out-patient Department and therefore the selection of suitable cases was left entirely to me.

As at first we had only four beds available, to select the most acute cases from such a large number as was daily arriving at the Hospital, required some care.

The cases of severe diarrhoea and sickness were first sorted out from the other patients by the Out-patient Sister and after they had all been carefully examined I proceeded to select cases for admission. In order to do this most efficiently Dr. Pearson advised that I should keep Holt's description of the leading physical signs and symptoms of Cholera Infantum prominently in view. ("Diseases of Children", Pages 376-377).

These leading physical signs and symptoms are:-

- (1) Previously unhealthy infant.
- (2) Very rapid development of cholericiform symptoms.
- (3) Vomiting, which may precede or begin simultaneously with the diarrhoea.
- (4) Stools which are frequent, large, fluid, alkaline and odourless.
- (5) Rapid loss of weight.

- (6) Depression of the anterior fontanelle.
- (7) General prostration and nervous irritation.
- (8) Temperature elevated.
- (9) Rapid pulse.
- (10) Respiration frequent.
- (11) Tongue red and dry.
- (12) Abdomen soft and sunken.
- (13) Almost insatiable thirst.
- (14) Urine small in quantity.

Thirty four cases in all were admitted as In-patients, and both from these cases and from the cases which continued to attend as Out-patients I am enabled to comment upon those leading symptoms which were more or less present in each case.

I. Previously unhealthy infant.

This unhealthiness could be accounted for by either

- (1) Previous illness.

Of the 34 cases admitted, 14 had histories of a previous attack of severe diarrhoea earlier in the same summer. Some of these had never completely recovered from this previous attack.

Thirteen had signs of early rickets. Five had had either measles, whooping cough or bronchitis the previous winter.

(2) Improper feeding.

Of the 34 In-patients, 13 had never been breast fed, 4 had been breast fed for less than six weeks, 8 for eleven months or more. Only two had been breast fed for a correct length of time.

Of the 120 Out-patient cases of which histories had been taken, 41 had been breast fed for six weeks or less, 65 for eleven months or more.

I found it impossible to obtain figures to show the relative frequency of the different foods used in those who were artificially fed, for in most of the cases the mothers had changed the feeding from cow's milk to condensed milk and vice versa indiscriminately.

All I can say is that of the 34 In-patients, 18 were being fed on condensed milk at the time of onset of their illness,

or (3) Family History.

What struck me very much on enquiring into the family history of the cases, was the frequency with which one got a history of phthisis in one or more of the near relatives. Only the father or mother, brothers or sisters, uncles or aunts were taken into account. Unfortunately I noticed the repetition of such a history too late to make statistics, but I particularly noted phthisis in one or more near

relatives in 24 Out-patients.

In 18 In-patients or 53% there was a phthisical history.

I may say here that no cases were admitted where the diagnosis was open to doubt, and the subsequent progress of the cases showed they were true cases of cholera infantum. Of 13 fatal cases amongst the In-patients a post-mortem examination was made in nine instances and in no one of these nine cases was found any trace of tuberculosis either miliary or any other type in the peritoneum, intestines, lungs or meninges.

II.

Very rapid development of Choleraform symptoms.

This extreme rapidity of onset is very characteristic and occurs in almost every case. The choleraform symptoms occur with particular rapidity in those cases where there has been a previous attack the same summer and, curiously, in those infants who have been previously the healthiest. A healthy child does not seem so liable to contract the disease but when it does the onset is of the most extremely rapid description. These observations have been confirmed by other observers I have spoken to including a Resident Medical Officer at the East London Children's Hospital, Shadwell.

An example of rapidity of onset of a second attack was case No. 1 of the In-patients. The child had been ill four days before being admitted for the first time. It was sent home from hospital in four days, being almost well and having gained 8 ozs. It continued to improve at home for three weeks and gained another 4 ozs, when it suddenly developed the acutest symptoms, was re-admitted to hospital, lost 25 ozs. in 36 hours and died. *v. case XVII on chart*

III.

Vomiting which may precede or begin simultaneously with the diarrhoea.

I have heard it stated that in true cases of Cholera Infantum as distinguished from the milder form of Zymotic Enteritis it is essential that the vomiting should commence before the diarrhoea. I do not think this is so for I have observed cases of true Cholera Infantum where the vomiting has begun simultaneously with the diarrhoea.

Undoubtedly in by far the greatest number of fatal cases the vomiting has commenced first and it may therefore be taken as an indication of the severity of the attack.

Amongst the In-patients the vomiting commenced first in 18 cases and in all those who had had a previous attack.

Amongst the Out-patients vomiting preceded the diarrhoea in 43 cases.

As Dr Pearson has shown there are two different stages of vomiting.

(a) The Primary vomit which at first consists of undigested food and to a large extent occurs only after the feeds. These vomits are of course acid in reaction. If uncontrolled this vomit later becomes a watery mucus tinged with bile. It is generally easy, by diet and drugs, to stop this primary vomit especially in those cases which are going to recover. This vomit invariably stops before the diarrhoea does.

(b) The Secondary Vomit. The primary vomiting has stopped, the diarrhoea is lessening in amount though probably increasing in frequency but the patient looks uneasy and is restless. The secondary vomiting commences, the diarrhoea stops or only blood is passed, the abdomen becomes swollen and the child has toxæmia written on every feature.

We considered this vomiting identical with the vomiting of an acute intestinal obstruction, This vomit occurs independently of the feeds, it has the appearance of white of egg and is often so

described by the mother, it averages from two to three teaspoonfuls in amount and is often alkaline in reaction. When this vomiting commenced we always gave the gravest possible prognosis for we found that it very frequently portended a fatal issue. If the case is going to improve the diarrhoea is re-started and the vomit becomes acid in reaction and gradually lessens in frequency. Unless intestinal peristalsis can be re-established and the diarrhoea recommenced it is impossible to control this secondary vomiting.

IV. Stools which are frequent, large, fluid, alkaline, and odourless.

As I have indicated a record was kept in which was stated, the colour, consistence, and reaction of every stool passed by each patient and also whether the stool was large or small in amount, whether it was offensive or not and the precise hour at which it was passed.

The reaction was arrived at by means of specially sensitised, glazed test-paper as supplied by Messrs Baird & Tatlock for ascertaining the reaction of the blood. The test paper was allowed free contact with the stool and then it was washed in neutral water. The nurses had standards to go by and it was easy for them to make such remarks as were made in the Records and a specimen of which is here shewn.

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To accompany them
by
D.C. Morrison

A chart giving details of all the cases admitted

Number	Sex	Age in months	Weight on admission	Below average weight at discharge	Gain or loss on discharge	Number of days in Hospital	How long ill before admission in days	Number of attacks	Temp. on admission	Maximum Temp.	Type of stool on admission	Type on last day	Pharyngeal
I.	F.	4 1/2	11 lbs.	2 lbs	+ 1/2 lb	4	3	1	97°	99.6°	Y. W. Alk.	Normal	
II.	F.	20	13 3/4	7	+ 1 1/8 lbs	6	2	1	98.4°	100.4°	G. W. Ac. O.	Normal	
III.	M.	22	20 1/2	2	- 1/16 lbs	5	5	1	98.6°	98.8°	Y. W. Alk.	N.	
IV.	F.	3	6 1/16	2	+ 1/2 lbs	6	1	2	96.8°	100.8°	Y. W. Ac.	G. Alk.	
V.	M.	24	16 3/4	7	- 2 1/2 lbs	30	1	1	101.6°	104°	Y. W. Alk.	N	
VI.	M.	5	10	0	- 1/8 lb	6	2	1	100°	100°	Y. W. Alk. O.	N	
VII.	M.	4	10	0	- 1 1/4 lbs	5	1	1	98.6°	98.8°	Y. W. Alk. O.	N.	
VIII.	F.	18	13 1/4	5 1/2	- 14 ozs	6	2	2	99°	99°	Y. W. Alk.	Y. W. Alk.	
IX.	F.	7	11 1/2	1 1/2	+ 8 ozs	7	3	1	100°	103°	Y. W. Alk.	G. Ac. Off.	
X.	F.	10	12 9/16	2 1/2	+ 1/2 lb.	4	13	1	96°	99.4°	Y. W. Alk.	G. Sl. Ac. F.	
XI.	M.	6	14 7/16	over	+ 1/4 lb.	7	5	1	99°	99°	Y. W. Alk.	N.	
XII.	F.	3	5 1/2	3	0	9	7	1	96°	99.8°	Y. W. Alk.	Y. W. Alk. B	
XIII.	F.	6	13 1/2	over	- 1 lb	11	6	1	97.4°	100	Y. W. Alk.	N.	
XIV.	F.	5	9 1/4	1/2 lb.	- 3/16 lb.	6	9	1	97°	102.2°	Y. W. very Ac.	V. frothy Ac	
XV.	M.	9	7 1/16	7	- 3/16 lbs	5	20+1	2	96°	99°	Y. W. Alk.	Y. W. Alk	
XVI.	F.	19	13 1/2	5		1	28+3	2	98.4°	105°	None at first	Blood	
XVII.	F.	8 1/4	11 7/16	1 1/2	- 1 9/16 lbs	2	21+1	2	98°	104.6°	Y. W. Alk. O.	Y. W. Alk. O. + Blood	
XVIII.	M.	13	15 3/4	2	- 1 oz	6	7	1	96.8°	100°	Y. W. Alk.	N	
XIX.	M.	6	11 9/16	0	- 1 lb.	8	12+1	2	100°	103.4°	Y. W. Alk.	Y. W. Alk	
XX.	M.	6	10 1/2	1	- 1 7/16 lbs	14	4	1	97.4°	101°	Y. W. Alk.	Alk. B.	
XXI.	F.	6	11 9/16	0	- 3 1/16 lbs	20	13+1	2	102°	104.4°	Y. W. Alk.	Y. W. Alk	
XXII.	M.	2	6 1/16	1	+ 1/4 lb	3	14+1	2	99.4°	100.2°	Y. W. Alk.	N	
XXIII.	M.	10	13 1/2	1 1/2	- 1 1/8 lb	20	9	1	98.4°	99°	Y. W. Alk.	N	
XXIV.	M.	12	14 9/16	3	+ 1/16 lb	3	7	1	100.4°	100.8°	Y. W. Alk. B.	Y. W. Alk. B	
XXV.	M.	8 1/2	12	1	0	3	20+1	2	98°	102.4°	Y. W. Ac.	Y. W. Alk.	
XXVI.	F.	6	9 1/2	2	- 1/4 lb	3	21+1	2	97.2°	97.4°	Y. W. Alk	Blood	
XXVII.	M.	4	8 7/16	1/2	+ 9/16 lbs	12	12+2	2	97°	102.2°	Y. W. Alk.	Y. W. Alk.	
XXVIII.	M.	17	19	0	- 1 lb.	8	2	1	102.2°	102.4°	Y. W. Alk.	N.	
XXIX.	M.	13	14 3/4	3	- 3/4 lb	11	20+1	2	100°	101°	Y. W. Alk	N	
XXX.	M.	26	18 1/2	8	0.	5	1	1	99.4°	100°	Y. W. Alk	N	No
XXXI.	F.	11	11 1/4	4	- 2 ozs	6	1	1	98°	99°	G. Ac.	N	No
XXXII.	M.	18	13	5	+ 1/2 lb.	5	28+1	2	98°	96°	Y. W. Alk. O.	N.	No
XXXIII.	F.	3	8 9/16	0	- 1/16 lb	18	5	1	99°	104°	Y. W. Alk.	N	Yes
XXXIV.	M.	16	15 15/16	0	+ 5 ozs	3	28+5	2	98.8°	98.8°	G. oily O.	N.	?

Y = Yellow Ac. = Acid
 G = Green B = Blood
 W = Watery F = Frothy
 O = Offensive v = very
 Alk = Alkaline sl = slightly
 N = Normal

to the Wards as In-patients

Rachitis	Whether Breast fed	Result	Remarks.
0	Never C.M.	Cured	Diarrhoea preceded Vomiting This is the same patient as No. XVII.
+	4 mths then N.M.	C.	D. preceded V.
+	Never	C.	D. " V by a few hours. Subnormal temp. (96°) for 3 days.
0	Never	Improved	D & V simultaneous. { First attack 2 months previously. Sibling since. Ten days after being discharged attended to large cervical abscess Died 1 1/2 days later
+	11 months	C	V. preceded D. Three months later very well & weighed 19 1/2 lbs.
+	3 weeks	C	D & V simultaneous { Temp subnormal from end of 1 st day to 5 th day. Quite well. 3 weeks later had another very acute attack & died at home after 36 hours illness.
0	6 weeks	C	D & V simultaneous. { Died 3 rd attack 7 weeks later. Three other children in family died of Lym. Entert. & 3 of Tubercular Peritonitis
+	12 months	Died	First attack 3 weeks previously. Never well since. Temp subnormal after 1 st day.
-	Never	C	D. preceded V. Had severe 2 nd attack 4 weeks later & died in London Hospital.
-	10 months	C	D. preceded V.
-	1 month	C	D. " V. Second attack at home 3 weeks later & died.
-	7 weeks	D	D " V. Admitted moribund
-	3 weeks	C	D " V.
-	Never	D	V " D. Onset very sudden.
+	Never	D	V & D simultaneous.
-	9 months	D.	Admitted moribund. Appendicostomy. (Tense abdomen). Marked Broncho-Pneumonia
-	Never	D.	D & V simultaneous. This is case No. I re-admitted Brain symptoms very marked.
+	12 mths.	C.	Another child had D & V at home and died
0	Never	D.	V. preceded D.
-	Never	D	V. " D. Discolouration of abdomen before death.
-	Never	Impr & D.	{ Recovered on 10 th day. Accidentally burned on 12 th day & died. Marked Br Pneum. { D & V simultaneous
-	3 weeks	C	V. preceded D. Died 3 rd attack two weeks later. 4 other children had died of D & V.
-	9 months	C	D & V simultaneous. 2 other children 4 & 5 had D & V at home. Healthy in Dec.
-	6 months	D	V. preceded D.
-	Never	D.	V. preceded D.
-	3 months	D.	V & D simultaneous
-	3 months	D.	D. preceded D Increased in weight - Pancreatic Emulsion. Died of Bronch Pneum.
+	18 mths	C	D & V simultaneous.
+	13 mths	C	V. preceded D. Measles Pertussis previous winter sibling since. Sibling in December.
+	14 mths	C	V " D Onset after eating shrimps. Strong in December.
+	11 mths	C	Healthy in December
+	13 months	C	V. preceded D.
-	Never	C	Died 6 weeks later at home: "wasting"
+	Never	C	V. preceded D.

C = Cured

V. = Vomiting

D = Died

D = Diarrhoea

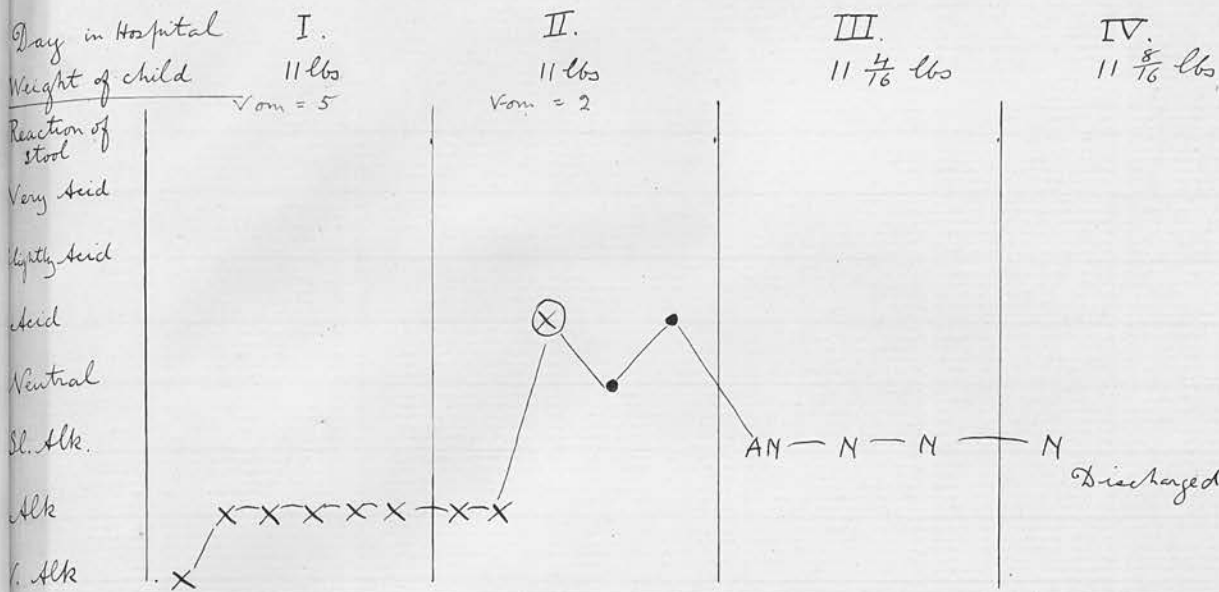
Impr = Improved

NB. There is often a considerable difficulty in getting a history as to whether the diarrhoea or the vomiting has begun first. In those cases in which one is told "they both began at the same time" generally it is found on further questioning that the vomiting began first.

Diagrammatic Charts showing the sequence of stools, as regards colour, odour, reaction and frequency; ¹ the frequency of the vomits, & the variation in weight of the patient of a few cases admitted as In-Patients.

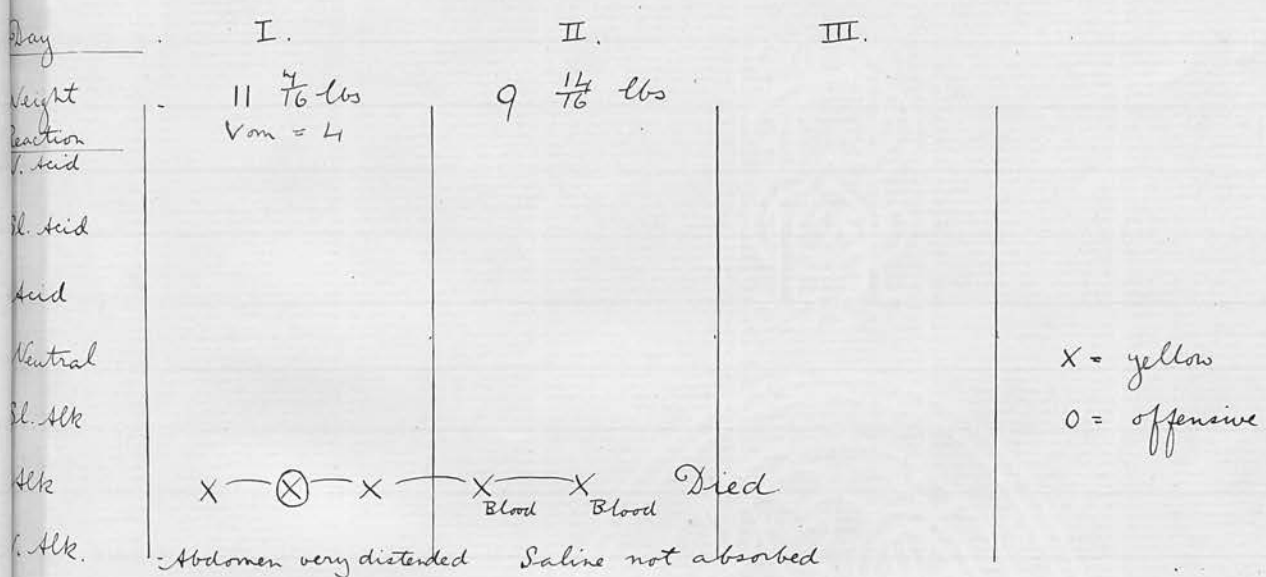
Such Charts were made of all our In-patient cases and were most interesting to study when taken together with the general condition as regards improvement or otherwise of each patient.

Case I



X = yellow
 ● = green
 ⊗ = offensive
 AN = approaching normal
 N = normal

Case XVII = Case I re-admitted with second attack

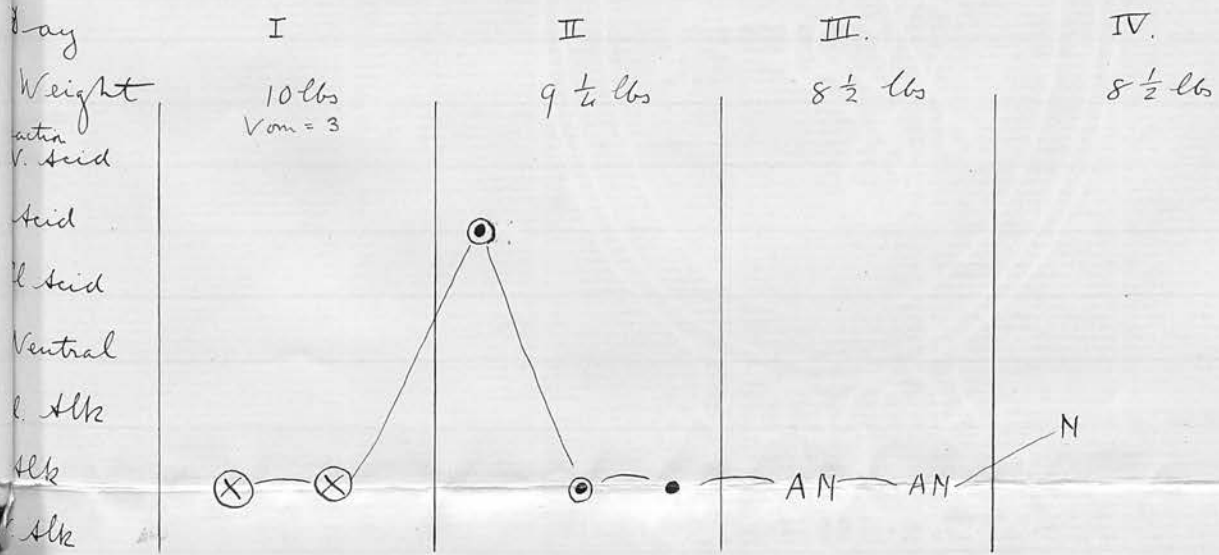


X = yellow
 ⊗ = offensive

Abdomen very distended Saline not absorbed
 Motions very small. Mag Sulph Enema - no result

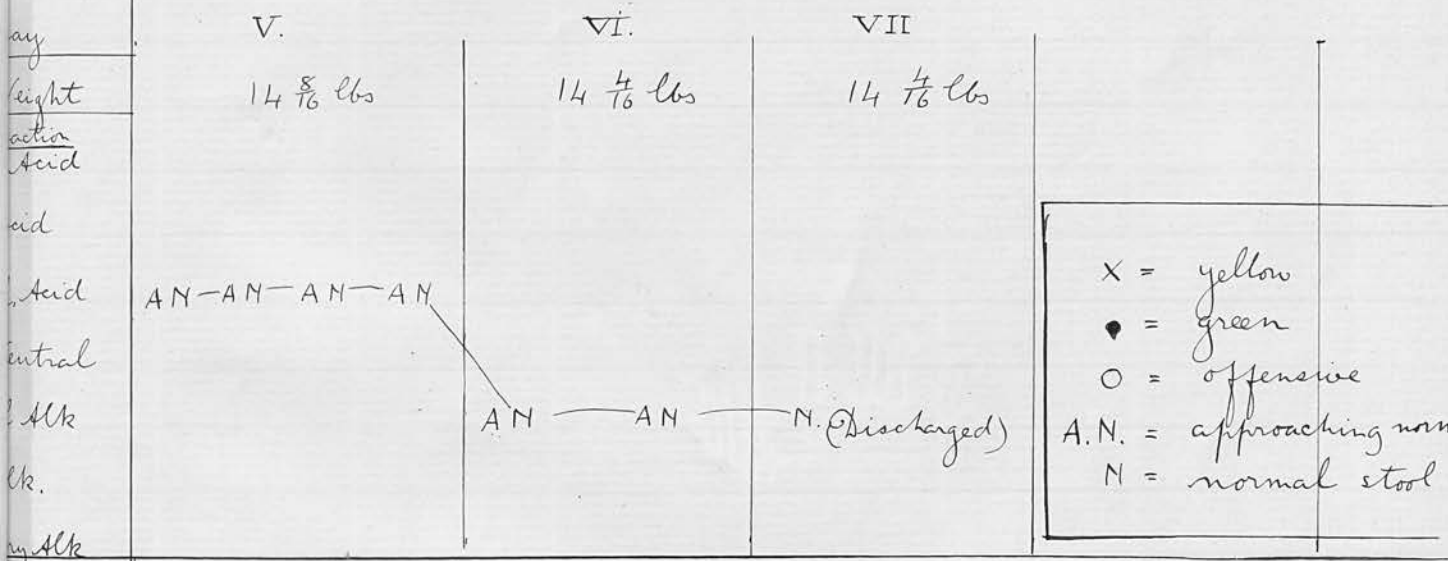
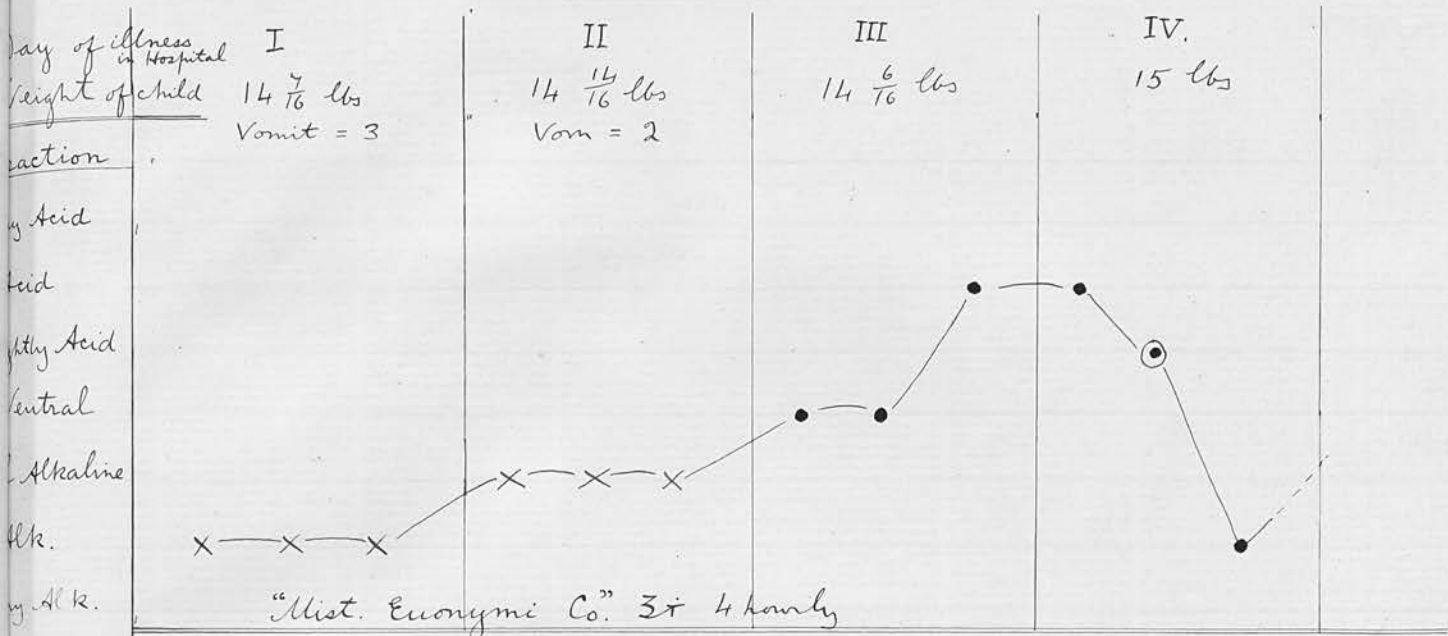
An example of the virulence of a second attack. Extremely rapid loss of weight

Case VII.

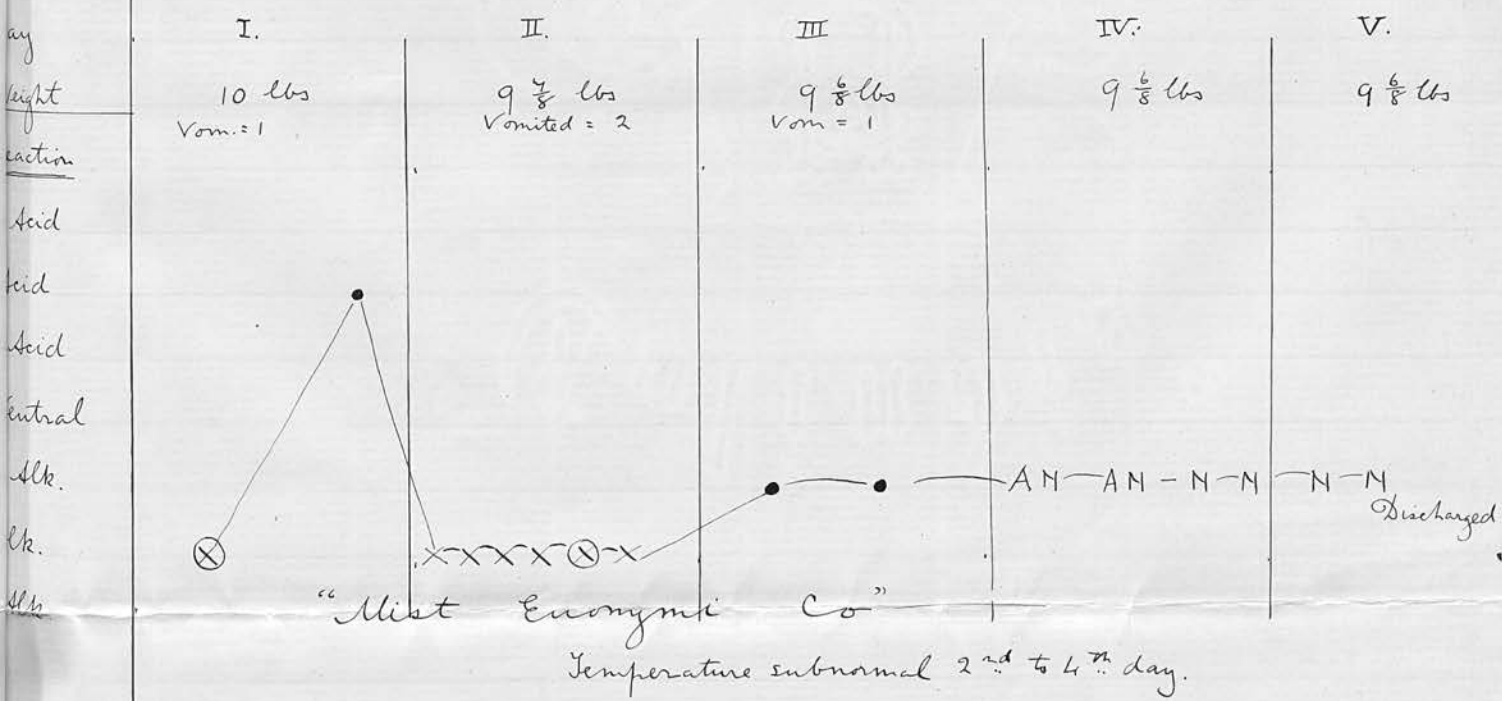


Breathing rapid & shallow

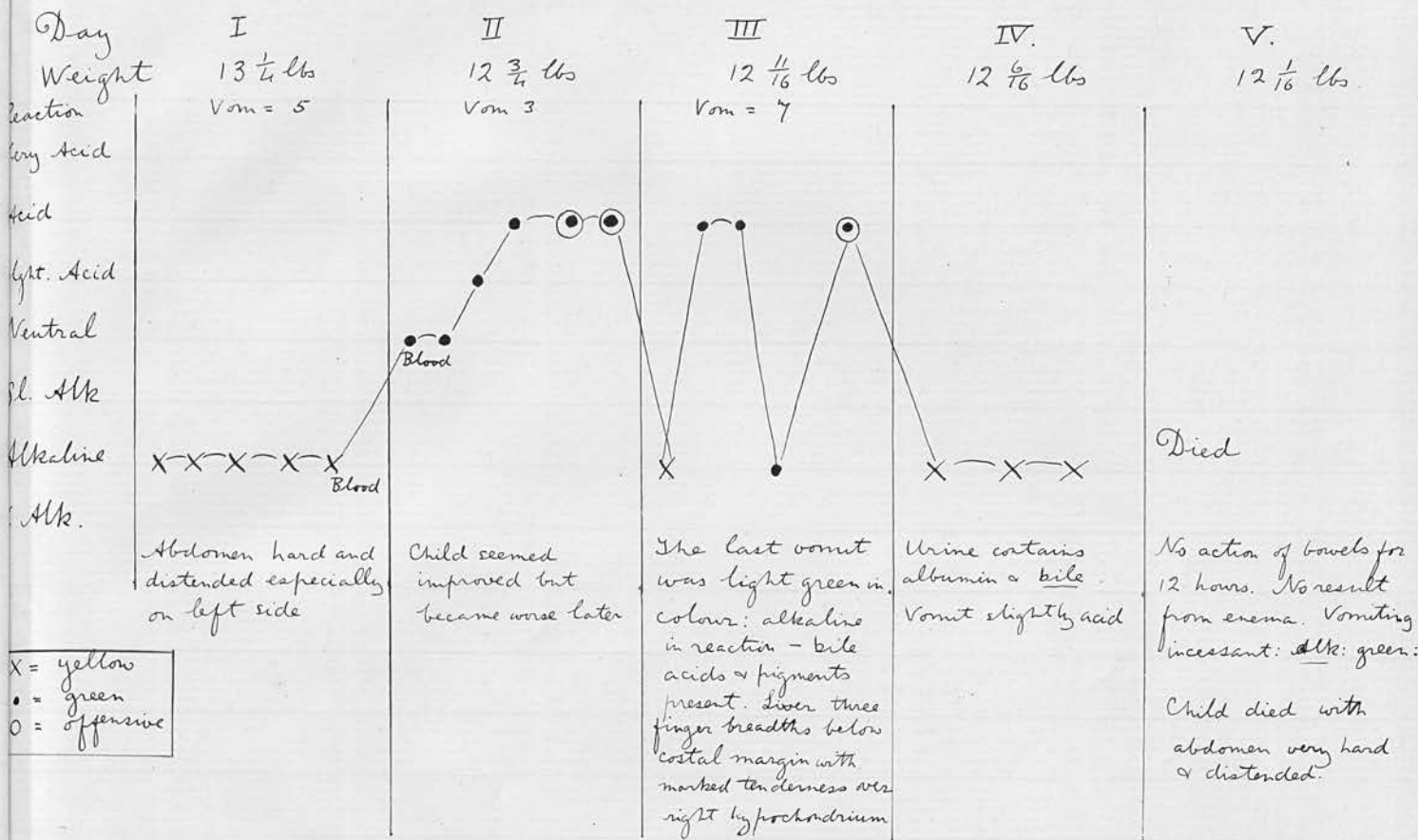
Case XI.



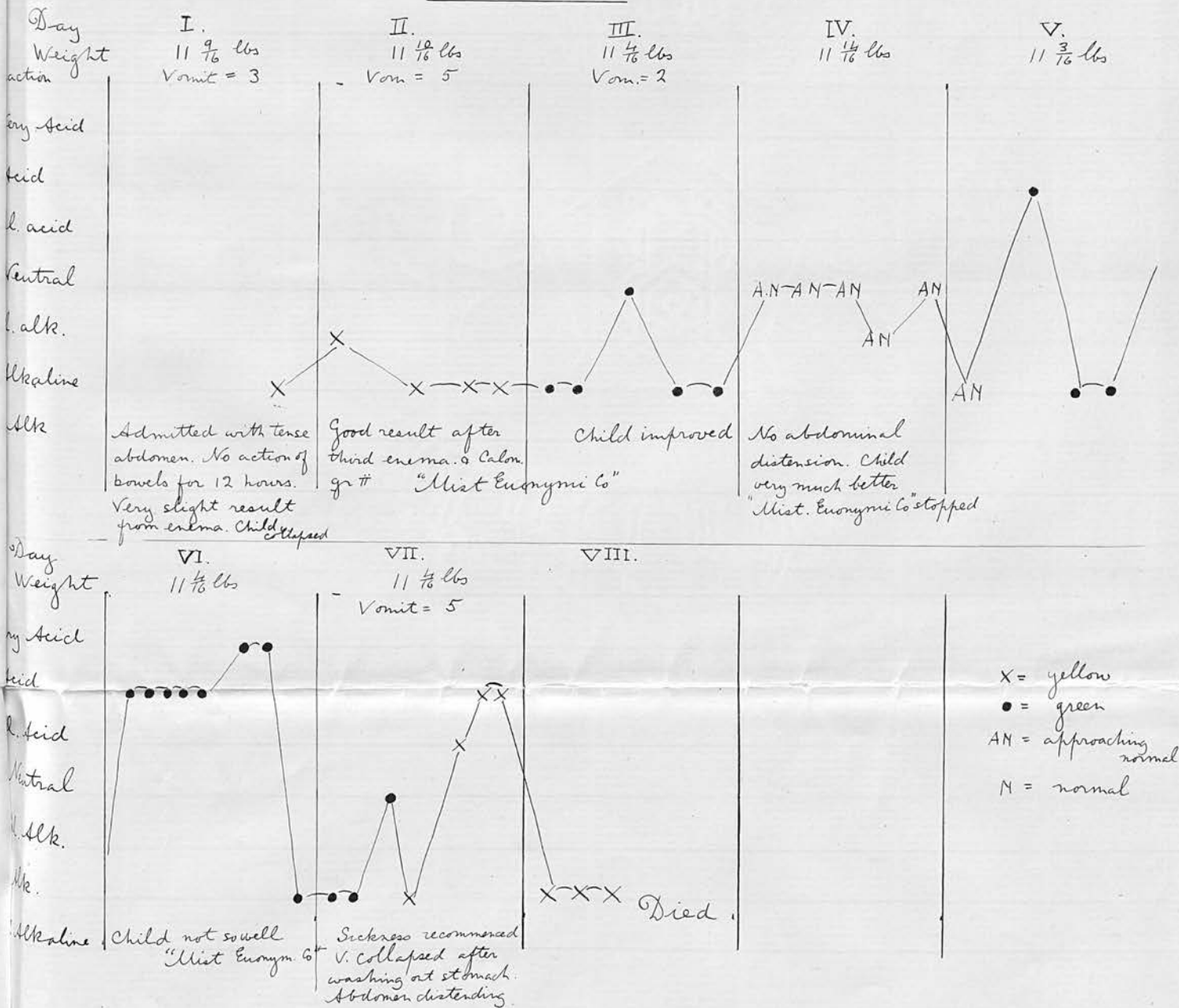
Case VI.



Case VIII.



Case XIX. shows a relapse



DAILY RECORD. (See Page 14)

By Nurse Liddle Williams & Blair

Name of Infant Bertie Johnson

Weight.

Date 30-8-06

rd 3 Day of Illness.

Feeding Begun.	Feeding Ended.	Amount Taken.	Rectal Irrigation.	REMARKS.
7.40. 10.20 12.20.	7.45. 10.30. 12.40.	3 ii a.w. 3 ii a.w. 3 ii a.w.		<p>Child grew steadily worse from 3 pm. and died 3.40 pm.</p> <p style="text-align: right;"><u>Thurber</u></p> <p>P. 2. v. def. Bronch. Pneumonia. Gut lines.</p> <p>Ulcer of Asc. Desc. Colon.</p> <p>Cyst. Ovar. impur. & full bladder v. distended.</p>

Stools.	CHARACTER.	Vomit.	CHARACTER.
12. 1.	<p>yellow loose. slight.</p> <p>yellow v. watery. pale.</p>		

Oxygen given twice between 9.30 & 10 a.m.

Calomel gr. i at 12.30.

Tepid sponged at 12.20 p.m. Temp. came down from 102² to 98²

Hypo brandy $\overline{m\bar{x}}$ at 1.15.

To have Tepid. Enema 3.0.5.

3 p.m. Child much worse hypo brandy $\overline{m\bar{x}}$ & Starch: $\overline{m\bar{ii}}$.

Other Remarks.

We found that the stools varied rapidly in all the particulars which were being noted, namely, the colour, consistence, reaction, size, smell and frequency, in a most perplexing manner but we soon observed that the variations had a direct and striking relation to whether or not there was an improvement in the condition of the patient.

Thus we found that nearly every one of the worst cases began the attack with stools which were yellow, frequent, large, fluid and alkaline, but not odourless, though they soon became odourless as the disease advanced. I would here again draw attention to the fact that these yellow watery non-offensive stools very rapidly become offensive on being allowed to stand and may at the same time change in colour from yellow to green. These yellow stools are free from bile pigments, bile salts or bile stained mucus and, if drops of them be placed on a slide and dried, crystals of cholesterin may occasionally be demonstrated microscopically - an indication of a great increase in mucus excretion.

The stools may remain yellow, watery and alkaline in character and in such an event the child remains acutely ill and keeps losing weight steadily even to the extent of 1 lb to $1\frac{1}{2}$ lbs in the 24 hours. Such stools in my opinion never persist

longer than 36 hours and seldom more than 24 hours. Either the child improves or gets worse. If the child is getting worse the stools get either gradually smaller in amount and are replaced by motions which consist of blood stained mucus or even pure blood, or else the diarrhoea suddenly stops, the abdomen becomes tense and tympanitic, the secondary vomiting commences and the child dies. On the other hand if the child is going to improve there is a sudden change from the yellow watery alkaline stools to one which is green, pasty and acid in reaction. This green stool is often frothy and seething in character indicative of active fermentation; it quickly becomes offensive on standing; it is free from bile acids and often gives a yellow reaction to the sublimate test of Schmidt described later. After one or two of these stools have been passed the child may have a relapse and the yellow watery alkaline stools again make their appearance or the child may continue to improve in which case the green acid stools soon pass to green alkaline, become more consistent, less offensive, and gradually assume all the characters and appearances of normal faeces.

In the event of a relapse now, or, at this stage it should be considered rather as a re-

infection, the reverse order of types of stool is generally gone through, namely, a change from normal to green alkaline, thence to green acid, thence to yellow alkaline offensive and finally to yellow watery, very alkaline, non-offensive.

In some of our cases this sequence was not followed; in some cases there was a jump from yellow alkaline stools to green alkaline and vice versa; in other cases yellow acid stools made their appearance. As I shall show later, under treatment, such divergencies from what I consider a sequence which regularly occurs in untreated cases could be attributed to the action of certain drugs which were being administered. It is difficult to offer an explanation as to why the stools change colour and reaction so rapidly and seemingly so indiscriminately but I am inclined to the view that the changes are due to abnormal processes occurring in the intestinal tract to its contents,

Harley & Goodbody in their chemical investigations of Intestinal Diseases 1906 state that the normal contents of the small intestine in its upper third are of a yellowish colour, of the middle third greyish and of the lower third green, while the contents of the large intestine are yellow in colour.

Such being the case then the variation in colour of the stools is due either to abnormal processes occurring to the intestinal contents, or to rapid elimination of normal contents, or to both.

But except early in the attack elimination of the contents very often is not rapid. Cases are got where the intestinal peristalsis is getting obviously weaker and cases are got where post mortem the intestines are found full, yet in such cases just the same variation in colour and reaction of the stools is seen.

If the variations were due to normal contents being rapidly eliminated, then a sequence of colours of stools corresponding to the sequence of colours of the intestinal contents would be got. Such an invariable sequence I did not observe.

During the latter half of the season I came to the conclusion for reasons I shall state later that it was of the utmost importance to ascertain whether the faeces contained altered bile or not. To ascertain this there is a test described by Prof: Schmidt in his book on Coprology, 1906.

The test is as follows: A teaspoonful of the faecal material is placed in a wide test tube and well stirred with a glass rod and a little sterile water added. The test tube is then filled 4 to 6 times the volume it contains with a saturated solution of corrosive sublimate, well stirred again

and allowed to stand 12-48 hours. By that time normal faeces have become coloured bright red from the presence of hydro-bilirubin while if unchanged bile pigments are present the colour becomes green. This test was tried on every case admitted after No. 18 and not once was a red reaction obtained nor was it obtained till the cured cases had been home some weeks.

Case No. 5 was the only exception. Here a red reaction was obtained the day before the case was sent home but that was 29 days from the onset.

I agree with Prof: Schmidt that the presence of unchanged bile pigments is due rather to a defect in the normal processes of reduction than to a too rapid elimination of intestinal contents.

Schmidt has shewn that if the corrosive sublimate solution turns the stool green that bil-verdin is present. In many of our cases the already yellow stools were not changed in colour or even sometimes green stools were changed to yellow confirming the absence of bile.

Thus by Schmidt's test was shewn:

(1) The invariable absence of hydrobilirubin when, considering the putrefacting processes, one would have expected an increase.

(2) The occasional absence of biliverdin.

As I shall mention later I had come to the conclusion from various observations that in Zymotic Enteritis and Cholera Infantum there is a disturbance of the bile secreting functions bringing about a diminution or even a total absence of bile in the intestines and Schmidt's test gave strong corroborative evidence in favour of my conclusions.

V. Rapid loss of weight.

Holt says in his "Diseases of Children" Page 377 the "loss of weight is more rapid than in any other pathological condition in childhood". Of this there can be no doubt but I venture to think that a great loss of weight does not occur in every case. In some of our cases the loss was very slight and in one fatal case there was even a slight gain in weight but in considering our weights as indicating the severity of the disease in each individual case it must be taken into account that from 4 ozs to 6 ozs or even more of saline were injected subcutaneously every day.

It may be taken that in untreated cases with the typical yellow watery alkaline stool there is much loss of weight but that invariably when the stools became green and alkaline the weight at once begins to rise.

VI. VI. Depression of the anterior fontanelle.

This is invariably present and may be taken as one of the distinguishing signs from meningitis when the cerebral signs are very prominent. I have never noticed so far the over lapping of the cranial bones mentioned by Holt.

VII. VII. General prostration and nervous irritation.

Considering the age of the patient and the loss of large quantities of fluid from the bowel it is to be expected that there must always be great general prostration almost from the outset. The resulting aspect of the patient presents one of the most vivid clinical pictures it is possible to have and is well described by Holt (ibid page 377). "The face, better, perhaps, than any single symptom, indicates what a profound impression has been made upon the system. The eyes are sunken, the features sharpened, the angles of the mouth drawn down, and a peculiar pallor with an expression of anxiety overspreads the whole countenance." At first the nervous signs are those of irritation, the child becomes very restless; it throws its head first on one side then on the other the hands are incessantly moving; if not restrained and if old enough it will raise up its head, try to stand up holding on to the sides of the cot, bang its head on the iron-work, then fall back on the bed with a moan and roll its head from side to side.

The child may moan frequently or make little plaintive cries but rarely if ever cries out loudly as a child suffering from meningitis does.

It may also be said that the knee jerks are usually exaggerated and there is no retraction of the head usually though I have noticed it in the very last stages in rare instances when there may be an accompanying convergent strabismus.

VIII.

Temperature elevated.

I cannot agree with Holt (page 377) that the temperature is invariably elevated. An elevated temperature is usual at the onset and one would give a very bad prognosis where it rose at once above 103° , but many of our cases where there was severe diarrhoea had subnormal temperatures. Where there was a persistent temperature we noticed very frequently that the disease was complicated by broncho pneumonia. I mention this complication later. It might quite well account for the increase of temperature in many cases.

In many cases where there was an elevated temperature the pyrexia was not suspected till after the thermometer had been employed for the skin remained clammy and the extremities cold.

IX. IX. Rapid pulse.

The pulse rate is invariably quickened and later becomes intermittent and weak. Comparing it with the pulse of other acute diseases it is wonderful how comparatively strong it remains in uncomplicated cases almost to the end considering the severity of the impression made by the disease.

X. X. Respiration frequent.

It has been frequently noted by observers that the rate of respiration is accelerated. Thus Holt says in his Diseases of Children page 377, "The respiration is irregular and frequent and may be stertorous."

No one I think has laid stress on the fact that broncho-pneumonia is a frequent and grave complication of Zymotic Enteritis.

My attention was first called to it by noting that it was a frequent sequela. The child got almost well of the enteritis when broncho-pneumonia made itself apparent and frequently brought about a fatal issue in the already much exhausted child.

Careful examination showed that broncho-pneumonia is a very frequent complication.

The pulmonary condition may be easily missed for there may be absolutely no cough throughout, yet in all the post mortem examinations made on the

worst cases there were definite broncho-pneumonic patches in both lungs, chiefly in the lower lobes. In those cases the breathing had been rapid, shallow and in some cases irregular and slightly stentorous.

The physical signs on auscultation were almost entirely negative. Occasionally rales were heard but the auscultatory signs were misleading as the breathing in the exhausted child is so very shallow and faint.

Dr Pearson went so far as to think that the rapidity of breathing and the heightened temperature is always in the later stages entirely due to a pulmonary lesion.

I cannot go so far as that but I know that in many cases we suspected the presence of a pneumonia and post mortem examination confirmed the diagnosis.

I frequently observed that as the diarrhoea and vomiting improved the pulmonary conditions became more evident and all the usual signs of a broncho-pneumonia showed themselves plainly.

The existence of this complication in severe cases is rather what one would expect for the child is in a very lowered condition, it is liable to inspire virulent material after vomiting and in many cases the Phthisical Family History might be taken into account.

XI. XI. Tongue red and dry.

The tongue, at first coated soon becomes red and dry. It has the appearance of being enlarged and is often slightly protruded. The entire appearance of the tongue and lips and their parched movements all point to an excessive loss of fluid and indicate that there must be great thirst.

XII. Abdomen soft and sunken.

The abdomen is invariably soft and retracted in the early stages of the disease and as long as the diarrhoea acutely persists.

Sudden distension of the abdomen is of very grave significance. It is generally accompanied by diminution and even entire stoppage of the diarrhoea or, more frequently by small bloody stools and occasionally by the passage of pure blood from the bowel. With the distension vomiting recommences and the child presents a picture of toxæmia absorption.

XIII. Almost Insatiable Thirst.

As might be expected from the great loss of fluid from the bowel extreme thirst is manifested. This is especially the case in the early stages when the child will drink greedily anything in the shape of fluid offered to it, even although the stomach immediately returns it.

As the child gets more drowsy thirst is the only cause of its rousing and fluid is the only thing it shows any desire for.

In the later stages still I noticed that the children often refused even fluids and that they invariably did so if secondary vomiting had commenced. But in the later stages subcutaneous infusions of normal saline 6-8-10 oz. were being given once or twice in the 24 hours and these may have had the effect of overcoming the thirst.

XIV.

Urine small in quantity.

Throughout the acute stages of the disease very little urine is passed or even none at all for 24 hours at a time. When passed the amount is very small - from 3 fs to 3 π it is highly coloured, generally there is albumen present; sometimes bile pigments and very often urates.

There were some other physical signs which I observed and which I am not aware have been mentioned in any text book. They at least have not been laid stress on.

They were:

I.

Tenderness on palpation over the right hypochondriac region.

In many of the cases I was struck by the fact that the lower margin of the liver was lower than normal; being in some cases from $2\frac{1}{2}$ to 3 finger

breadths below the costal margin. This was not a mere downward displacement of the liver resulting from emaciation but was a definite enlargement and this enlargement was confirmed in the fatal cases post mortem.

On palpating over the region of the liver distinct tenderness was found present. The child would lie quiet till this region was reached when it would cry or moan, retract from the palpating hand and flex its thighs on the abdomen.

This observation was confirmed by Dr Pearson. Later in the year it was brought to our notice that Dr. A. McC. Weir of Liverpool had written to the "Lancet" of June 16th, 1906 a letter in which he said, "The liver in all these cases is grievously affected and the absence of bile causes the foetid putrid stools." Dr Pearson wrote to Dr. Weir asking him from what outside source or on what original work he made this statement and he has kindly let me see Dr. Weir's reply. Dr. Weir after describing carefully the type of enteritis with "green curdy offensive stools" states that "the liver is reduced to almost half its normal size and its secretion is at a standstill."

In no case which I examined amongst either our In-patients or Out-patients or in any post mortem examination, did I find any reduction in

size of the liver. In many cases as I have already stated there was actual enlargement

II. An appearance of slight jaundice.

In several cases and especially in those cases in which there was tenderness over the region of the liver, I noticed that there was a distinctly yellow tinging of the skin.

It could not be called a true jaundice for in no case did I observe a yellowness of the sclerotics.

I also noted with interest that in such cases the urine was especially high coloured, that the faeces were generally very light coloured and that, as will be shown later, such cases showed distinct improvement when treated by a cholagogue.

Complications:

I. BRONCHO-PNEUMONIA: I have already mentioned and discussed this complication and would only here again draw attention to its frequent occurrence and how it greatly aggravates the case.

II. CORNEAL ULCERATION:- In the later stages of a severe case the eyes remain half open: they are seldom completely closed and there is often no attempt at blinking of the eye-lids. The result is that the eyes become first coated with a mucus film and later there is, in a few cases, actual

corneal ulceration. Such cases never recovered. In five of our thirteen fatal In-patient cases this complication was observed.

III STOMATITIS: This is a most frequent complication or perhaps it should rather be considered a sequela, for it occurs generally when the diarrhoea symptoms are not quite so acute.

It was a common observation on the part of the mother that "the diarrhoea is a little better but his mouth is full of ulcers". The stomatitis occurring in those cases was found peculiarly intractable to all the usual methods of treatment.

IV. SCLEREMA: This condition occurred in cases which invariably proved fatal. It was noticed in five of the fatal cases. Much more frequently the skin merely gets dry and inelastic.

OBSERVATIONS FROM POST MORTEM EXAMINATIONS.

In 9 of the 13 fatal cases amongst the In-Patients a post mortem examination was permitted.

Owing to the great mass of other work I had to attend to I was unfortunately unable to make the searching and exact post mortem examinations I should have wished to have made and I much desire a future opportunity for exhaustive work in this connection.

Most of the pathological changes were found as described by Holt in his Diseases of Children. The condition of the intestinal tract was found to follow closely the appearances described by him on pages 360 and 361.

The lesion in the kidneys was as described on page 362 and in which he says that some degree of acute degeneration is found in every case and that in a few there is acute diffuse nephritis. In every case, I found acute congestion. As he says on page 378 there is nothing apparent in the brain to account for the acute nervous symptoms often present.

A slight hypernemia in a few instances was all that I found.

In every case the strictest search was made for tuberculosis especially in the lungs, intestines and meninges. No trace of such was found in any case.

Examination of the brain and meninges excluded all possibility of the nervous symptoms being due to a meningitis.

There were some points of which I took particular notice. Some of them are mentioned in the text books, some of them have as far as I know never previously been taken note of. They were:

I. DISTENSION OF THE GALL BLADDER:

II. PARTIAL OR COMPLETE OCCLUSION OF THE CYSTIC AND COMMON BILE DUCTS:

In four cases the cystic duct admitted the passage of nothing, not even a stream of coloured water from a fine pipette. The finest lachrymal probe could be entered the common duct from the duodenum but its passage was soon brought to a stop.

In five cases it was only with difficulty that the finest lachrymal probe could be passed along the cystic duct.

The hepatic duct was invariably patent.

In many cases in which the patency of the ducts was tested in infants who had died from other

causes, we found there was no difficulty in passing a medium lachrymal probe along all the ducts.

We were unfortunately for want of time unable to make sections of the occluded parts for microscopical examination.

III. In all the cases examined there were present small patches of Broncho-pneumonia and these occurred chiefly in the lower lobes of the lungs.

IV. Holt in his description of the intestinal tract (ibid, page 36k) says:

"The greater part of the small intestine and sometimes the entire colon, are distended with gas, and contain material of a greyish white colour about the consistency of a thin gruel. It has a mawkish odour, but usually not a very offensive one. The mucous membrane of almost the entire intestinal tract has in most cases a pale, "washed out" appearance. Sometimes this is only seen in the small intestine, while there are areas of congestion in the colon.

"In some cases, where the symptoms have been those of choleric diarrhoea, there are found evidences of an intense diffuse gastro enteritis, as shown by congestion of the stomach and almost the entire intestinal tract, with swelling of the mucous membrane and especially of Peyer's patches."

In all of our cases injection occurred throughout the intestinal mucous membrane. There was swelling of the mucous membrane and prominence of Peyer's patches. In one case there were present pin point recent ulcers extending from the colon almost up to the duodenum. The intestines were invariably distended with gas.

V. As might be expected from the toxæmic nature of the illness decomposition is of an extremely rapid description so much so that it was our rule, in order to avoid misleading appearances, to make our examinations within four hours after death.

I would now submit the following conclusions I have come to regarding the aetiology, pathology and treatment of this disease. In doing so I rely mainly on my own observations but am entirely indebted to Dr. New (late of the Lister Institute) for his investigations on the bacteriological factors of the disease and his suggestions as to the aetiological importance of these factors.

(I) This disease, Zymotic Enteritis, with its severer form, cholera infantum, occurs in its greatest epidemics and produces its highest mortality in the slums of towns, and when the mean temperature of the air has been over 60°F for some days. It occurs chiefly in infants under two years of age and, for the most part,

in infants who have been previously unhealthy. As I have already shown this unhealthiness may be due to previous illness, improper feeding or to a bad family history.

In connection with improper feeding I have shown that this may have gone on for some time and thus have produced a weakened condition of the alimentary mucous membrane but here I would add that the feeding may have been proper up to the day preceding the attack when some quite improper article of diet may have been given and as a result a state of acute indigestion set up and subsequently an attack of Zymotic Enteritis. Thus I had cases coming up for treatment which were stated by the mother to be entirely breast fed but on further enquiry the fact was elicited that the child had in one case been given a tomatoe and in another case some fried fish the day previously.

(II) II. The disease is caused by a micro organism acting in an alimentary canal whose resistance has been weakened. I consider the micro-organism to be the *B. Proteus Vulgaris*.

Booker, in the John Hopkins Report for 1897, Vol. VI, describes the *B. Proteus* as being invariably present in the stools of the cholera infantum type of case. This bacillus has been taken little notice

of of late but it has never been entirely discounted as a factor. Dr Sidney Martin has written many papers on its toxins and has shown they produce in animals symptoms similar to Zymotic Enteritis.

As little attention has of late been paid to this bacillus in connection with Zymotic Enteritis Dr New's observations were of great interest.

Dr New was asked to look for the micro-organism described by De Morgan as occurring so frequently in cases suffering from Zymotic Enteritis and to report the type of stool in which it was generally found. He was unable to find this micro-organism in any specimen sent but on examining the yellow alkaline stools he invariably obtained an almost pure culture of the *B. Proteus Vulgaris*.

In two cases he obtained a definite agglutinating reaction with the *B. Protens* in dilution of 1 in 200.

In several instances he isolated at the same time as the *B. Proteus* the *B. Pyocyaneus*.

I was unable at Plaistow to personally confirm these observations and as Dr New is going to publish an account of his investigations I cannot give fuller details here.

(III)

The severity of the attack in each individual case varies according to the power of resistance of

the individual but also according to whether the individual has been poisoned by active bacilli forming toxins or by the ingestion of the toxins without the bacilli.

Thus Zymotic Enteritis is produced by food which has been previously sterilised but which contains specific toxins. Helping to confirm this theory it was found that the stools of cases suffering from ordinary Zymotic Enteritis did not contain the B. Proteus.

Cholera Infantum on the other hand is produced by food which contains active bacilli and their toxins. It was found that non-sterilised food which had been exposed to contamination by bacilli could produce this type of the disease but the very worst cases resulted from being given food which had first been sterilised and then been left exposed to contamination by bacilli.

The reason for this is that food which has been sterilised more or less by boiling has no bactericidal action and if left exposed to contamination by bacilli it may soon become much worse than at the start. Again food which has been boiled is of a low nutritive value and this has an important bearing where feeding on boiled milk has been resorted to for some length of time.

This supposition, if correct would help to explain:

(1) The comparative rarity of the disease ~~am~~ amongst the upper classes. Here the milk is more carefully handled from the very first. It is more carefully stored, more frequently delivered, is fresher on being used and is more carefully prepared immediately before use.

If sterilised it is not boiled and is kept carefully free from contamination between sterilisation and use.

(2) The rare occurrence of the disease in babies entirely breast fed.

(3) The comparative rarity of the disease in infants fed on condensed milk where each feed is freshly prepared and where great care is exercised in such preparation. From enquiry I learnt that it is a common practice amongst poor parents, when condensed milk is used, to prepare a stock sufficient to last 24 hours and to leave this standing exposed in the living room.

(4) The frequency with which the disease occurs in infants fed on cow's or condensed milk which has been boiled and then allowed to stand unprotected for 12 or 24 hours.

Dr Myer Coplans in the "Lancet" of October 11th 1907 shows that bacilli grow best in diluted condensed milk and next best in previously boiled milk. He has not however, I believe, investigated the growth of B. Proteus in this connection.

(IV) IV. The household fly is closely associated with the disease. This is supported by the following facts and observations.

(1) The disease occurs with much greater frequency in those summers or parts of those summers in which there is the greatest plague of flies. In 1904 and 1906 flies were especially abundant and in those years Zymotic Enteritis was rampant. In 1907 there was a scarcity of flies till the end of August and it was not till then that really bad cases of Enteritis were observed.

(2) Dr Nash in the "Practitioner" of May 1906 and Dr Newsholme have shown that the greatest number of cases and the greatest mortality occur:

- (a) In towns and districts where there is much made ground.
- (b) In towns where the closet and drainage systems are most accessible to flies.

(c) In towns where the work carried on acts as an attraction to flies. This has been demonstrated by Dr. Newstead amongst others in his Report to the Corporation of Liverpool, Nov: 1907.

(3) Dr Bridge in the "Practitioner" of October 1907 states that isolated cases occur in the country where the surroundings are so far healthy but where a plague of flies has been attracted by a manure heap at no great distance.

(4) I have heard that it is the custom in some parts of Africa and Australia in order to protect food from the swarms of flies that abound, to sling it up in the highest trees. I have seen larders so hoisted up in trees in this country.

In the summer of 1907 I went down to Plaistow on purpose to investigate the disease further. Owing to its being a wet cold summer I only saw two cases of Cholera Infantum during my stay, although I was there during the last week in July and the first three in August, the very time when experience told the very worst cases might be expected in large numbers.

I was able however to inquire into some cases of bad diarrhoea in regard to what floor of the dwelling house they lived on.

From such cases and from cases of Zymotic Enteritis which occurred after I left, the particulars of which were kindly kept for me, I am able to give the following interesting statistics.

Of 94 cases investigated

68 cases or 73.3% occurred on the Ground floor.

20 " or 21.3% occurred on the First floor

6 " or 6.4% occurred on the second floor.

No case was found which lived on the third floor, but I believe patients of this hospital rarely live in houses which reach to a third floor. As there are very few houses in Plaistow having basements or underground floors cases occurring on such floors were not taken into account.

It would be most interesting and important to collect such figures from large numbers of cases in a year when Zymotic Enteritis was very prevalent and in towns where high tenements and underground floors are the general rule.

(5) At the end of 1906 Dr Pearson prepared a map of the West Ham District locating by spots the dwellings of 600 of the Out-patient cases.

On visiting the streets showing the abodes of the greatest numbers of cases he was struck by the almost invariable close proximity of either stables or large rubbish or manure heaps.

(V) V. The summers having the greatest number of cases and the highest mortality are those in which the most favourable conditions for putrefaction, bacterial growth, and fly multiplication exist. This has been shown by:

- (1) Dr Newstead's Report on the habits of the fly.
- (2) Drs Nash and Newsholme in the "Practitioner" already quoted.
- (3) The fact that when flies are not plentiful till late summer cases of Zymotic Enteritis are scarce till late summer. Also when the summer is wet and cold throughout and flies few in number Zymotic Enteritis is scarce.
- (4) The fact that when the temperature of the earth 4 feet below the surface is less than 56° conditions inimicable to Zymotic Enteritis exist as proved by Ballard. Such conditions are also inimicable to putrefactive bacterial growth and fly multiplication.

(VI) •VI. The fly carries the B. Proteus a micro-organism of putrefaction to the food given to the infants and even directly to the infant in some cases. This is supported by the following:

Celli⁺ and Volpino^{*} have demonstrated the presence of the B. Proteus on the legs and feet of flies and in their faeces.

No one has yet demonstrated the presence of the B. Proteus in milk which has been left exposed in the dwelling rooms of patients suffering from or likely to suffer from Zymotic Enteritis. This is needed to entirely prove the truth of my supposition and should be undertaken at an early date.

It has been shown by several investigators that flies are undoubtedly the carriers of infection in several diseases. Amongst others this has been shown by Dr Buchanan in the "Lancet" of July 27th, 1907 while Drs. Hoffkine and Macrae in the "Lancet" of Nov: 30th 1907 have shown that the spirillum of cholera may be carried to milk by flies.

Again Dr Newman in 1907 showed pretty conclusively that Zymotic Enteritis is a purely domestic infection.

⁺ Celli, Anuali D'Igiene Sperimentale, Vol. XVI.
Nuova Serie, 1906, Page 315.

^{*} Volpino, Archivi per le Scienze, Mediche XXIX.

It is true that we did not have separate lots of nurses, one lot to attend to the feeding, another to change the diapers and otherwise attend to the infants, but beyond this every possible precaution was taken to prevent re-infection. Each child had its own entirely separate outfit as regards rectal washing out apparatus, thermometer etc., and every care was taken by the nurses as regards disinfecting their hands after attending on one patient and before attending on another. Yet in spite of these precautions we had cases which were on the road to recovery and then were undoubtedly re-infected.

I am therefore of the opinion that flies can carry infection directly to the child. Try as we might we could not entirely exclude flies from the wards. In the diarrhoea wards there seemed to be a special attraction for them. They hung lazily and pertinaciously about the children and were with difficulty driven away only at once to return. They behaved as flies may be observed to do when feeding on attractive putrefactive material. Before steps were taken by gauze nets to prevent it they were observed to crawl lazily over the faces and lips of the children who were too ill to make any effort to get rid of them. Thus from one patient to another they would go and, undoubtedly, in my opinion carrying infection in their train.

(VII)

The disease is highly infectious.

While it has for long been known, that, once introduced into a ward containing infants of a suitable age Zymotic Enteritis may rapidly spread till eventually the whole ward may become infected, it is only quite recently that special wards have been provided in some hospitals solely for the treatment of this disease.

I have just shown that the disease in my opinion can be carried by flies directly from one patient to another but there is little doubt it can be conveyed by the hands of doctors and nurses, by various implements such as feeding bottles used without proper care, by soiled diapers not being carefully removed and at once destroyed or disinfected and even possibly by the dust of stools which have been allowed to dry.

It is noteworthy that adults are sometimes liable to mild attacks of the disease. The three special nurses in charge of the diarrhoea wards were each, during the first three weeks, incapacitated for work for a day or two by reason of an attack of mild diarrhoea which could not be attributed as far as could be discovered to improper feeding.

The attack, in each case, began with a sore throat: there was a temperature of from 100° to 101°

there was no vomiting, but the feeling of depression and ill health was out of proportion to an ordinary diarrhoea.

I have heard of other instances of similar attacks in adults who had been in attendance on cases of Zymotic Enteritis.

(VIII)

It is probable that the infection is a descending one.

Apart from the Stomatitis which occurs late in the disease and which attacks the child merely from its much lowered condition there is at the very commencement of the attack some abnormal redness of the mucous membrane of the mouth and fauces. In the few cases in which the child is sufficiently old the mother tells one that the child has indicated that its throat is sore. The fact in the majority of bad cases the vomiting commences before the diarrhoea is another indication that, though the disease subsequently quickly spreads through the whole intestinal tract, it attacks first the upper parts of the tract.

From clinical observations but chiefly from Post Mortem examinations I have found the following pathological conditions to exist in Cholera Infantum in connection with the alimentary system and I think it probable that these conditions arise more or less in

the order in which I name them, and that thus a descending infection is indicated.

(1) Acute Gastritis. That such exists is seen on post mortem examination. That it occurs early in the attack is pointed to by the fact of the vomiting in the majority of cases preceding the diarrhoea.

(2) Cholangitis with more or less occlusion of the cystic and sometimes of the common bile ducts with a resulting distension of the gall-bladder.

This is supported by:

- (a) The post mortem observations (vide. p.34
- (b) The fact that no bile or bile pigments appear in the typical stools of Cholera Infantum (vide pp.22 and 23.
- (c) The appearance of unchanged bile in the stools of patients convalescent from the disease.
- (d) The fact that cases improve under the influence of cholagogues (vide p.57
- (e) The fact that cases improve whenever the flow of bile is re-established as shewn by the appearances of the stools.

(3) Probably in some cases a hepatitis and in others a pancreatitis exist.

No microscopic examination was made by us either of the liver or the pancreas, but the pancreas in some of the cases was distinctly brittle and Holt (ibid., page 362) mentions that degenerative changes in the cells of the liver may occur.

- (4) Enteritis. Observed Post Mortem.
- (5) Entero-colitis " " "
- (6) Colitis with ulcer formation and a resulting toxæmia and a partial or complete paresis of the gut.

The Colitis and ulceration of the gut is confirmed by post mortem examination.

The partial or complete paresis of the gut is evidenced by:

- (a) Diarrhoea diminished or entirely stopped.
- (b) Abdominal distension.
- (c) Colon and sigmoid not empty
- (d) Vomiting recommenced.
- (e) Condition improved if bowels can be made to act again.

(IX) The variations in the types of the stools are probably the result of abnormal processes occurring in the intestines, such abnormal processes being produced by the action and interaction of various bacilli.

Although many micro-organisms may be found in the intestinal tract of children suffering from this disease I believe as I have already stated that the

B. *Proteus Vulgaris* is the bacillus directly associated with the disease.

In the stools of Cholera Infantum, besides the B. *Proteus Vulgaris*, we almost invariably find Klein's gas forming bacillus, the B. *Coli Communis* and sometimes the B. *Pyocyaneus* and in some outbreaks in some localities we find the bacilli described by De Morgan, Shiga, Flexner and others.

It might be expected if the dominant bacillus is the B. *Proteus* that the stools would be very offensive throughout the attack, and although the yellow watery stools of Cholera Infantum are very offensive at first they soon become non-offensive, yet, if those non-offensive stools are allowed to stand they rapidly become most offensive. Is it not probable therefore that one or more of the other bacilli just mentioned are at work in and out of the intestine and that either by their action or interaction they alter the colour, smell and reaction of the faeces according to the varying degree of their respective predominance?

This question of action and interaction of micro-organisms in the intestinal tract, together with the influence of drugs thereon offers a vast field for future research. As, so far as I am aware, practically nothing has been done in this connection I cannot bring forward anything in support of my suppositions but the following are of much interest in relation to those suppositions:

(1) Dr New invariably found the *B. Pyocyaneus* in stools which had been yellow on leaving Plaistow but which were found on arrival at the Lister Institute to have changed to green.

(2) On an enema of Mag: Sulph being administered the result is invariably green in colour whatever the colour of the motions may have been previously.

(3) Weak acid or alkaline rectal wash outs did not in the slightest degree alter the existing reaction of the stools. They rather tended to intensify the already existing reaction.

(4) Bienstock has shown that *B. Coli* and *B. Lactis Aerogenes* prevent the putrefactive decomposition of albumin by *B. Putrificans*.

(X)

Death is due to:

(1) Toxaemic absorption resulting from paresis of the gut.

As I have already stated such paresis is generally sudden. The abdomen suddenly distends Vomiting re-commences, the diarrhoea gradually lessens and either entirely ceases or blood alone is passed in small quantities, there is a sudden rise of temperature followed often by a sharp fall and frequently a persistent subnormal temperature. This temperature curve is identical with the one observed by Dr Sidney

Martin after the inoculation of animals with the toxins of the B. Protens. When there is ulceration of the intestine the toxæmia is more acute and small quantities of blood are passed.

(2) Broncho-Pneumonia; or both.

I have mentioned Broncho-Pneumonia as a frequent complication. It may become very acutely prominent and carry off the child before a toxæmic condition has been established. Where there is an active broncho-pneumonia present the temperature keeps persistently more or less high and Dr Pearson thinks that a persistent high temperature towards the end of a fatal case invariably is caused by a broncho-pneumonia. I cannot corroborate but think it quite possible.

Some of our cases died undoubtedly of a toxæmia and yet post mortem small patches of broncho pneumonia were found. Such cases towards the end had had a subnormal temperature following on a sharp rise. The toxæmia in these cases probably over-ruled the broncho-pneumonic effect on the temperature.

During convalescence from a very acute attack I found the resulting marasmus an extremely difficult condition to treat. The greatest care has to be taken in the diet but although plenty of

nourishing food was being given it was often impossible to get the child to increase in weight. I am inclined to think this troublesome marasmic state may be due to the fact that the biliary functions have not been properly re-established. Certainly in three such cases Schmidt's test remained negative as long as the marasmus lasted.

The principal object I had in view was to make clinical observations of the disease and to attempt to account for the great variations in the types of the stools.

The first few In-patients cases were therefore left untreated by drugs.

They were given rectal washouts with normal saline eight hourly. They were fed on Albumen Water 2 ozs to 4 ozs three times a day; and were given if in a state of collapse a saline infusion of from 6 ozs to 8 ozs with the addition of Adrenaline.

I had made myself acquainted with the almost endless list of different drugs which have been tried at one time or another, and more or less unsuccessfully. When I began the treatment of the Out-patient cases I therefore did not hope to find a specific, but rather confined myself to watching the effect of various drugs on the stools in this disease. Many different drugs were tried and as so many produced little or no apparent effect I will here touch

upon only those which seemed beneficial in the various types of cases mentioned and which produced a distinct change in the character of the stools.

Thus I found:

(1) That Hydrarg cum Cret gr. $\frac{1}{4}$ to $\frac{1}{2}$ every 8 hours is probably the most valuable drug in a case of ordinary mild summer diarrhoea when the stools are green, slimy, offensive and, as they usually are, alkaline. Those stools are quickly, under the influence of this drug, converted into stools which are yellow and approaching normal. Where the diarrhoea is mild and the stools yellow and acid, the effect of this drug is to convert them into green alkaline stools and then into normal. This result may possibly be produced as the result of a cholagogue action - acholic stools becoming green from the presence of biliverdin, and biliverdinous stools yellow from the presence of hydrobilirubin

(2) That 'Mist Iodi Co' i.e. Tinct Iodi

Glycer Ac Carbol aa M̄

Aq. Carui ad 3i

8 hourly

is valuable when the offensiveness of the stools is very marked and when the early vomiting is very troublesome.

(3) That Mist Bismuthi Bismuth Carb. gr $\frac{ij}{ss}$
 Mag. Carb. gr. $\dot{\dagger}$
 Muc. Tragac. q. s.
 Aq. Carui ad Zi

is useful where blood appears in the stools.

That Mist Bismuth Salicyl, i.e.

Sod. Salicyl
 Sod. Bicarb, $\bar{a}\bar{a}$ gr $\frac{ij}{ss}$
 Liq. Bismuthi hx
 Aq ad Zi

6 hourly

is useful in the very irritative diarrhoeas of older infants. It was given after a castor oil purge. It was beneficial also where the stools were pasty, frothy and offensive and also where there were streaks of blood present.

These mixtures were selected from the mixtures contained in the Hospital Pharmacopoeia, and although they were beneficial in the respective types of cases just mentioned of Zymotic Enteritis they proved to be of no avail in cases of true Cholera Infantum.

From clinical observations I had made respecting the derangement of the biliary functions, and from the post mortem corroborating evidence of such a derangement, I determined to try the effect of administering a cholagogue in cases where the liver was large, with tenderness over the hepatic region, and

where the stools were light coloured and offensive. Euonymin was the drug chosen and after some consideration the following mixture was arranged to be given under the name of 'Mist Euonymi Co.'

R_x Tinct Euonymi ℥i ii
 Liq Hydrarg Perchlor^{℥i}
 Spt Chlorof ℥i ii
 Aquam Anethi ad 3i

6 hourly.

The administration of this mixture in cases where the stools are light coloured and especially when there are also signs of some, even though it be slight, biliary functional derangement, produces a most markedly beneficial effect.

The stools very quickly become darker in colour or even green in some cases, they become less offensive, less watery, less frequent and, as experience showed me was the invariable result of such a change in the character of the stools - the child at once began to stop losing weight and showed every sign of being in a better state of health.

The earlier in the disease this mixture was given the more beneficial its action was. Of all the drugs or combination of drugs which were tried throughout the summer nothing, in my opinion, produced such good results as this Mist Euonymi Co when it was employed in cases which seemed suitable

for its exhibition. Once its action has been well established as evidenced by the change in colour of the stools and the improvement in the condition of the child its administration should be stopped for I think it possible to persevere in its use too long, with detriment to the child. Another drug should be substituted and the best drug in this instance I found to be Hydrarg cum Cret. I found if the Mist. Euclymii Co was persevered with for some time after its beneficial effect was obtained that a constipation ensued, only small hard brown lumps of faeces being passed and that as a result the general condition of the child ceased to improve.

Keeping in mind that here we have to deal with cases which are absorbing toxins from the intestinal canal, and that, if untreated, an acute toxæmia and an intestinal paresis will result, I am strongly in favour of clearing out the intestinal tract as thoroughly as possible of its toxic materials, and endeavouring to keep up a gentle peristalsis throughout the course of the disease.

While I consider that, towards this end, it is of the greatest importance that rectal irrigation should be employed throughout the attack I cannot agree with writers on the subject as to the beneficence of gastric lavage.

At the commencement of the attack washing out the stomach does no harm but I cannot see that it is productive of much good. I have never found the primary vomiting an obstinate symptom once appropriate treatment with diet, drugs and rectal irrigation had been begun. In the later stages of the disease when the secondary vomiting has begun and when the child is generally much collapsed, there is often a distinct risk in washing out the stomach: there is always an accompanying additional strain which the exhausted child can ill afford to bear.

In one of our cases the child survived only after energetic restorative measures, in a second case death resulted immediately after the passage of the stomach tube.

Rectal irrigation on the other hand is of great value and should be employed throughout.

A pint to three pints, according to the age, of warm water or saline solution may be employed at each irrigation, the catheter being passed high up - 12 to 14 inches of possible.

From rectal medication we obtained no results which justified the persistence of this means of administering drugs but I would here note the effect of giving acid and alkaline rectal wash outs. In cases which were passing alkaline stools it occurred to me to try the effect of changing the reaction of the contents of the lower bowel by administering an acid

rectal wash-out. Not only was the desired effect not obtained but the subsequent stools were even more alkaline in reaction. In like manner acid stools were not changed in reaction by alkaline wash-outs, they retained their acidity and in some cases were if anything more acid.

An active purge should be employed at the commencement of the attack to clear out the small intestine. For this purpose either Calomel grs 2-3 or castor oil $\mathcal{Z}\dot{\text{i}}$ to $\mathcal{Z}\ddot{\text{i}}$ are the best drugs .

I am convinced that we saved many lives by the prompt use of a cathartic again at a later stage when the diarrhoea had suddenly ceased or where only a small quantity of blood or serum was being passed accompanied by much tenesmus. In such cases Calomel gr $\dot{\text{i}}$ to gr $\ddot{\text{i}}$ was given, and if with no result, or if the child seemed getting worse, an enema of

Mag Sulph $\mathcal{Z}\dot{\text{i}}$ to $\mathcal{Z}\ddot{\text{i}}$

Turpentine $\text{M}\ddot{\text{i}}$

Aq Callid enough to dissolve

was passed high up the colon. A good action generally soon occurred and it was remarkable that the result was invariably green in colour. If there was a small action of the bowels or no action at all the early death of the child was the certain result. We found that the action of the calomel was helped by giving a hypodermic injection of Liq. Strychnin M $\dot{\text{i}}$ to $\text{M}\ddot{\text{i}}$ at the same time.

The patient's strength must be maintained as much as possible and stimulants must be given frequently and in small quantities. One of the best stimulants I found to be brandy and it should be given either by the mouth, or, if vomited, hypodermically. Ether, camphor, or strychnine may also be used with advantage.

It is very important that fluid should be supplied to the blood of these patients to make up for the great loss of fluid from the body from the excessive intestinal discharges. This is best done by the injection of normal saline into the cellular tissue of the abdomen or axilla. Half a pint at least, in twelve hours should be given and if the child is in a fairly satisfactory condition the fluid will be rapidly absorbed. If however, the diarrhoea has ceased, the abdomen is distending, the secondary vomiting has commenced and the child is in a toxæmic state, the absorption of the fluid may be extremely slow, being hardly appreciable and it taking some hours for 5 or 6 ozs to be absorbed. To avoid bruising or sloughing of the tissues and with a view to more rapid absorption it is important to see that the fluid is injected deeply into the cellular tissue. In one or two cases I injected the saline into the peritoneal cavity with beneficial results.

The stomach and small intestine become inflamed and many abnormal processes occur there but Dr Pearson and I believed that the greatest mischief occurs in the large intestine, especially at its commencement. Here the toxins are absorbed and if ulceration is going to occur it occurs here. We have seen ulceration here and also in the small intestine but never in the small intestine alone. Dr Pearson therefore considered the advisability of appendicostomy and obtained the consent of the parents to an operation in two instances. He had previously practised the operation in all the post mortems of the previous Spring and was able to complete the operation very speedily and simply. The Appendix was drawn out, stitched in position, packed round and opened. A No. 3 Jacques catheter was inserted and two pints of warm normal saline were passed through the large intestine. Perhaps an operation in those two cases was unjustifiable for one was suffering from broncho-pneumonia and both were practically in extremis. In both cases the improvement in their condition was immediate but unfortunately, it was not sustained. The first child died 10 hours and the second 26 hours after the operation but we considered that their lives had not been shortened but if anything prolonged by the operation. Dr Pearson considers that, having regard to the present state of knowledge concerning the

treatment of Cholera Infantum, it is justifiable and very desirable that appendicostomy be performed as soon as Choleriform symptoms manifest themselves and he believes that, if the large intestine was kept clear and in good tone by injections through the appendix many cases which would otherwise prove fatal would recover.

The question of diet is a very important and difficult one. Following the advice of many writers on the subject milk was stopped entirely and the children were fed on albumin water. I am convinced that the continuation of this diet for longer than 48 hours is bad. Many cases which were having particularly offensive stools were found on enquiry to have had nothing but albumin water for the previous week or so and Dr New pointed out that albumin water was an ideal pabulum for the B. Proteus. Sherry Whey seemed to answer just as well as albumin water in the matter of being easily retained by the stomach but sherry whey contains too much sugar - another excellent pabulum for B. Proteus.

Having had much success with the use of Pancreatic Emulsion as prepared by Messrs Savory & Moore in the treatment of the intractable marasmus of convalescence I suggested its use in the acute

stages of the disease and it seemed to answer well in keeping up the child's strength and condition.

We considered it is beneficial because:

1. It stimulates pancreatic and hepatic secretion.
2. It contains no starch, albumin, or added sugar.
3. It contains glycerine and fat, both valuable foods and both antagonistic to the growth of the B. Proteus.
4. It is easily taken by the infants, is quickly absorbed and is simple to prepare with sterile water.

Its only objection is its expense.

Where it is not obtainable I think it is good treatment after the vomiting has ceased and that should be almost invariably within 48 hours, to place the child on a diet of condensed milk diluted according to age and freshly prepared each feed rather than persevere in the giving of albumin water and the encouragement of intestinal putrefactive processes.

I certainly do not agree with Holt regarding the great danger of giving milk within periods of from one to two weeks from the onset.

By prophylactic measures much could be done to diminish the huge number of infants attacked by this disease, and when, those measures having been taken, the disease made its appearance, I am per-

suaded it would do so in a less virulent and less fatal form.

Taking into account the fact that the disease claims as its victims on an average, nearly 20,000 infants yearly in England and Wales alone, and that those victims are the children of the poorest and most ignorant class I consider that the whole question of enforcing prophylactic measures is one for State control.

I would put forward certain suggestions as to what measures I consider should be taken to endeavour to prevent the onset and spread of Zymotic Enteritis, and in doing so I would formulate those suggestions in the shape of a scheme. If such a scheme were adopted I am convinced the death rate from this disease would rapidly fall and in any case our knowledge of the disease would be considerably widened.

I would therefore propose:

I. That Zymotic Enteritis with certain restrictions be made a certifiable disease. The restrictions would suggest themselves as experience was gained but to start with it would be advisable to make a limit as regards age, e.g. only those cases to be certified which were between the ages of 6 months and three years. And again only those cases to be certified which were not being breast fed.

II.

That Public Notices be posted in the poorer districts of all large towns and that leaflets be distributed at hospitals, dispensaries, etc., pointing out:

- (1) The dangers of the household fly, its connection with the spread of the disease, and the methods by which the numbers of flies may be diminished such as by thorough "spring-cleaning" which destroys so many flies' eggs.
- (2) The best manner of storing infant food in covered vessels so that it is not accessible to flies.
- (3) The best methods of feeding in hot weather, such as:
 - (a) The avoidance of weaning in hot weather.
 - (b) The importance of regular feeding or if the child be already weaned
 - (c) The dangers of improper or over-feeding.
 - (d) The proper proportions in which the feeds are to be prepared and the importance of having each feed freshly prepared.
 - (e) The importance of attending to the scrupulous cleanliness of nipples, bottles, etc.,

- (4) The importance of plenty of air, sunlight and cleanliness of the homes as preventive measures.

III.

That the Medical Officer of Health should see

- (1) That as far as practicable refuse material is burned and that as far as possible manure heaps in towns is entirely done away with.
- (2) That sheds, privies, stables, etc., are washed down with Paris Green Water as recommended by Dr. Newstead of Brighton.
- (3) That there is a good milk supply in the district, and that the milk when delivered is delivered fresh.
- (4) That the dairies are particularly warned against the danger of flies, and that they are advised as to the best methods of preventing flies coming into contact with the milk.

IV.

That lady visitors be appointed to see that proper methods are carried out in the homes as to (1) the cleanliness of the homes and person and the airing of the rooms, (2) the proper storage of the milk particularly as regards its efficient protection from flies, (3) the washing out of closets, cupboards and privies with solution

supplied by the Public Health authorities, (4) the immediate placing in antiseptic solutions and subsequent careful washing of soiled diapers.

V. That certified cases be visited by the M.O.H. or his assistants and that if the case is found to be incapable of being properly isolated and treated at home that it should be removed to a temporary sanatorium.

VI. That temporary sanatoria of wood or iron be erected on pillars in open spaces and if possible on high ground outside the town. It might be possible in some cases to utilise old small-pox hospitals. The temporary sanatoria should be so arranged:

- (1) That the ground floor was used by the administrating and nursing staff both for living and sleeping.
- (2) That the upper floors were used for patients.
- (3) That the windows of the wards could be removed and their place taken by fine gauze netting.
- (4) That the walls, floors and entire furniture could be washed with some harmless solution inimicable to flies. Tannic acid has been suggested as such a solution.

- (5) That verandahs were attached to the wards or means provided for the patients being in the sunlight several hours each day.
- (6) That each child was allowed 1,000 cubic feet air space.
- (7) That no stabling was near the sanatorium.
- (8) That there was an efficient furnace for the destruction of all soiled material.

The burial of fatal cases would be undertaken with the same care as is given in deaths from other infectious diseases.

Much would be gained in our knowledge of the disease were such sanatoria existent. It is true we have no specific treatment which cuts short an attack of this disease but I am confident that many of the worst cases if treated in such sanatoria would have their attacks lessened in intensity and that many lives would be saved.

But the chief advantage at first gained would be our better acquaintance with every phase of this plague. For by careful clinical observations, precise records of all the details of every stool passed by each patient, exhaustive post mortem investigations, in short, by detailed records of the whole history of every case from first to last, it would inevitably follow that our knowledge would become wider and more definite and as a result our treatment more certain and effective.

Great assistance could be given by philanthropists in helping towards the prevention and spread of the disease, and it is the duty of the Medical Profession to bring to the notice of such members of the lay public the ways by which by their pecuniary assistance many of the most valuable lives of our population could be saved. We are told that in New York and in many other large American cities the mortality from the disease has been largely reduced by the provision of fresh air funds, seaside homes and the means for supplying pure milk to infants. Fresh air and sunlight are of the very highest value in the prevention and treatment of the disease and the providing of means by which they can be brought within the reach of the infant population of all our large towns cannot be too highly commended. Large sums of money which are being at the present moment expended on, as far as one can judge, comparatively unimportant philanthropic objects might well be diverted into such a useful channel as this. Certainly no more laudable object for practical philanthropy could possibly be found.

The most of the work recorded here was done during the Summer of 1906. I delayed writing this Thesis till after the Summer of 1907 in the hope that I might make further investigations and put my suppositions further to the test. Unfortunately for my work the summer of 1907 proved to be

almost barren of cases of Zymotic Enteritis, and although I went down to Plaistow for a month on purpose to be in a position to make further enquiries into the subject, I was unable to carry out my projected plan of investigation except in being able to gather together some interesting percentages of the floors on which cases of bad diarrhoea are usually found and those figures I have duly commented upon.

I am fully alive to the fact that the suppositions I have arrived at throughout this Thesis have been made on observations carried out on a small number of cases and that it is possible many of these suppositions will be proved to be unfounded, yet although the clinical and post mortem observations occurred in a small number of cases they may all be relied on as having actually been observed, for their exactitude was vouched for in every case by my colleague Dr Pearson and in some cases by other trained observers.

I have the good fortune to be at present a Resident at the East London Hospital for Children, Shadwell and this ensuing summer I hope to have the opportunity for further work in connection with Zymotic Enteritis and Cholera Infantum. Some of the members of the Honorary Staff

have expressed their willingness that opportunities should be given for the work and have promised that special wards exclusively for the treatment of this disease shall be opened. Given the opportunity I should like to carry on the work on much the same lines only on a much larger scale than in 1906.

I would give the same careful attention to recording the history from first to last of each case; keeping the same record of stools and trying to investigate further the causes of their rapid variation and the effect of drugs thereon. I would give fuller attention to the pathology, macroscopic and microscopic; and bacteriological observations in connection with the broncho-pneumonic patches and the secretions particularly the hepatic, pancreatic and those of the mouth would be made.

The question of fostering our infant population is indeed a most vital one and I consider that the aspect of it to which least attention has been given - the saving of infants' lives from the ravages of the diarrhoeal diseases - to be the most important one. I confidently predict that, at a date not very far distant, the full significance of the declining birth rate will make itself manifest and then there will be an awakening. But why should there be any further delay? Why should any effort, any

expense be spared when the matter at issue is such an important one? The victims are helpless infants. It is our duty, individually as citizens of a great Empire, collectively as the foremost nation to see that every effort is made and that quickly, that this waste of lives is stopped. Let not the crime be laid at our doors that we failed in this duty.