ENTERAL DISORDERS OF CHILDHOOD.

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INTRODUCTION.

The disorders classified by the Registrar General as "Diarrhoea and Enteritis" have been the subject of much investigation but there can be few about which so many contradictory statements have been made.

HISTORICAL REVIEW.

Diarrhoea at teething was observed by Hippocrates. Aretaeus in 100 A.D. described cholera infantum as a most acute illness associated with vomiting and diarrhoea, and states that the condition tends to occur in hot weather. In 852 A.D., Rhazes notes the connection between teething and diarrhoea. Begallardus (1492) also remarks on this association and condemns milk contamination.

In 1565, de Vallenbert again brings out the association between teething and diarrhoea. In 1653, Pewell stated that one of the most important causes of enteritis was milk contamination and advised avoidance of milk for a day as treatment of the condition. Harris in 1647 had already stated that enteritis was most common from July to September and that he thought the heat of the season weakened the infant. The Heberdens in 1710 were of the opinion that too much or unsuitable food could be held responsible in many cases and emphasised the importance of careful regulation of the infant's diet. They considered tincture of opium to be a most valuable adjunct to treatment.

Benjamin Rush (1745) was the first to write anything like

a systematic account of the condition and he uses the term cholera infantum to describe the form of enteritis which was most common in hot weather. He noted that the most susceptible ages were from two weeks to two years. His description of the illness shows that it was almost exactly the type which is later described as acute choleraic enteritis. He is very strongly of the opinion that dentition aggravates the condition. He considers that no remedy is reliable.

Vogel (1866) was the first to draw attention to the fact that enteritis is most common in artificially fed infants. He recognises the association with teething.

In contra-distinction to many of the previous writers, Goodhard (1885) believes "summer diarrhoea" to be easily curable by simple means. He makes the important statement that any measures directed towards the improvement of hygiene will reduce the incidence of acute diarrhoea.

Smith (1884) recognises three types of enteritis. (a) A simple non-inflammatory type due to improper feeding and teething. (b) An acute inflammatory type (entero-colitis) which is commonest in hot weather and which affects bottle-fed babies. He makes the important statement that the condition is infectious. (c) The third type he calls choleraic diarrhoea which he believes can be associated with all the above-mentioned factors.

West (1884) attempts a classification into simple catarrhal diarrhoea and dysentery. He found that these conditions

were commonest in children of six months to two years of age and that they occurred mainly in the months of August, September and October.

From the time of Hippocrates then up to the nineteenth century, certain facts came to be recognized, namely, that enteritis was a disease of infants most of whom were artificially fed which tended to occur in the hot months of the year. The association of the disease with dentition, faulty diet, imperfect hygiene and towns was noted. (59, 141, 154, 156, 166.)

CLASSIFICATIONS.

The classifications of the enteral disorders of childhood are many and varied, reflecting the difficulty found in assessing the different entities. The first classification was based upon the clinical aspect of the various forms of enteral upset, upon symptomatology, the term cholera infantum being one of the designations used. Then, later, when it was found that this scheme was not satisfactory, a further attempt at classification on a pathological basis was made upon what was supposed to be the anatomical site of the lesion. Thus, such a term as ileocolitis came to be introduced. It was soon recognised, however, that there was apparently no relationship between the morbid anatomy and the various forms of enteritis. The next phase coincided with the increase in bacteriological knowledge which led up to the recognition of the dysenteric group. This classification was based upon the various micro-organisms which, from time to time,

were thought to be implicated. This point will be discussed more fully later when the question of etiology is debated. I can now state briefly that no one organism has been definitely isolated which could be shown to cause the important and most deadly form of enteritis. Further classifications were based upon the theory that imperfect diet and parenteral infections were potent causes. In fine, it may be said that a certain amount of progress was made because from the main bulk of cases of enteritis could be separated those due to known bacteria, to dietetic errors and to the presence of parenteral infections. There still remains one group of cases of acute enteritis, the most deadly type by far, which has in turn been included in each of the categories enumerated above and it will be to this group in particular that I shall refer in succeeding pages. under the title of "Acute enteritis of uncertain etiology". Upon examining suggestions as to the etiology of this last type of enteritis, I found that the bacteriologist and biochemist vied with each other to produce a multitude of bacteriological and biochemical causes while the clinician, as inferred above, remained faithful to two main theories, that of a dietetic cause and that of a parenteral cause. Among the organism which have been incriminated in the last twenty years are B.Morgan 1, B.proteus, unnamed gram negative bacilli, anaerobes, B. MacConkey, B. paracolon, abnormal types of B. coli and various fungi. The typhoid, paratyphoid, dysentery and salmonella organisms have been purposely omitted from this list because, although until fairly recently they were thought to be

implicated as causes of acute enteritis of uncertain etiology, there is a growing literature to show that the disorders caused by these organisms are quite separate clinical entities (8,60).

It is obvious upon surverying this formidable list of organisms that the bacteriology of acute enteritis of the type to which I am now referring is far from being in a satisfactory state especially when it is remembered that the majority of the organisms mentioned above can be found in the stools of children who are apparently well and who have no history of any intestinal upset.

The biochemist has been nothing if not ingenious in providing causes for this severe illness. The biochemical theories vary from those of diminution of gastric secretion of hydrochloric acid, increase in the alkalinity of the contents of the upper levels of the small intestine and increased permeability of the intestine to those somewhat obscure metabolic disorders variously ascribed to changes in air temperature and subsoil temperature. To do the biochemist justice, however, it may be said that unquestionably his work on the body fluid and cell changes in dehydration and starvation have been of the greatest assistance to the clinician in his treatment of cases of enteritis. (5, 6, 8, 19, 20,21,22,23,29,30, 31,32,33,34,39,40,43,48,54,60,62,63,70,71,72,73,77,79,80,81,83,84, 85,86,87,78, 90,92,94,95,103,104,106,109,110,113,118,119,120,133,136, 138,142,143,144,145,146,147,157,162,165.)

So far as the clinician's theories of a dietetic or a parenteral cause of this acute type of enteritis is concerned it is my belief, speaking from personal experience, that although there

are definite clinical grounds for their inclusion as causes of certain forms of enteritis, they play no part in the production of acute enteritis of the deadliest type and, as such, they have been given undue prominence in the literature. I think that a survey of the points indicated above will show ample justification for the qualification "of uncertain etiology."

GENERAL ETIOLOGICAL FACTORS.

To return to the etiological factors which have a bearing upon the whole group of enteral disorders of childhood, we are upon much surer ground when we discuss the Public Health aspect. It is agreed that the steady reduction in the incidence of, and to a lesser extent the mortality from "Diarrhoea and Enteritis" is directly due to the introduction of legislation improving housing and sanitary arrangements in general with a consequent reduction in the number of flies and of propinquity. Other advances having a bearing on this reduction are the provision of centres at which mothers are taught safe methods of feed preparation as well as general cleanliness in the running of their homes. The careful supervision of the production of cow's milk, the general use of pasteurisation and the manufacture of almost sterile dried milks have also played a large part. I believe that the greatest reduction in enteral disorders has been in the less fatal types which, in my opinion, are due to faulty feeding and to parenteral causes and which are most easily influenced by public health measures.

Reduction is apparently not taking place in the bacterial

forms of enteritis and dysentery of the Sonne type especially is becoming much commoner in London. This is no doubt in part due to better diagnosis but there is an actual increase also. See Table 1.

Table 1. Taken from Registrar General's Returns.

No of Cases of Dysentery notified in:-

	1928	1929	1930	1931	1932	1933	1934	1935	1936.
No.	683	573	538	809	924	783	763	1,177	1,333
Deaths	155	98	97	116	133	92	103	116	87

I believe that acute enteritis of uncertain etiology has remained almost stationary in its incidence for the following reasons. While the diminution in the number of deaths from "Diarrhoea and Enteritis", for example from 5,394 in 1926 to 3,220 in 1936, appears great, a rather false impression is given. It must be admitted that there is a great reduction in the incidence of enteral disorders. Conversation with any clinician in a children's hospital will confirm this but the figures regarding deaths are apt to be misleading. When it is remembered that the birth rate (Rate per 1000 population) in a diminishing population has fallen from 17.8 in 1926 to 14.8 in 1936, it is realised that a smaller infant population is at risk now than even such a short time ago as ten years. In this case then the reduction in the number of deaths is partly a reflection of the diminishing birth-rate and the apparent reduction is not so great as would be supposed. This is seen when the infant mortality figures (Deaths per 1000 live births)

are considered, namely 7·77 in 1926 and 5·32 in 1936.

DETAILS OF THE PRESENT INVESTIGATION.

After a survey of the facts which have been set out above and with the knowledge that my own clinical observations of cases of acute enteritis showed material differences from the majority of recorded observations, certain questions emerged.

(1) What were the reasons for the differences of opinion?

(2) Might it not be possible that the clinical basis which must form the foundation of every investigation was at fault? In other words, was the true clinical state of affairs being lost in a maze of complex biochemical and bacteriological research?

(3) In close association with the last question, had the reduction in the incidence of diarrhoeal upsets of the less fatal types brought to light any new clinical entity which until recently had been obscured? Obviously the last and most important question could only be answered if a thorough clinical investigation was carried out.

Fortunately at the time, two and a half years ago, when these questions presented themselves, the opportunity to proceed with such an investigation arose. The London County Council, in view of the increasing difficulty in dealing with cases of enteral disorders in children in general hospitals, principally on account of the increasing number of ward outbreaks, decided to admit such cases to their isolation hospitals. The various medical officers

of health circularised the local practitioners and hospitals notifying them that arrangements had been made to provide accommodation in the local isolation hospital for cases of enteritis in children.

At first it was considered that open wards should be set aside for the reception of these children but, after strong representations from the superintendents of the isolation hospitals who fully appreciated the danger of such a procedure, it was decided to adapt a separation ward for this special purpose. The Eastern Hospital was the first of the isolation hospitals to do this. A ward containing nineteen separate single bedded cells and one dayroom containing two cots was redesigned in order to deal with the difficulties which it was felt might arise. Overflow cases were to be admitted to the ordinary separation blocks and nursed in separate single-bedded cells. The details of nursing technique, kitchen accommodation, methods of food preparation and the various problems which arose will be considered later.

Although the first intention was to admit to this special ward only such cases as were certified as "Gastro-enteritis" "Acute Enteritis" or "Zymotte Enteritis", it was soon found most convenient to admit all cases of enteral disorder in children rather than to distribute them throughout the hospital. Thus those cases certified as "Dysentery" were sent to the special ward also. This had the added advantage that one medical officer dealt with all such cases. As far as possible the children of five years of age and under were admitted to the special ward while those children of

over five years of age were sent to the overflow ward. This being the case, it was decided to study the whole range of cases of enteral disorder admitted to these wards.

This necessarily meant a considerable increase in the scope of the investigation but it was felt that a complete inquiry would ultimately be of more value than the study of a limited number of cases of one particular type.

With these introductory remarks, I shall now proceed to a description of the objects of the investigation, the facts brought to light and the conclusions reached.

MAIN OBJECTS OF THE INVESTIGATION.

(1) To study clinically all types of enteral disorder of childhood admitted to the special wards and to endeavour to throw fresh light upon the various forms of enteral disorder.

(2) To try to improve upon existing methods of diagnosis at the bedside and to form a simple routine for this purpose.

(3) To assess the value of the various forms of treatment which have been suggested and to try any fresh methods that might be indicated by the clinical findings.

(4) To evolve the safest possible method of nursing, in hospital, a number of young children suffering from a variety of enteral disorders.

CLASSIFICATION.

It was obviously necessary to have a scheme of

classification to work upon and that which I used was based upon existing bacteriological knowledge and upon personal experience of ward outbreaks of enteritis during the measles epidemic of 1936. It was not found necessary to revise this classification in the light of subsequent experience and it is probably as complete as one upon such a basis can be.

The cases are first divided into two main groups, the first being:-

A. Non-infective Enteritis.

This is sub-divided into three sub-groups:-

(a) Dyspeptic Enteritis. This term is intended to cover the enteral upsets caused by faulty feeding or unbalanced diet which may give rise to such conditions as coeliac disease, carbohydrate dyspepsia and fat dyspepsia. Another type included under this heading is that associated with dentition.

(b) Mechanical Enteritis. Under this term come all those cases of enteritis associated with such conditions as pyloric stenosis, intussusception and worms.

(c) Symptomatic Enteritis. Corresponding to the type commonly associated with a parenteral infection such as otitis media, mastoiditis or pyelitis, to mention only a few.

The second group is termed:-

B. Infective Enteritis.

This group is further sub-divided into two sub-groups:

(a) Infective Enteritis of known etiology.

This was intended to include all enteritis due to a known micro-organism such as the dysentery organisms Shiga, Flexner and Sonne, the typhoid and para-typhoid organisms and organisms of the Salmonella group.

(b) Infective Enteritis of uncertain etiology.

This heading designates the type of enteritis which may be described briefly as that type in which no known causative organism can be incriminated, no parenteral, dyspeptic or mechanical cause can be found but which takes the form of two completely different clinical entities which have been designated:-

1. Acute Toxic Enteritis;
2. Acute Choleraic Enteritis.

The inclusion of these two types in the infective group was based upon experience of outbreaks in wards when the infectivity became obvious and the two types were clearly distinguishable.

Finally came a group of cases which could not definitely be included in any of the above mentioned categories. These were termed simply:-

C. Unclassifiable types of enteritis.

GENERAL PRINCIPLES OF THE INVESTIGATION.

The investigation of cases was originally planned to extend over a period of two years but, although this report is based upon the results of this two years' survey, so many points came to be raised, as will be seen from what follows that it may have to be continued over a period of several more years. The maximum age limit was set at fifteen years and the total number of cases investigated was 275. All these cases were admitted directly from outside, internal transfers being excluded as they were mostly

suffering from some other complaint with consequent obscuring of the clinical picture. The routine examination of each case was very complete and included, besides the necessary complete physical examination, a thorough search for parenteral infections.

The parenteral infections which were particularly looked for were otitis media or mastoiditis, catarrhal conditions of the upper respiratory passages and pneumonia, as is so strongly stressed by Graham (60). The urine was examined for any abnormal constituents such as pus cells and organisms. The faeces were tested for abnormal organisms on two separate occasions at an interval of three days. In the later cases serological examination was done when possible. In addition, mothers were closely questioned about diet to find out if there was anything wrong with the food or its method of administration.

The reasons for this investigation and details of the ideas behind it being stated, the next step is to consider the findings.

DISTRIBUTION OF THE 275 CASES USING THE CLASSIFICATION DESCRIBED.

A. Non-infective Enteritis. Total = 35.

(a). Dyspeptic Enteritis. No. = 15.

(b). Mechanical Enteritis. No. = 7.

(c). Symptomatic Enteritis. No. = 13.

B. Infective Enteritis. Total = 198.

(a). Enteritis of known Etiology. No. = 89.

(b). Enteritis of uncertain Etiology. Total = 109.

1. Acute Toxic Enteritis. No. = 32.

2. Acute Choleraic Enteritis. No. = 77.

C. UNCLASSIFIABLE TYPES OF ENTERITIS. Total = 42.

GENERAL ETIOLOGICAL FACTORS IN ENTERAL UPSETS IN CHILDREN.

Before passing on to a detailed analysis of the cases, it is best to deal now with certain etiological factors which have a bearing on all forms of enteritis. Of these the most important are feeding, social conditions, climate and Public Health.

Feeding.

It is generally agreed that enteritis is usually found in children who are artificially fed. (29, 47, 76, 132, 153).

The present series of 275 cases bears this out as only three of the children were breast fed and three had combined feeding. The remaining 269 cases were either fed on dried milks, cow's milk or in the case of the older children on "the run of the house." The relationship of breast feeding to the occurrence of enteritis is very difficult to assess. It is suggested that children on the breast are much less liable to contract enteral disorders and this is certainly borne out by the figures of various authors and by those of the series under discussion. The reasons for this comparative immunity of the breast fed infant from the various forms of enteritis must be several. Breast milk is the physiological food for the infant and children so fed are less liable to develop the dyspeptic forms of enteritis. Catarrhal children are usually

bottle fed. There is less risk of contamination of the breast milk with dust, dirt, pathogenic organisms or toxins with a consequent reduction in the incidence of infective enteritis in such children. A further possibility is that immunity may be passed from the mother to the child in the milk. This seems possible in the enteritis of uncertain etiology as the disease is not recognised in adults and is rare in breast fed infants. (3,8,104,115).

SOCIAL CONDITIONS. the various forms of enteritis are commonest among children of the poorer classes living in over crowded and unhealthy surroundings. All my cases came from the poorest class and Findlay and Maitland-Jones from wide experience in private practice, state that enteritis is uncommon among private patients. (47,80,104).

CLIMATE. Climatic changes seem to have some bearing upon the epidemiology of certain forms of enteritis. The bacterial forms tend to occur in the warm weather of the late summer while both types of enteritis of uncertain etiology are, in my experience commonest in the winter and autumn months. So far as London is concerned "Summer Diarrhoea" in a severe form has disappeared. Christie informs me that during the warm spells of the summer of 1939 there was a considerable number of very mild cases of diarrhoea in Willesden. None of these cases was of a serious nature and all responded to simple dietetic measures. The outbreaks rapidly came to an end when the warm spells finished. None of these cases were admitted to my wards. (24).

C H A P T E R 1.

A. NON-INFECTIVE ENTERITIS.

Total number = 35.

This type forms a small part of my series (12·7%) and several explanations for the smallness of numbers suggest themselves. A steady decline in the frequency has been recorded during the past forty years. The public are now educated to recognise that enteritis may be due to faulty feeding and digestive upsets and to seek early advice at Maternity and Child Welfare centres where the children can receive out-patient treatment. The family doctors are now better able to give advice on the proper control of diet. As the result of these improvements only the acutely ill patients need in-patient hospital treatment. The paucity of cases belonging to the mechanical and symptomatic groups cannot be explained in this way. The mechanical type would not ordinarily be diagnosed as enteritis but would be diagnosed and treated according to the mechanical defect. Thus cases of pyloric stenosis, intussusception and worms would be sent to general hospitals. As for the symptomatic type the explanation is more obscure. While it is likely that some of these cases are sent to general hospitals for the treatment of the parenteral infection, which is considered to be the cause of the enteritis, I think the main reason is that although diarrhoea is often associated with parenteral infections, especially those of the respiratory tract, it is seldom of a severe nature. The association, as I shall try to prove later, has been over-emphasized in the past.

(a). Dyspeptic Enteritis.

Only fifteen cases of this type (5•5% of the total) were admitted during the two year period; ten were due to dietetic errors and five were associated with dentition. These figures support Smellie's thesis who considers the incidence negligible (153). There were two deaths in this group, one a child of one month who had been taken off the breast for no apparent reason and fed on Ostermilk, the other, an infant of fifteen days, who had also been taken off the breast after ten days. Both were admitted in a state of inanition from which there could be little hope of recovery. In neither case was there any evidence of a parenteral infection and no abnormal organisms were found in the stools. Permission for post-mortem examinations was refused in both cases. Details of ages and duration of illness of the remaining eight cases are given in the accompanying table. (Table 2). Seasonal incidence was unimportant as the cases were regularly distributed throughout the two years.

Table 2. Table showing age and sex distribution and duration of illness of cases of dyspeptic enteritis in children surviving.

Case No.	1.	2.	3.	4.	5.	6.	7.	8.
Ages.	9/12	7/52	11/52	3/12	9/12	16/12	2/12	8/12
Sex	F	F	F	F	F	F	F	M
Days of Illness	3 days	7 days	8 days	10 days	3 days	2 days	7 days	2 days

Average duration of illness = 5.25 days.

The foregoing figures show that the illness is mild and short-lived if treated in the early stages. It is important to question the mother closely to find the dietetic error. This is often gross and simple correction has the desired result within a very short time. This is in agreement with the opinions of Graham and Maitland-Jones (60, 104).

All my cases developed diarrhoea after a sudden change of diet. Six of the children became ill a day or two after being taken off the breast and put on artificial feeding. The remaining four developed diarrhoea shortly after a change from the food to which they had been accustomed. One child, eight months old, had been fed on cakes.

In the five cases where enteritis was associated with dentition the illness was milder than that due to faulty feeding. The ages, sex, distribution and duration of the illness are shown in the following table (table 3).

Table 3. TABLE SHOWING AGE AND SEX DISTRIBUTION AND DURATION OF ILLNESS OF CASES DUE TO DENTITION.

No.	1	2	3	4	5
Age	6/12	6/12	7/12	9/12	7/12
Sex	F	M	M	M	M
Duration of Illness.	3 days	3 days	2 days	4 days	5 days

As soon as the tooth erupted the diarrhoea ceased. The only treatment necessary was careful supervision of the diet and attention to mouth toilet.

(b). Mechanical Type of Enteritis.

Seven cases fell into this group and there was one death. The fatal case, a child of nine weeks, had intractable diarrhoea with occasional vomiting and abdominal distention. No other abnormality could be found on examination. The general condition rapidly became worse and death occurred in three weeks. Post-mortem examination revealed a hypertrophic pyloric stenosis which was the probable cause of the enteral upset.

Of the remaining six cases three were due to constipation-pseudo-diarrhoea - two to round worms andone to intussusception. The latter case was detained long enough to make a diagnosis and then was sent to a general hospital for operation. The response of the other five cases to treatment was good. Regulation of the bowels in the three constipated children and treatment for round worms in the other two soon resulted in cessation of the diarrhoea.

Of these two groups, (dietetic and mechanical) it may be said that recovery will take place if suitable measures are adopted. Two provisions must be made. Early treatment is essential when enteritis is due to faulty feeding. Both cases which died were almost moribund on admission while the remainder had only been ill for 3 days at the most. In the mechanical group the cause of enteritis may be of such a nature that surgical treatment is necessary.

This must be borne in mind when each case is under investigation.

(c). Symptomatic form of Enteritis.

Under the designation are placed all forms of enteritis which are directly associated with an ex-enteral focus of infection. It is generally agreed that of these ex-enteral foci the respiratory system is the most important although infections elsewhere can be associated with diarrhoea. While this theory is widely accepted, there are certain divergencies of opinion of very considerable importance. The actual mechanism by which the diarrhoea is produced still remains in doubt. Various suggestions have been made and it is possible that they are all partially correct. Of these I think that swallowed sputum and the excretion of toxic through the intestinal wall are probably the two causes most likely to cover the greatest range of possibilities (46, 60, 110,). While the idea of swallowed sputum may quite well explain the enteral upsets associated with such conditions as otitis media, tonsillitis and pneumonia, to mention only a few infections of the respiratory tract, this mechanism cannot explain the diarrhoea that may be associated with such conditions as pyelitis or impetigo. It is in these conditions that the theory of absorbed and excreted toxin holds most promise. Perhaps it may be said in a general way that a combination of both factors is nearest the truth. However, the

mechanism by which the enteritis is brought about does not seem to be subject to such difference of opinion as the actual association between parenteral infections and enteritis. One school of thought, and this is mainly American, is of the opinion that acute enteritis is a development of a parenteral infection of which the most important is otitis media (48, 77, 81, 113, 153).

The counter to the argument that treatment of such a parenteral focus of infection is not of great value is that it has not been thorough enough. Thus, from America has come the suggestion that antrostomy should be performed in cases which do not respond to simple paracentesis. In such cases it is suggested that "masked mastoiditis" is the cause. The more conservative members of this school, mainly in Great Britain, while agreeing with the theory of an ex-enteral cause, have come to the conclusion that such extreme interference is not justified. (48, 77, 81, 113). The other school of whom I shall mention Findlay, Mitchell and his co-workers, Yambolska and Johnson and their co-workers (47, 80, 118) are of the opinion that otitis media, upon which most work seems to have been done, is not associated specially with enteritis. Findlay in his paper shows in a very conclusive manner that, although enteritis may be associated with otitis media, it may equally well be associated with any other ex-enteral infection of childhood. He condemns the practice of

antrostomy and is guarded in his opinion on the merits of even paracentesis. The theory of the connection between the middle ear inflammation and enteritis appears to be based mainly upon the frequent post-mortem finding of pus in this situation in fatal cases of enteritis. Le Mée (92) is of the opinion that the pus found in the middle ear at post mortem was simply that which had drained there from the nasopharynx in consequence of the shortness of the Eustachian tube in infants but Ebbs did not support this view because, in his opinion, milk would also gravitate up the Eustachian tube of the infant if this mechanism was effective. He supported the view that otitis was a potent cause of enteritis and advocated paracentesis at times followed by mastoidectomy. Certain other authorities tend to steer a middle course and believe that extra-enteral infections can be associated with enteritis but that this is rarely of the type associated with toxaemia and intoxication (42, 47, 60). Graham has stated that this type of enteritis is on the increase but considers that it may simply be due to greater frequency of recognition. Personally, when I commenced this investigation I was uncertain whether ex-enteral infections played an important part or not and for this reason my clinical examination always included a thorough search for such a focus as was advocated so strongly by Graham. (60). I may state briefly at this point that in no case of acute enteritis

with toxaemia or dehydration did I find any parenteral focus of infection at the onset of the illness. This induced me to believe that such foci when found in association with acute enteritis either formed part of the illness, that is to say, that they were part of a definite clinical syndrome or that they were due to a secondary infection. This latter opinion coincides with that of Mitchell and his co-workers, who state that the source of the otitis is to be found in the habit of herding children together and of Maizels and Smith who consider that the significance of otitis media varies with different patients (103, 118,). Of the total of 275 cases which are the subject of this thesis only thirteen (4.7%) were ascribable to a parenteral focus of infection. By this is meant that they could not be placed into any of the other categories and that each case was suffering from an ex-enteral infection of one or other kind. Certain cases cleared up when the parenteral focus was treated but the majority did so long before cure of the focus and appeared simply to respond to the careful hospital régime.

One death occurred of a child of two years of age who was admitted with a wide spread broncho-pneumonia and died on the seventh day after admission. During the seven days the stools were frequent and green but there was no vomiting. The youngest child was five months old and the oldest six years old. Six of the cases were under one year. The

six distribution was of no significance, there being six male children and seven females. Although the number is small and the ward conditions were such that only a limited number of cases could be accepted it is noteworthy that eight of the cases were admitted between August, 1937 and January, 1938, and four between August, 1838 and December, 1938. That is to say, the greatest incidence was in the autumn and winter months at the time when catarrhal conditions are most prevalent. The table below shows this point.

TABLE 4.

TABLE SHOWING SEASONAL INCIDENCE.

OF CASES OF SYMPTOMATIC ENTERITIS.

Year	Jan.	Feb.	Mar.	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
1937	-	-	-	-	-	-	1	1	1	1	2	1
1938	2	-	-	-	-	-	-	2	-	-	1	1

Clinically few of these cases gave rise to anxiety, nine being assessed as "fair" on admission and four as "poor". Included in the "poor" group was the child who died. Vomiting was absent in all cases. None of the cases showed toxaemia which could be attributed to the enteritis or dehydration. The diarrhoea consisted of fairly frequent, relaxed green stools (average 7 in 24 hours). The average duration of illness was 10.7 days. Bacteriological examination of the faeces revealed

no abnormal organisms in twelve cases and B.Morgan No.1. in one non-fatal case.

The parenteral causes are listed below:-

Broncho-pneumonia	2
Pertussis	2
Otitis media	2
Acute Rhinitis	1
Tonsillitis	1
Thrush	1
Infected Scabies	1
Impetigo	1

Infection of operation site.

1.	Tonsillectomy	1
2.	Circumcision	1

The response varied slightly. The pertusis cases which were in the early stage both cleared up when the disease frankly declared itself. The cases with otitis media immediately settled with the onset of otorrhoea. These cases following operation improved with improvement in the local condition as did those with rhinitis, thrush, impetigo and infected scabies. As would be expected the diarrhoea in the two pneumonic cases was of the longest duration.

The conclusion reached from a study of these cases was that symptomatic enteritis is not an important complication of the disease

which produces it as it tends to follow the course of the original disease improving as it improves or persisting as it persists. The fatal case was obviously a pneumonia death and would no doubt have terminated in this way whether there had been diarrhoea or not.

I was fully aware that when collecting these figures I had laid myself open to the criticism that my opinion was based upon an insufficient number of cases. Realising that respiratory tract infection was regarded as the most important ex-enteral cause of enteritis, I analysed the cases of measles, a disease in which respiratory catarrh is prominent, which occurred in the 1937 epidemic. The object was to ascertain if enteritis was frequent in these cases and if it was of a mild or severe type when it did occur.

To simplify the analysis, only case reports of children up to a maximum age of 2 years were considered as that range would, in my opinion, include the majority of children susceptible to enteral upsets. The cases were divided into two groups of 0-1 years of age and 1-2 years of age. It will be seen from table 5 that the percentage of cases with complications is 21.8 in the first group and 26.2 in the second group, while the individual number of complications is 36 and 107 respectively. Details of complications are given in the same table. It will be noticed that the majority of these belong to the group which can properly be called parentral infections of the resperatory tract.

TABLE 5.

	0-1 years	1-2 years.
Total cases of ages	142	350
No. of cases having complications	31 (21.8%)	92 (26.2%)
No. of complications	36	107
Details of individual complications		
(1) Otitis media	19	54
(2) Ac. Bronchitis	3	8
(3) Br. Pneumonia	13	30
(4) Adenitis	-	5
(5) Tonsillitis	-	1
(6) Peroncolosis	-	2
(7) Stomatitis	-	1
(8) Abscess	-	5
(9) Encephalitis	1	1

Table 6 shows the cases which had diarrhoea at the onset of measles, during the course of the illness and at the onset and during the course. The number of cases which had diarrhoea in the initial catarrhal stage is fairly high namely 21 (14.8%) in the youngest group and 24 (6.8%) in the older children. The difference in the percentage incidence between the two age groups is very obvious and is what would be expected. The important fact, however, is the average duration of the diarrhoea, namely 2.8 days in the younger children and 1.8 days in the older children.

The course of the disease is taken to be from the second-tenth day. Those cases which developed diarrhoea during the course as above defined were 8 (5.7%) in the first group and 12 (3.4%) in the second group. The average duration of the diarrhoea once again was short, namely 3.9 and 4 days respectively. A few cases had diarrhoea at the onset continuing through the course. As will be seen from Table 6, the average duration was 18.5 days and 14 days. At this point it should be stressed that the last day of diarrhoea was estimated upon a strict basis, the appearance of the stools and the frequency of defecation being absolutely normal at this point.

TABLE 6.

Cases having diarrhoea	0-1 years	Average Duration	1-2 years	Average Duration
(a) At onset	21 (14.8%)	2.8 days	24 (6.8%)	1.8 days.
(b) During course	8 (5.7%)	3.9 days	12 (3.4%)	4 days.
(c) At onset and during Course	2 (1.4%)	18.5 days	2 (0.57%)	14 days.

Further analysis of the figures reveals the fact that in the youngest group only 8 cases (5.6%) and in the older group only 5 cases (1.4%) had diarrhoea associated with the onset of a complication That is to say in the younger group only 25.8% of the cases having complications developed diarrhoea directly associated with the onset of the complication, while in the older group only 5.4% of the complicated cases had diarrhoea in a similar association. Among

these cases are three deaths, two in the younger group and one in the older group. Details are shown in Table 7.

TABLE 7.

Age Groups.	Cases having diarrhoea associations with complications.	Complications with which diarrhoea was associated.	No.	Deaths.
0-1 years	8 (5.6%)	Broncho-pneumonia	5	2 (1 Br.Pn. 1 Otitis-Mastoiditis
		Otitis Media	2	
		Mastoiditis	1	Both with term. Enter.
1-2 years	5 (1.4%)	Broncho-pneumonia	3	1 (Tubercle and Br.Pn. with Terminal Enteritis.
		Otitis media	2	

The facts gathered from analysis of the measles cases along with the impressions formed from the study of the thirteen cases which have already been mentioned allow certain conclusions to be drawn which are on a firm clinical basis. These conclusions are as follows. Enteritis of the symptomatic type is a definite clinical entity but, as such, it is of little significance because in the great majority of cases it is mild and of short duration. It is rarely the cause of death which is determined by the ex-enteral condition and rarely by the enteritis. My previous statement that the diarrhoea subsides before the parenteral infection clears up appears to be amply justified, although in certain cases it tends to follow

the ups and downs of the parenteral infection. From this, a tentative suggestion may be made that treatment of the ex-enteral focus which results in cure will have a similar effect upon the diarrhoea. This would amply account for the successes claimed by supporters of the principle of active treatment such as paracentesis and antrostomy. How then can the argument of the other school of thought be justified? I believe, and I hope in later pages to amplify this point, that the conservative school are discussing the types which I have called the acute toxic and the acute choleraic types of enteritis and not the true symptomatic type. In these forms complications when present are often in the respiratory tract. The treatment of this form of enteritis by dealing with the ex-enteral focus is not satisfactory because the main cause of the illness does not lie in the ex-enteral focus at all. Thus I believe that the two entirely different schools of thought can be reconciled. The association of diarrhoea with otitis media can also be said to exist but the association is no closer than with any other ex-enteral focus of infection.

C H A P T E R II.

INFECTIVE ENTERITIS.

(a) Of known etiology. This title is meant to include all cases of enteritis from the stools of which known pathogenic organisms can be recovered or in which a serological examination reveals agglutination of a known pathogen in a diagnostic titre. The

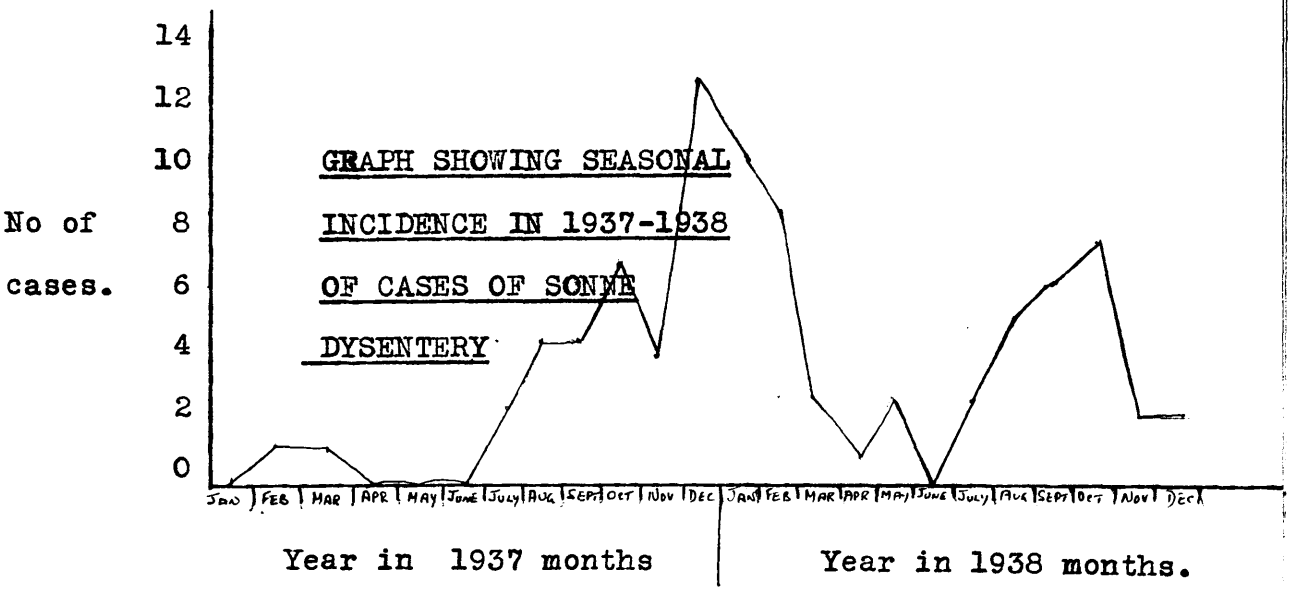
pathogens referred to are the typhoid and para-typhoid organisms, the dysenteric group of which the bacilli of Shiga, Flexner and Sonne are the best known, the complex and highly populated Salmonella group and the rare cause of enteritis, the staphylococcus. In my experience, the principal organism causing diarrhoea is the B. dysenteriae Sonne. In the series under discussion, out of a total of 89 cases of enteritis of known etiology 82 (92.1%) were attributable to this organism. For this reason I shall confine my remarks mainly to Sonne dysentery. Then I shall finally discuss the remaining seven cases.

There has been a marked increase in the number of cases of dysentery notified in England and Wales in the last ten years from 440 cases in 1927 to 1,333 cases in 1936. (8) This may be due partly to increasing interest as suggested by Felsen but it is becoming increasingly obvious that dysentery due to the organisms of Shiga, Flexner and Sonne is endemic in this country and no doubt the numbers will further increase as the value of serological examination becomes more widely recognised. (46).

SEASONAL INCIDENCE. It seems to be generally agreed that the incidence of dysentery is greatest in the late summer and early autumn and my figures for Sonne dysentery tend to bear this out (8). This is demonstrated in the graph below which shows the seasonal incidence of my cases. It will be noted that there is a definite drop in the numbers from April to July inclusive, in both 1937 and 1938. The incidence from August to October in each year (late summer and early autumn) shows a definite increase while the

winter months of 1937-1938 have a particularly marked increase. The figures for November and December of 1938 are not comparable with those of the previous year which may have been abnormal. It should be noted that none of the cases under discussion was involved in an institutional outbreak which might have accounted for these differences. The only deduction that can be made is that the months of April, May, June and July are the quietest so far as Sonne dysentery is concerned while there is an increase in incidence from August to October. In certain years of which 1937 may have been one, there may possibly be an increase in the case incidence in the winter months. The increase in cases in the winter of 1937-38 has not been an accidental one as it continues over a period of four months. Although the 1939 cases are not being discussed in this thesis I may say that the peak of December, January and February (1937-38) is not repeated in 1939.

TABLE 7.



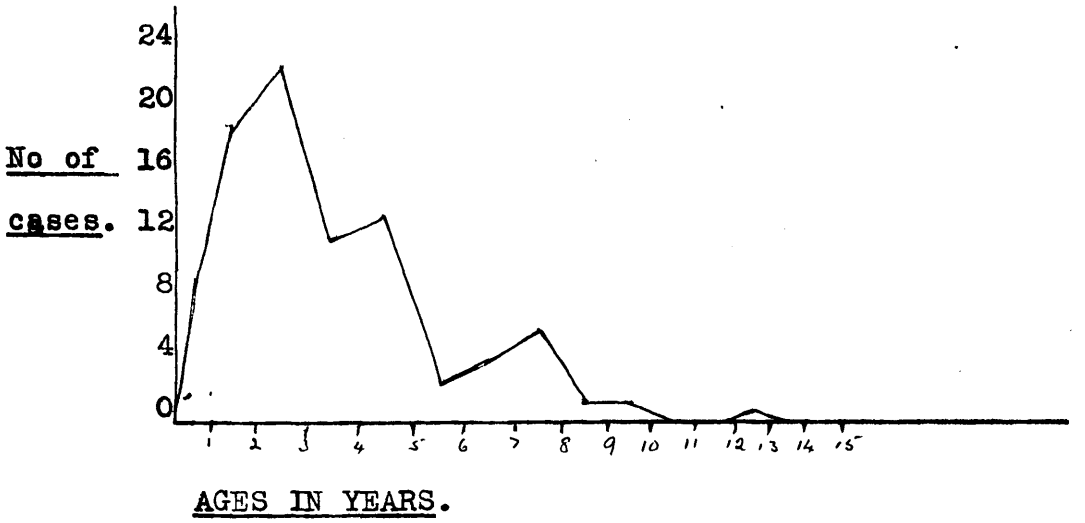
INCUBATION PERIOD. 3-7 days. This has been determined by the study of three ward outbreaks and in each the general outbreak occurred from three to seven days after introduction of the source of infection. This has always been an unrecognised mild case.

AGES ATTACKED.

The general impression formed from a study of the 82 cases of Sonne dysentery admitted in the two year period was that children under one year of age are relatively immune but this is not wholly true. Ward outbreaks have been fairly frequent at the Eastern Hospital during the years 1938-39 (August) and have been particularly troublesome in whooping cough wards where the age level is the lowest in the hospital. My opinion is that the age attacked is mainly influenced by the age of the particular population at risk. Thus in one outbreak in a whooping cough ward containing eleven children, of whom three were under one year of age, one of the three (eight months old) was subsequently shown to be the source of the outbreak while one of the remaining two (six months old) developed the disease. Out of a total of ten children exposed to infection six developed Sonne dysentery. A similar state of affairs was noted in other outbreaks. It would appear then that an infant is just as susceptible to infection with B. dysenteriae Sonne as any other person. It can therefore be stated that Sonne dysentery may attack persons of any age although it appears to be commonest among children (from the point of view of hospital admissions) from the ages of 1-5 years. The graph

below shows the age distribution of the cases under discussion. It must be remembered that this investigation has been confined to a group of children with an upper age limit of fifteen years so that the graph only covers this range of ages. Fyfe records a milk borne outbreak where 45% of cases were under 20 years of age. (53).

TABLE 8. AGE INCIDENCE OF CASES OF SONNE DYSENTERY 1937-38.



METHOD OF SPREAD. The dysenteries must take their place among the diseases spread by carriers or mild unsuspected cases. The route of transmission will therefore be most commonly water, milk or food contaminated by one of these carriers or mild cases. In each ward outbreak which I have investigated the source of infection has been an unsuspected mild case but recently I accidently discovered an adult carrier. The diagnosis of carriers will be dealt with later.

CLINICAL FEATURES. The onset of Sonne dysentery is abrupt. The statement is based upon observations during the ward epidemics which have already been mentioned. This opinion supports that of Frazer et al (51). Generally speaking, in my opinion, Sonne dysentery is only a moderately severe illness and is only of importance when it attacks a child already debilitated by some other illness and even then is often of little consequence. I have not seen the severe type described by Graham and mentioned by Frazer and his co-workers (8, 51). This experience is shared by my colleagues and where more than one case occurs in a ward we no longer isolate them. Even the degrees of severity vary greatly in a ward outbreak. The only sign may be the passage of one or two relaxed stools which may or may not contain mucus and/or blood and may simply be green and relaxed. This latter is especially common in infants and may be the only sign. The severity varies and in the milder forms the child has a slight pyrexia, some colic and passes 5-8 relaxed stools in 24 hours which, once again, may or may not contain blood and/or mucus. In these cases there is often a palpable and slightly tender colon. This tenderness is usually most marked in the right iliac fossa. The explanation of this, no doubt, lies in the fact that the lesions are most numerous in the ileum and upper colon. The spleen is not palpable. The diarrhoea in a case of this type rarely lasts for longer than 48 hours. In the most severe type which I have seen the child is lethargic and looks toxic with slightly sunken eyes and a circum-oral pallor. The temperature may be 100°F with an equivalent rise in the pulse and

respiration rates. The appetite is poor but thirst is considerable. The abdomen is slightly distended and tympanitic while the colon tenderness is more easily elicited than in the type which has just been described. This rather ill condition, however, is short-lived and by the third day after admission the child is sitting up and looking quite normal while the tympanicity and tenderness have also disappeared. It is in this type of case that the presence of blood and mucus is commonest and although these disappear in 2-4 days the stools may be relaxed and offensive for as many as 9-10 days. The stools are seldom more frequent than twelve in 24 hours and it is only rarely that faecal material is not present. In my experience vomiting never occurs as described by Moncrieff, neither have I seen the catarrh of the respiratory tract which Frazer and his co-workers state to be so frequent an accompaniment of the infection and my experience bears out that of Fyfe An occasional case develops a generalised papular urticaria in the early stages of the illness. This fact accounted for two of my cases being diagnosed as scarlet fever before admission to hospital. (51, 53, 119).

COMPLICATIONS. There have been no complications in my experience.

DIAGNOSIS. As may be imagined the ease of diagnosis varies with the severity of the case. Those which have been described as the severest are the easiest to diagnose while the mildest are almost impossible to diagnose on a clinical basis alone. It can

thus be seen that the notification figures do not in any way represent the true state of affairs because the infected person may by ambulant and may never come within the doctor's province (51, 116, 117). The presence of blood and mucus in the stools is stressed as being of great importance by certain writers (8, 46, 51, 119). I can agree with this opinion to a certain extent but it should be fully appreciated that blood and mucus are not present in the stools in Sonne dysentery only and are often absent in that disease. If, however, one can exclude such conditions as ulcerative colitis, tumours of the lower bowel and rectum, haemorrhoids and intussusception the presence then of blood and mucus in the stools is of definite value in diagnosis and it can be said with some accuracy that the patient is suffering from infective enteritis of known etiology. I have never seen blood and/or mucus in the stools of children suffering from enteritis of unknown etiology. To demonstrate the point mentioned above, I would emphasise the fact that of my 82 cases of Sonne dysentery only 38 (46.3%) had blood and mucus in the stools. While there can be no doubt that the diagnosis of Sonne dysentery has been rendered more accurate in consequence of the increase in the skill of the bacteriologist in isolating the Sonne organism since it was first discovered by Sonne, the bacteriological examination of the faeces in this disease is at the best an unreliable method. I did not fully appreciate this fact when I first began this investigation but when I was revising the case papers at the end of my first year's work upon it I noticed certain features which brought the point forcibly home to me.

The figures given below are for the two year period and they demonstrate the point even better than the figures for one year. Table 9 shows the original diagnosis of the 82 cases made before admission. It will be seen that only 32 cases (39%) were correctly diagnosed. If the term dysentery is accepted, then 53 cases (64.6%) were correctly diagnosed before admission. Twenty two of the 32 correct diagnoses were based upon the discovery of the Sonne organism in the stools, the remainder upon the presence of blood and mucus in the stools or from their association with known cases of sonne dysentery. If the 22 cases are excluded, it will be seen first how inaccurate clinical diagnosis can be. Thus of the remaining 60 cases only 11 (18%) were correctly diagnosed, an error of 82% which would appear to be excessive. The table 9 shows the designations given to these cases.

TABLE 9.

TABLE SHOWING ORIGINAL DIAGNOSIS OF 82 CASES OF SONNE DYSENTERY.

DIAGNOSIS	No. of cases
Sonne Dysentery	32
Dysentery	21
Enteritis	13
Gastro-Enteritis	13
Diptheria	1
Scarlet Fever	2
TOTAL	82

If we now consider the clinical findings aided by bacteriological investigation it will be seen that the position is very little better. Table 10 below is used to illustrate this point. It will be seen that of the 22 cases found to be positive before admission, not one was found to be positive after admission and that out of 82 cases thoroughly investigated after admission, only 54 were found to be positive, an error of 34.2%

TABLE 10.

STOOL	CULTURES	
Before admission	After admission	Number
+	-	22
Not investigated	+	54
Not investigated	-	6
	TOTAL	82

It might be argued that inefficient choice of specimens was at fault but this hardly holds good because each of the two examinations was carried out upon a rectal swab and a specimen of faeces. The above findings are of course easily explained because it is widely recognised that the Sonne organism soon disappears from the faeces (8, 51, 139). All this raised the question, what would have been the position of the twenty two cases which had been bacteriologically examined in the early stages of the disease if

this examination had not been possible? Obviously opinion would have had to be based upon the unreliable clinical method. After reading articles by Blacklock, Graham, Felsen and Ritchie (8, 46, 139) I decided that serological investigation of these cases would probably be a valuable addition. Investigations on this subject have been carried out for the last twelve months and although they are not yet complete, certain figures are available from which definite deductions may be drawn. The first point to determine was the diagnostic titre level. With this object in view the blood of 200 patients convalescent from various diseases was examined. The ages of these cases varied from six months to twenty-five years. Briefly the results were as follows. Only three of the 200 subjects showed agglutination with B. dysenteriae Sonne in a dilution of 1 in 20, the remaining 197 samples of serum failed to show agglutination even in a dilution of 1 in 10. This disposed of the idea that it was necessary to re-examine cases for a rising titre as any delution of over 1 in 20 could be taken as diagnostic. This agreed with Sears et al (46, 152). From serial testing it was quickly found also that it was useless to examine the serum before 14 days from the onset of the illness as the Sonne organism apparently produces agglutinins slowly, as suggested by Blacklock and Sears (8, 152). As this preliminary work necessarily took a long time it was unfortunately only possible to apply the principle to a few of the cases discussed in this paper. Of the six cases in which no organisms could be found in the stools (Table 10), four

were extremely mild and were diagnosed serologically. The other two were more ill and presented a typical clinical picture. Obviously a further point to investigate was whether agglutinins always appeared and although there is not a large number of cases from which to quote, it may be said that out of 30 cases investigated all but one gave agglutination in titres from 1 in 40 to 1 in 680. Another point which is being investigated is the length of time for which agglutinins remain in the blood. Once again, although the figures are small, principally through difficulty in persuading parents to bring their children back to a fever hospital, certain tentative deductions may be made. Eight cases have been so investigated, two after an interval of two months and six after an interval of five months from recovery from Sonne dysentery. The first two cases, whose serum agglutinated B. dysenteriae Sonne originally in dilutions of 1 in 40 and 1 in 160 respectively, now did so in dilutions of 1 in 20 and 1 in 10 respectively while the remaining 6 did not agglutinate. This disposed of Felsen's theory of the value of titre levels in assessing the infant's immunity to Sonne dysentery. He considered that lack of agglutinins could be taken to indicate susceptibility (46). In Sonne dysentery I take agglutination to a delution of over 1 in 20 to be diagnostic. Sears considers that over 1 in 40 should be the diagnostic level (152). It will be seen that serological examination of all cases with a clinical appearance such as has been described would be of considerable value. I now do a routine serological examination of all cases of enteritis whatever the clinical diagnosis. Although

so far I have failed to find an unsuspected case of Sonne dysentery among those which have been classified as enteritis of unknown etiology I found a few among those placed in the dyspeptic group in 1939. I should like again to emphasize the fact that the above remarks apply only to the diagnosis of Sonne dysentery. The serological diagnosis of the typhoid and para-typhoid groups is familiar to everyone, the difficulty of assessing the titre rise having been resolved by the separation of the "O" and "H" agglutinins. The position is difficult as regards the diagnosis of the Salmonella group and of the Shiga and Flexner infections because agglutinins may be present to a fairly high delution in the serum of persons who have not had an attack and who are apparently healthy (116, 152). Fortunately, in my experience, it has been quite easy to recover the aertrycke, Gaertner and Newport organisms from the stools of such cases. I cannot speak of the Shiga and Flexner organisms from this point of view as I have not seen any of these cases. However, during the serological work involved in the investigation of Sonne dysentery 59 normal children had a complete serological examination carried out with the following results.

1 case agglutinated B. typhosus "O" in a delution of 1 in 10

1 case agglutinated B. paratyphosus B in a delution of 1 in 20

5 cases (8.5% agglutinated B. enteritidis (Gaertner) in a delution of from 1 in 20 to 1 in 80

4 cases (6.8%) agglutinated Salmonella group suspension in a delution of 1 in 20

1 case agglutinated B. flexner (X and Z) in a delution of 1 in 20

5 cases (8.5%) agglutinated B. flexner (W. W. Y.) to a delution of from 1 in 20 to 1 in 40.

As all these cases were children it would appear obvious that the number of agglutinins would be even more varied and in stronger force in adult serum. Ritchie (139) considers that agglutination to a dilution over 1 in 128 for B. dysenteriae (Flexner), over 1 in 64 for B. dysenteriae (Shiga) and over 1 in 32 for the paratyphoid organisms to be diagnostic. The Salmonella group is so constantly being added to that an opinion as to diagnostic titre levels is hardly practicable, a very high or rising titre being required. Fortunately this trouble does not exist in connection with Sonne dysentery and in this disease serological diagnosis can be 98% correct for the reasons which I have already given.

CARRIERS. It is only natural that the question of carriers of B. dysenteriae Sonne should be considered as the ultimate source of an outbreak. From the investigations which I have carried out so far I considered it likely that a carrier might be defined as a person who might or might not have a history of a previous attack of Sonne dysentery depending upon the severity of that attack, whose serum did not show agglutination of the organisms above a dilution of 1 in 20 but from whose stools B. Dysenteriae Sonne could be recovered. I have only come across two cases so far which tended to bear out this theory. During the routine investigation of the stools of a woman of sixty-eight years of age who complained of occasional attacks of abdominal colic sometimes accompanied by diarrhoea, B. dysenteriae Sonne was recovered. Examination of the serum failed to reveal any agglutination of the organisms but she gave a typical history of an attack of Sonne dysentery two years

previously. The other case was a child who had been involved in an outbreak of enteritis in a children's ward of another hospital and in the course of a routine investigation of the stools of all the children in that ward, the Sonne organism was recovered from his stool. Once again the serological examination of the patient was negative but this time there was no history of any previous attack of diarrhoea. It is obvious that much more work must be done in this direction before any definite conclusions can be drawn.

ROUTINE METHOD OF DEALING WITH A WARD OUTBREAK OF SONNE DYSENTERY.

The method adopted at the Eastern Hospital on the occurrance of an outbreak of Sonne dysentery is as follows. The outbreak is usually explosive, several cases occurring at once. Originally these cases were at once removed from the ward and isolated, to be followed by any subsequent cases or by any children from whose stools the organism had been isolated. This routine has been changed as the result of experience which has shown the illness to be very mild in most cases and the infection to have already been spread. Now it is our custom to remove from the ward any child seriously ill from some other illness leaving the cases of Sonne dysentery there, each one on strict "barrier nursing" (each child has his own utensils, gowns, bedpans and the attending nurse scrubs up before and after giving attention to the child). Where only one case is reported on the first day this case is removed and isolated in the hope, usually a vain one, that no further cases will arise. The ward, of course, is closed to admissions until

14 days after the last case occurs or until all stools are negative on two successive occasions. Admittedly this method is not above criticism but isolation accommodation is limited in all fever hospitals and usually severely taxed and so far this routine has been extremely successful.

ROUTINE INVESTIGATION OF A WARD OUTBREAK.

Stools and rectal swabs are cultured from each child in the ward on two successive occasions unless the first specimens are found to grow B. dysenteriae Sonne. A certain percentage of even the obvious clinical cases have negative stools. Fourteen days from the beginning of the outbreak a serological examination of each child who was in the ward at the time of the outbreak is carried out. So far this has been the method of discovering the original sources of the outbreaks, their stool cultures being negative. At this point I cannot do better than describe the investigation of an outbreak of Sonne dysentery in a small whooping-cough ward. This ward contained 12 cots and 11 patients. On 27th February, 1939, a child of eight months with whooping couch was admitted. On 28th February this child passed two green relaxed stools. No other signs or symptoms were observed. On the fifth and sixth days after the introduction of this case into the ward a total of six children developed clinical signs of Sonne dysentery. On examination of stools from the eleven children four were found to be positive, all being cases which had presented typical clinical signs. The remaining seven specimens were negative including that of the child

introduced into the ward on 27th February. Fourteen days later serological investigation was carried out. In seven specimens agglutination was found to delutions from 1 in 10 to 1 in 80. One of these (1 in 10) from a child who had had no clinical upset was disregarded on the grounds that this titre level was below that which had already been decided to be diagnostic. Of the remaining six five were of children who had been clinical cases of Sonne dysentery and of whom two had had negative stools The sixth (1 in 40) was from the child who had been introduced into the ward on the 27th February and who now could be said to be the cause of the outbreak. It will be observed that one case of clinical Sonne dysentery with positive stools failed to show agglutinins. I have already mentioned this case in the discussion of diagnosis. I have not, so far, encountered a similar example of this contradictory state of affairs although, as I have already stated, thirty cases of Sonne dysentery have been investigated from this point of view. It will be seen that serological examination forms a necessary part of the investigation of a ward outbreak.

DIFFERENTIAL DIAGNOSIS. First of all, conditions such as intussusception, rectal and lower bowel tumours, haemorrhoids and ulcerative colitis must be excluded. When this has been done diagnosis must be made from the other members of the enteritis group. This point is adequately covered in this paper in later pages.

TREATMENT. This is entirely symptomatic, the children being put

on a light diet until the diarrhoea shows signs of abating. Ample fluids in the form of water, fruit and glucose drinks are given Powdered Kaolin has some effect in reducing the offensive odour of the stools but it is doubtful whether it has any effect in reducing their frequency. In the odd case where diarrhoea is really troublesome, starch and opium enemas are helpful.

PROGNOSIS. In my experience this is uniformly good as no deaths have occurred over a period of two years. In this opinion I am in agreement with Frazer et al (51) who consider that a fatal issue is likely only in exceptionally acute choleraic cases or in patients previously debilitated with other diseases. Fyfe also supports this view. Graham, however, reports a mortality of 22% with the qualification that only the severest types were admitted to his wards. (53, 60).

POST-MORTEM FINDINGS. As no deaths occurred in the series under consideration, I cannot quote from cases in the series. However in 1936 a child in a severely debilitated condition following an attack of severe enteritis of uncertain etiology, was involved in an outbreak of Sonne dysentery and subsequently died from the disease.

The post-mortem findings were quite typical. An acute colitis was present with small superficial ulceration.

The lower ileum was also involved in this process. The ulcers were clear cut and had not coalesced and their surface

was covered with a pale yellow exudate. No abnormality was found in any other organ. This picture fits in exactly with that described by Boyd and could readily account for the tenderness in the right iliac fossa which has already been described. A point of interest is that, although three stool specimens had been negative, the Sonne organism was isolated from a swab taken from the base of an ulcer. This confirms the view expressed by Blacklock (8).

Having fully discussed the disease caused by infection with the B. dysenteriae Sonne on the basis of 82 cases studied, I shall briefly notice the remaining 7 cases. These cases were as follows:-

Bacillus Aertrycke Infections = 5

Bacillus Enteritides (Gaertner) Infections = 1

Bacillus Newport Infections = 1

Clinically these cases showed strong resemblances. The onset was fairly acute with headache, abdominal pain and vomiting. Diarrhoea commenced within 48 hours of the onset of the illness and in the mild cases lasted for 3 days, in the severest for ten days. In two of the Aertrycke infections there was blood and mucus in the stools. Clinically the children initially appeared more toxic than those suffering from Sonne dysentery. In no case was abdominal tenderness present. Stool cultures were positive in the five Aertrycke infections, the Gaertner infection being diagnosed serologically on a rising titre. (From 1 in 2560 to 1 in 81,120 in 7 days) as the stools were negative. The Newport infection was diagnosed upon positive stool culture. It is obviously wrong tc form any opinion from such a small number but one deduction may correctly be drawn and that is that the Salmonella group of infections is

relatively rare as compared with the Sonne group. Diagnosis from the Sonne group is based upon the clinical features which show certain fairly well-marked differences. The onset is acute but there is a period of "seediness" of two or three days' duration before the onset of diarrhoea. The child is more ill than is usual in Sonne dysentery and the stools are less frequent although they may occasionally contain blood and mucus. The apparent ease of culturing the organism from the stools is of considerable value in diagnosis. Serological examinations would appear also to be of value. From my previous remarks it will be seen that out of 59 healthy children in whom a full Widal examination was carried out no case agglutinated the Aertrycke organism while only 5 agglutinated B. enteritides Gaertner to a maximum dilution of 1 in 80. Therefore it would appear likely that, as far as Aertrycke infections go, any agglutinins to a dilution of over 1 in 20 could be regarded as diagnostic. In the two cases which were investigated in this way the dilutions were 1 in 40 and 1 in 80 respectively. The fact that agglutination of B. enteritidis Gaertner to dilutions as high as 1 in 80 may appear in the serum of normal persons would not appear to offer any serious difficulty as apparently this organism is a rapid agglutinin producer, as witness the enormous dilution of 1 in 81, 120 noted in the single case of this infection.

Diagnosis is therefore possible if a routine similar to that recommended for Sonne dysenteryis followed.

TREATMENT. This is symptomatic and similar to that outlined for Sonne dysentery.

(b) INFECTIVE ENTERITIS OF UNCERTAIN ETIOLOGY.

(1) ACUTE TOXIC ENTERITIS.

(2) ACUTE CHOLERAIC ENTERITIS.

I have already stated that I believed the symptomatic variety of enteritis to be quite a different condition from the type to be discussed presently.

To recapitulate briefly I am in agreement with Findlay and Graham (47, 60,) in that I have never seen an acute toxic type of enteritis which could be directly associated with an ex-enteral focus of infection as the cause. I have seen otitis media associated with enteritis which cleared up when the middle ear infection cleared up. I have also seen otitis media occurring either as a complication of acute choleraic enteritis or as a part of a syndrome, namely acute toxic enteritis. This can easily and naturally explain the differences of opinion which exist regarding the treatment of these parenteral foci. Otitis has engaged most attention and as a result most has been written about its association with enteritis. It is obvious that treatment of otitis which has given rise to enteritis will result in improvement or lessening of the diarrhoea. But it will have no effect in improving the initial condition where otitis is either a complication or part of the disease. In fact radical treatment may have a bad effect upon the seriously ill patient where expectant treatment would be best.

So far as the division into two distinct types is

concerned, this is done upon clinical and pathological grounds as will be found in succeeding pages. Graham and M'Kinnon suggest that the two types are simply different stages of the same disease but in my experience the two entities breed true. In ward outbreaks due to the introduction of an infectious case of a specific type the secondary cases always are of the same specific type. Another point in favour of this theory is the tendency for a child in the convalescent stage of one variety of enteritis to be re-infected with the other variety. This must have occurred in the experience of everyone who has had any experience of children's wards. In most cases, however, the re-infection is called a relapse. (8, 115).

INFECTIVITY OF BOTH TYPES.

It has often been stated that enteritis is infectious but the extreme degree of this infectiousness does not seem to have been fully appreciated until lately. Rice et al, Frant and Greenberg et al have strongly advocated stringent isolation in their papers upon acute enteritis of the new-born. (8, 43, 50, 60, 61, 104). From personal experience of ward outbreaks, I can state that the infectivity of the types of enteritis now under discussion is very high. This applies particularly to the acute toxic variety and I think several reasons for this may be advanced. They will be discussed later.

Although the dyspeptic, mechanical and symptomatic forms of enteritis are not infective the differential diagnosis of those kinds of enteritis from the infective forms is not usually one which

can be carried out under 48 hours; no diarrhoeic case therefore should be left in an open ward.

(1) ACUTE TOXIC ENTERITIS. No = 32.

This is the most deadly form of enteritis among children and has a mortality of 53.1% in this series.

INCUBATION PERIOD. Two to four days when based upon experience of ward outbreaks.

ETIOLOGY. From purely clinical observation it has not been possible to throw any definite light upon the etiology of this condition but inferences can be drawn which point to investigations which might profitably be carried out.

INFECTIVITY. This disease is exceptionally infectious and the attack rate very high, especially among children under one year. In 1938, there was an outbreak of this variety of enteritis in a ward in the Eastern Hospital containing twenty cases of measles, seven of whom were under one year. Five of the seven children were attacked with four deaths, while only one case occurred among the children of over one year.

MODE OF SPREAD. While infection may be carried on the person and the clothes, routes which are easily controlled, it appears that the most important method of spread is by spray or droplet. When the special ward was opened, the greatest care was taken to ensure that the entire staff was free from any resperatory infections especially

those of the upper respiratory tract. Nurses had strict instructions to report any abnormality of this nature. No masks were worn, the remainder of the precautions being directed against infection being carried on the hands or uniform. Very soon after opening the ward a case convalescing from a mild dyspeptic form of enteritis developed the acute toxic type of enteritis and subsequently died, two further cases occurring shortly after this. As the prescribed precautions were being rigorously applied it was decided to try the effect of masking the nurses who looked after the children and who prepared the feeds. Since then no further cases of cross infection have occurred. It would appear on the face of it that infection may be carried in the respiratory tract of apparently normal adults and from these may be conveyed either direct to the children or to their feeds. From the general picture the former seems the likelier mode of spread.

BACTERIOLOGY.

In six of the cases B. proteus was isolated from the stools and in three B. Morgan No.1. As in my experience both organisms can be equally well isolated from the stools of children long since recovered from any form of enteritis and as secondary cases seldom yield the same organisms, I do not feel disposed to regard them as being of any significance except perhaps in the case of B. protens which is a common concomitant of putrefaction. B. Morgan No. 1. is fairly often recovered from the stools of children convalescent

from Sonne dysentery. As already indicated no serological investigation of these cases was carried out until the last six months of 1938. However in twelve cases subsequently investigated serologically no agglutination of any known pathogen could be found. In one case from which B. proteus had been isolated the serum failed to agglutinate B. proteus. It seems probable that the disease is caused either by some unknown bacillus, by a filter passing virus or by a toxin produced by an unsuspected organism. The extremely high degree of infectivity, and the great difficulty in preventing cross-infection resemble the behaviour of measles, chicken-pox and small-pox very closely and these are all diseases caused by filter passers. That is however a personal opinion based upon undoubtedly very slight evidence and must necessarily await more definite confirmation.

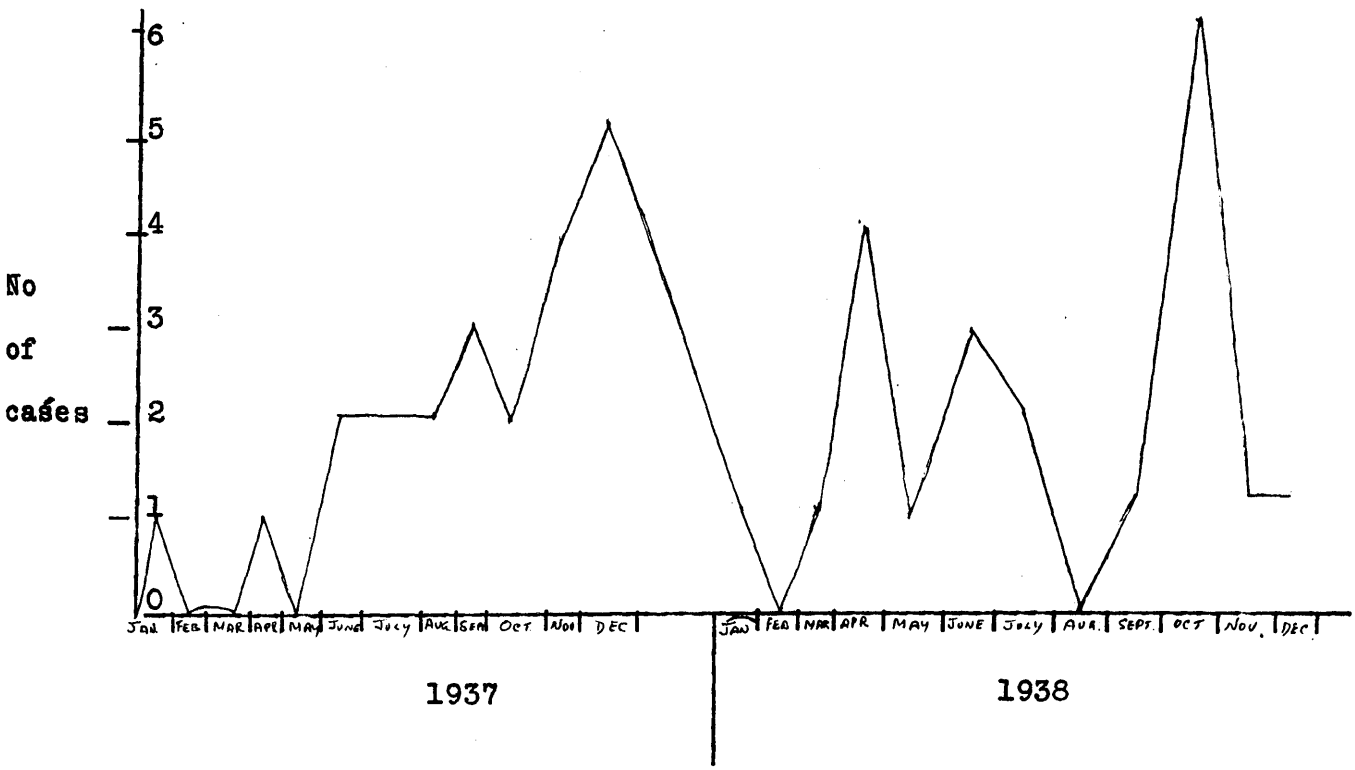
SEASONAL INCIDENCE. The numbers are small but probably sufficient to show that this type of enteritis tends to occur irregularly throughout the year (Table 11). Peaks are most obvious in the late autumn and winter. These peaks occur in both winters, the summer figures are quite irregular and there is little resemblance between those of 1937 and 1938. It would be unwise to conclude more than that the disease occurs irregularly throughout the year with peaks in the late autumn and winter months. The clinical impression was that these cases were relatively infrequent during July, August and September and became more common in the autumn and winter. This impression is still borne out by events in 1939. At the moment of writing (August) there is no case of acute toxic enteritis in the

hospital while there are many cases of bacillary dysentery.

TABLE II.

TABLE SHOWING SEASONIAL INCIDENCE.

OF ACUTE TOXIC ENTERITIS.



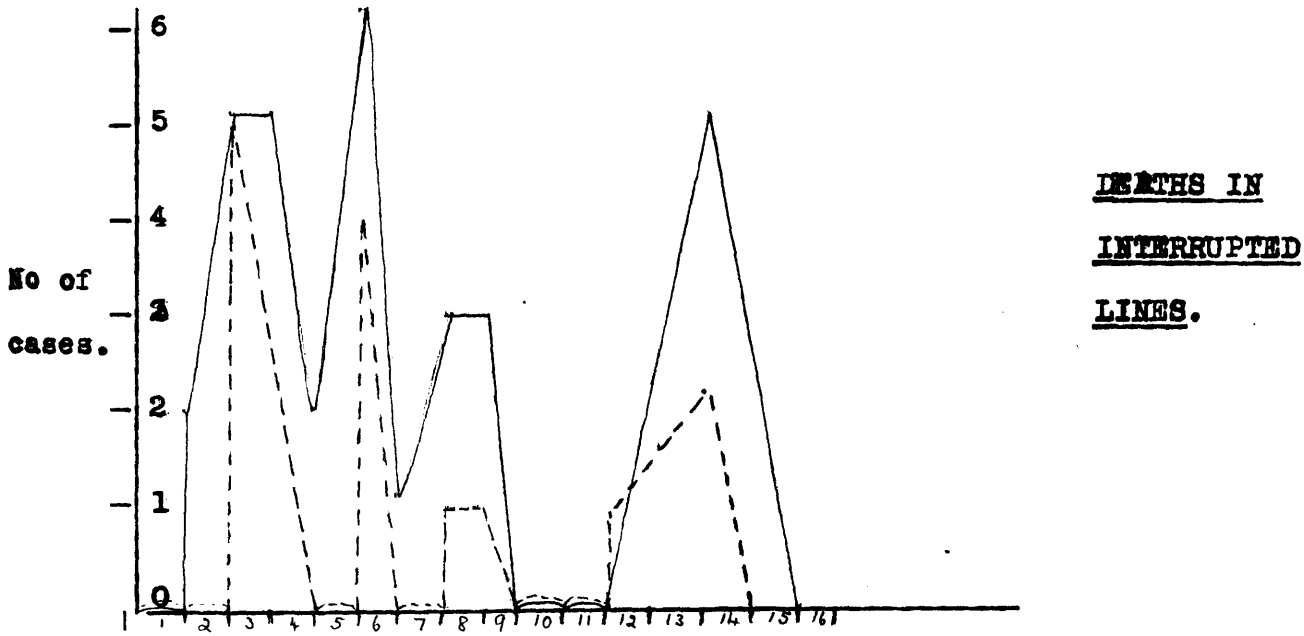
AGE INCIDENCE. Almost two-thirds of the cases occurred among infants of from six weeks to six months old (62.5%). Five cases occurred among children between twelve and fifteen months old.

From Table 12 it will be seen that the greatest attack rate is between the ages of one and a half months and six and a half months with two peaks at 2-4 and 4-6 months. There is also a peak between 12 and 15 months. No cases occurred over the age of 15 months and no cases between birth and six weeks of age. It can be concluded from this that children in the neo-natal period and of over 15 months

are relatively immune. This point will be discussed later under the heading of immunity.

TABLE 12.

TABLE SHOWING AGE INCIDENCE AND DEATHS AT AGES OF ACUTE TOXIC ENTERITIS.



SEX DISTRIBUTION. This shows no particular selection as 16 cases of each sex occurred.

CLINICAL PICTURE. The onset of the disease is sudden in all cases whether moderate or severe. The child usually first passes one relaxed stool and does not appear unduly ill. Within a few hours the temperature rises slightly, the child developes a slight, short

unproductive cough which is one of the typical signs of this form of enteritis. The stools become more frequent, more watery and green, and vomiting occurs whenever feeding is attempted. The stools rarely more frequent than twelve in 24 hours and the water content is high but not excessive. In the late stages of a fatal case stools are frequently orange in colour. As the disease progresses into the third day dehydration becomes obvious, the urine becomes scanty, concentrated, and may or may not contain a trace of albumen. The eyes are dull and sunken and the skin inelastic. But overshadowing all this is the clinical picture of toxaemia. The child is lethargic, the expression, which in my opinion is typical, is one of fear in those cases which ultimately prove fatal. The skin becomes pale and has a yellowish tinge. The temperature which may fall in 48 hours after the onset of the illness often rises again on the fifth to seventh day. This is a bad prognostic sign. The pulse which for the first 24-48 hours is bounding becomes steadily more frequent and feebler until on the seventh to tenth day it is thread-like and uncountable. The cough which was present at the onset of the illness becomes more frequent and moister but is unproductive. Auscultation of the chest reveals harsh breath sounds and terminally a few moist sounds at the bases. The ears which are normal in the first 4-5 days of the illness may show dulness of the drumheads but never infection or bulging. I have never seen a typical acute otitis media in one of these cases. On the 7th to 10th day the child may quite suddenly stop vomiting, the stools slowly return to normal and the toxic features disappear,

within three days. In my series of 32 cases the average duration of illness was 14.4 days. That is to say 14.4 days until the child was thought to be out of danger. However in a distressingly large number of these cases the illness progresses to a fatal conclusion with terminal hyperpyrexia and broncho-pneumonia.

In my series this occurred on the average on the 7-8th day. However since commencing this thesis I have seen one case which died in a convulsion on the second day. This child (ten months old) had passed two relaxed stools at home and was sent in as a case of enteritis. On the day after admission the child appeared lethargic and dull. Vomiting had occurred only once and the bowels had not opened since admission. 36 hours after admission the child had a severe convulsion and died within five minutes. Post mortem examination showed the signs which we have learned to associate with acute toxic enteritis and which will be described in the section devoted to post mortem findings. Bratton tells me that he has recently carried out post mortem examinations on two similar cases. (9) Then, in my experience, acute toxic enteritis follows three main courses. These are sudden death in a convulsion in the first day or two, death from toxaemia in 5-10 days or recovery in 15 days.

COMPLICATIONS. In three cases which died two had a definite terminal broncho-pneumonia and the third developed lobar pneumonia (Group IV pneumococcus). In all cases observed there was dulness of the membrana tympani in the later stages of the illness but no infection or bulging and the ears did not discharge on any occasion.

I do not tend to regard the changes in the drumhead as being complications but rather as being a part of the whole condition which to my mind is not only enteral but a general infection with the respiratory and alimentary tracts principally involved. In no case was there any evidence of urinary infection.

DIAGNOSIS. Bacteriological and serological examinations reveal nothing of significance. This point has already been dealt with in the discussion on etiology. The further points in diagnosis are brought up in the discussion of both types which follows later.

TREATMENT. As the general treatment of both types is very similar it is most conveniently dealt with later. The undoubted toxaemia in this type of enteritis is the great obstacle to treatment. Acting on the assumption that a degree of immunity is possessed by adults and that it can be passed on to the infant in the mother's milk, I have given 20 c.c. doses of maternal whole blood intramuscularly. I have observed no useful result from this form of treatment. I have not yet tried either serum from the mother or from recovered cases of this type of enteritis. There is considerable difficulty in persuading parents that their baby's blood can be usefully employed in treating other infants. I have not been able to find any reports upon the effects of this form of treatment which in my view forms the only possible hope until the etiology of the condition is rendered clearer. It is possible, however, that as in measles, such serum will not be of value in the active treatment and may only be of use as a prophylactic. A very significant point is that only two of my

cases were entirely breast fed. These children were breast fed throughout the illness and both survived. This question will be raised again in the general discussion on treatment of both types. Christie informs me that he has used the Campolon brand of liver extract in a few of these cases and he considers that there may be a slight improvement following this. The basis if this treatment is the theory advanced by Parsons that in toxaemia there is a defective assimilation of glucose due to lack of "Insulin Kinase" an enzyme produced by the liver. Parsons states that this enzyme is present in the less refined liver extracts such as campolon. This form of treatment was not tried in any of the cases in this series. (24)

PROGNOSIS. Table 13 has been drawn up to show the important points in prognosis and also includes certain other facts which have already been mentioned, In assessing the chances of recovery certain points are of impottance.

GENERAL CONDITION WHEN FIRST SEEN.

This proves to be of value in prognosis as it will be seen that of those cases assessed as "fair" on admission 63.6% recovered while of those assessed as "poor" only 10% recovered. This bears out the experience of Findlay (28).

WEIGHT ON ADMISSION. It will be noticed that only sixteen cases are quoted. This is because the weighing of these children was

discontinued on the grounds that too much manipulation was involved. However it will be seen that children below weight on admission are the worst risk.

PYREXIA. Pyrexia in the initial stages alone is of little prognosis significance but if it occurs late the prognosis is rendered worse. 22 (67.5%) of the cases in this series had pyrexia in the early stages. 13 cases (40.6%) had pyrexia on the 5th-7th day and all died. In other words 76.4% of the cases dying had late pyrexia.

VOMITING. This does not appear to be of much significance from the point of view of prognosis as all with the exception of one case vomited.

EARLY DEHYDRATION. This is of considerable importance in prognosis. It will be seen that 58.8% of the cases dying were showing signs of dehydration on admission. This was not because these cases were admitted late, for the average number of days ill before admission of the cases dying was 2.8.days while the corresponding average for those recovering was 3.3 days. In other words the early appearance of dehydration is of grave significance.

TABLE 13. GENERAL FACTS REGARDING 32 CASES OF ACUTE TOXIC ENTERITIS.

	Cases RECOVERING (15)	Cases DYING (17)
HISTORY OF PREV.ATTACK.	-	-
FEEDING.		
(a) BREAST	2	-
(b) COMBINED	1	-
(c) ARTIFICIAL	12	17
WEIGHT ON ADMISSION (16 cases)		
(1) ABOVE EXPECTED WEIGHT	7	2
(2) AT EXPECTED WEIGHT.	2	1
(3) BELOW EXPECTED WEIGHT.	1	3
GENERAL CONDITION ON ADMISSION		
(1) FAIR	14 (63.6%)	8 (36.4%)
(2) POOR	1 (10%)	9 (90%)
ONSET - ACUTE.	15	17
PYREXIA		
(1) EARLY	9	-
(2) EARLY AND LATE.	-	13
VOMITING.	14	17
DEHYDRATION ON ADMISSION	2	10
AVERAGE DAYS ILL	17	7.8

AGE. I have not seen a case of acute toxic enteritis in any child over the age of 15 months. 8 cases (25% of total cases or 47% of the total deaths) occurred in children between 2 and 4 months old. 50% of cases between 5 and 9 months old and 60% of cases between 9 and 15 months died. Table 12 has been arranged to show the relationship between cases at ages and deaths at ages. To summarise, the worst risks are the children below four months, (with the exception of those in the first six weeks of life) who show poor general condition, with some early dehydration and who develop a pyrexia subsequently on the fifth to the seventh day. There will obviously always be the chance of being "caught out" by the occasional case which dies in a convulsion in the first 48 hours of the illness. The mortality in this series of cases was 53.1%, surely a shocking figure.

POST MORTEM EXAMINATION FINDINGS. Permission for post mortem examination was only obtained in six of the seventeen cases which terminated fatally. However a certain amount of information was obtained from these examinations and is set out below:-

GENERAL APPEARANCES. In all cases the body showed evidence of dehydration and loss of subcutaneous fat.

HEART. Cloudy swelling of the myocardium. In one case there were subpericardial petechial haemorrhages. No valvular lesions were noted.

LUNGS. In four cases there was general congestion while in other two there was evidence of broncho-pneumonia. I have already mentioned these two fatal cases.

PLEURAE. With the exception of the two pneumonic cases the pleura was normal.

LIVER. Congestion, enlargement and fatty change which are usually obvious to the naked eye.

SPLEEN. Slight enlargement and congestion were present in all cases.

KIDNEYS. Congestion and cloudy swelling. No evidence of any pyelitis in any case.

SUPRARENALS. The cortex was pale and thinner than normal in all cases. Microscopic congestion was found.

STOMACH. The stomach showed marked mucous catarrh in all cases.

INTESTINES. The ileum showed the most characteristic changes - giving the general impression of a piece of flannel. Peyer's patches were hypertrophied and pink. Microscopically there was an acute inflammatory infiltration of the mucosa of the ileum. The

bowel content was scanty, green and offensive. Ulceration was only present in one case and was of the pinhead variety and confined to the ileum. In two cases there was an acute inflammatory infiltration of the mucosa and submucosa of this colon.

GENERAL NERVOUS SYSTEM. The gray matter was pink, the brain oedematous and congested.

MIDDLE EARS. Sero-purulent fluid was only found in one case at post-mortem and in this case there was no evidence of any antral involvement.

GENERAL POINTS. The post-mortem picture is primarily that of toxaemia as exemplified by the changes in the liver, kidneys and heart associated with an acute inflammation of the respiratory and gastro-intestinal tracts with the most typical changes in the latter. It is mainly on such pathological findings together with clinical findings that I base my opinion that the illness is a general one and not localised only to the intestimal tract. It is difficult to assess where the infection alights in the first instance but it may well be in the upper respiratory tract as is the case in influenza.

ACUTE CHOLERAIC ENTERITIS. TOTAL NUMBER = 77.

This is a commoner and less deadly form of enteritis. The mortality rate in the present series was 12.9% The incubation period may be put at 5-8 days. This is considerably longer than

the incubation period of the acute toxic type and is another point in favour of the suggestion that they are two different clinical entities.

ETIOLOGY. Once again it has been impossible to throw any definite light upon the etiology of this form of enteritis but it has been possible to form certain broad conclusions from the behaviour of the disease. Spread of the choleraic type of enteritis is not so difficult to control as that of the acute toxic type. In my opinion this is because the infecting organism is situated in the alimentary tract and is carried from one person to the other in dust or food, and on the hands or persons of individuals in attendance upon the patient. In short, infection is conveyed to others by the same routes as the infection of typhoid fever, the dysenteries and other allied diseases. It is well known that a case of typhoid fever can safely be nursed in an open ward because ordinary methods of barrier nursing are sufficient to prevent spread. I believe that there is the reason for the comparative ease with which spread of the choleraic type of enteritis is prevented. As an added point in favour of the method of spread which has been suggested, it may be said that no case of cross infection of this type occurred in the special ward even before masks were worn by the nursing staff.

BACTERIOLOGY. In eighteen cases B. proteus was isolated from the faeces and B. Morgan No. 1 in three. No abnormal organisms could be found in the faeces of the remaining fifty-six cases. As I have

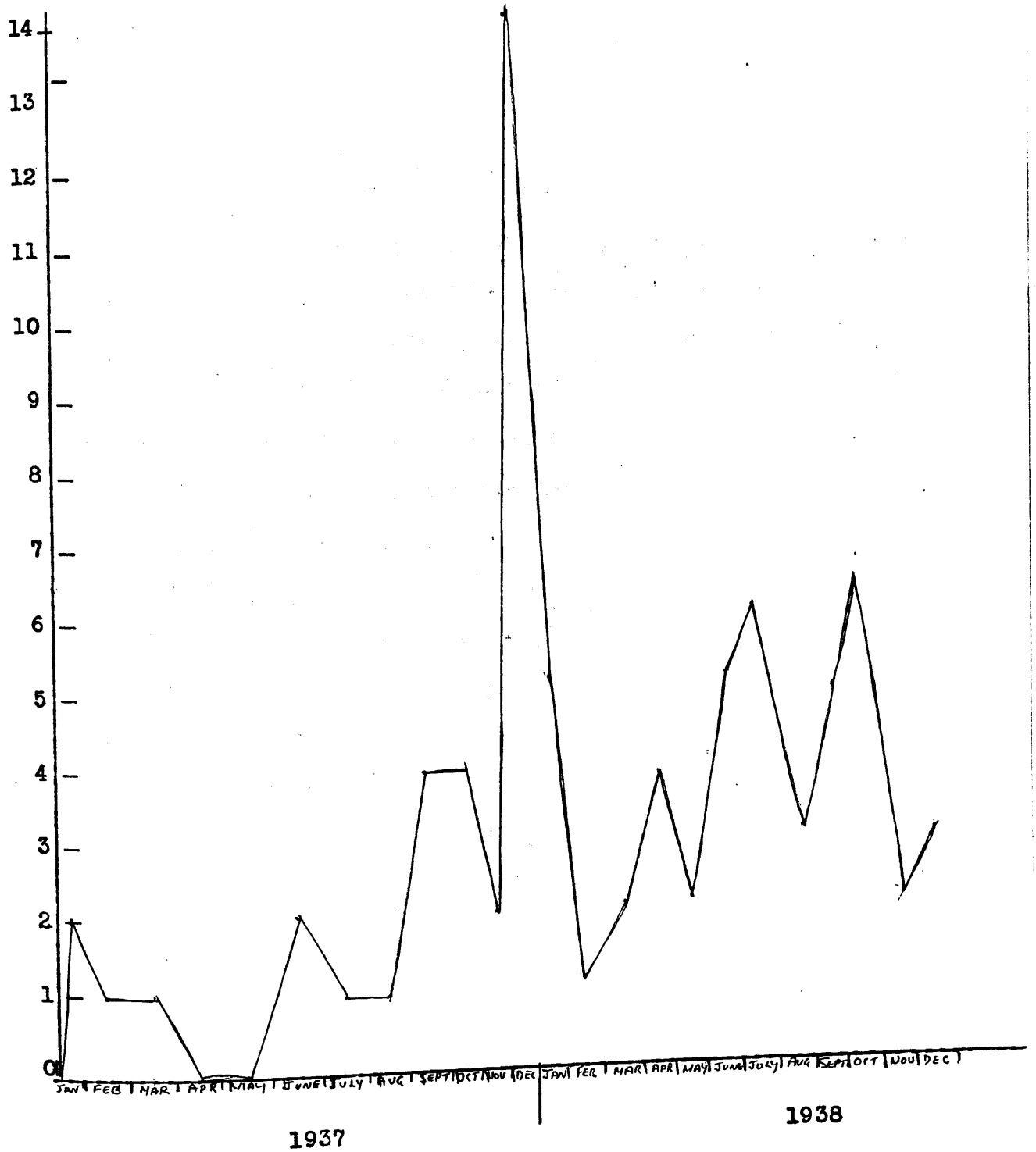
already stated I do not consider that these organisms are of any significance as an etiological factor. Serological examination in ten cases failed to reveal agglutination of any known pathogenic organism. To my mind it seems that the behaviour of this type of enteritis presents many features similar to those of the bacillary types. In the discussion of the clinical and pathological aspects it will be seen that the site of infection is the bowel and that the signs and symptoms bear this out. All the above points seem to indicate an infection which is introduced into the alimentary tract through the mouth just as is the case in the enteric and dysenteric diseases. All these facts to my mind point to a bacillary cause.

SEASONABLE INCIDENCE. TABLE 14.

Once again the numbers are small but certain broad deductions can be drawn from the graph below. The cases are least common in February, March, April and May of the two years under discussion with a peak period towards the autumn and winter months. It falls to be noted that a similar rise in the winter months occurs in the dysenteries.

TABLE 14.

TABLE SHOWING SEASONIAL INCIDENCE OF ACUTE CHOLERAIC ENTERITIS



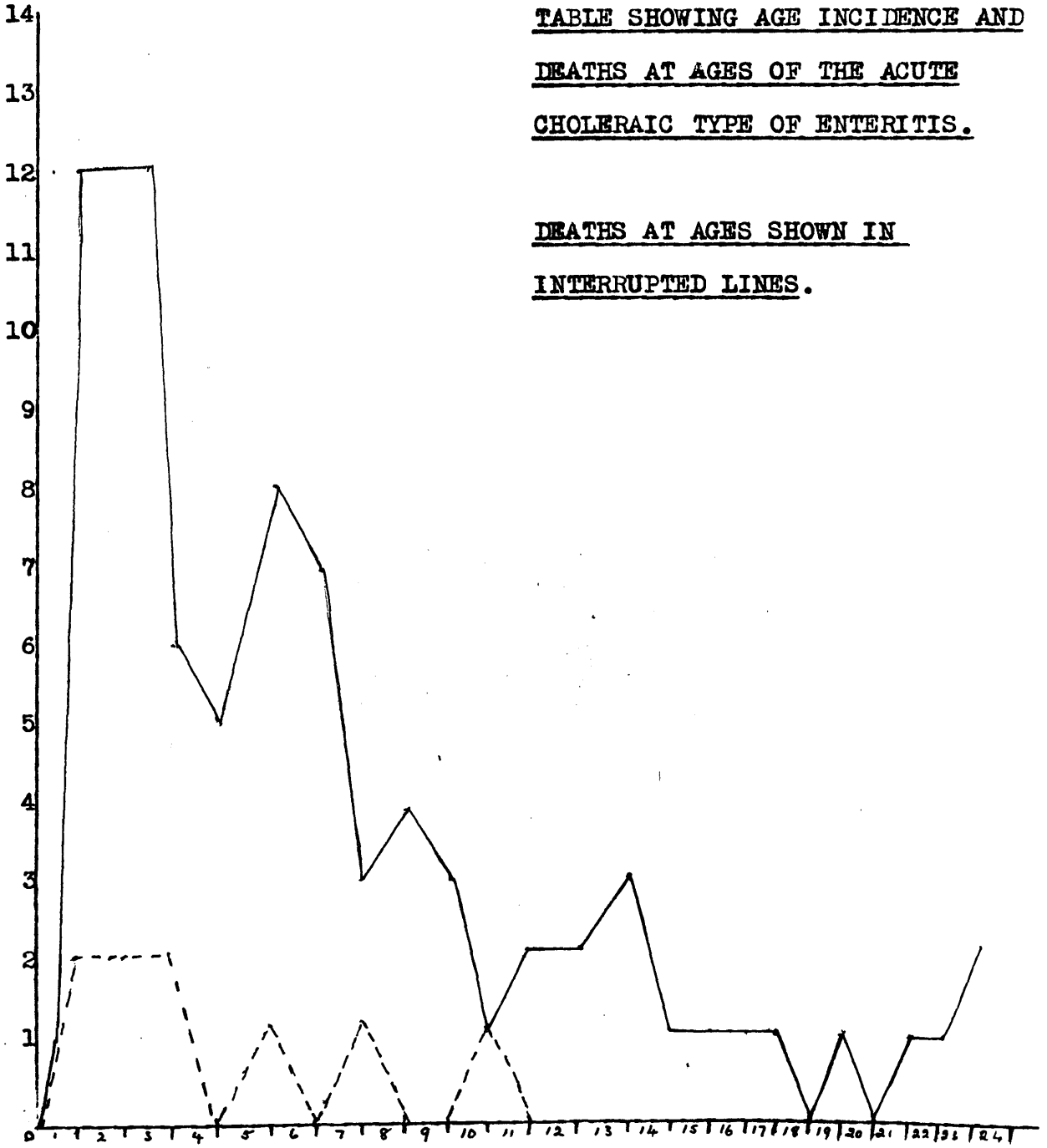
AGE INCIDENCE. Once again the disease falls heavily upon infants of from 1½ to 6 months (55.8%). It will be seen that the age limit is somewhat higher than is the case in acute toxic enteritis. In the present series the oldest child affected was two years old but there was a fairly even incidence of cases throughout the second year. In all fourteen cases occurred in the second year of life. (18.2%)

Table 15 shows these points in graph form. The neonatal period is relatively immune, only one case occurring in the first month of life. The sharp rise in incidence after the first month is particularly obvious and also the steady drop to the age of ten months after which the incidence remains more or less constant. There is no sudden rise in the incidence of cases after the first year of life.

TABLE 15.

TABLE SHOWING AGE INCIDENCE AND DEATHS AT AGES OF THE ACUTE CHOLERAIC TYPE OF ENTERITIS.

DEATHS AT AGES SHOWN IN INTERRUPTED LINES.



SEX DISTRIBUTION. This showed no significant difference, there being 39 males and 38 females in the present series.

CLINICAL PICTURE. The onset of the illness is gradual, the first sign commonly being refusal to take food or occasional vomiting, usually after feeding. This is associated with the passage of one or two green relaxed stools. A toxic erythema occasionally occurs initially. The temperature is usually normal at this stage of the illness. In the fatal cases the condition slowly becomes worse, the stools becoming more frequent, watery and green. Vomiting is not often more frequent than once or twice a day and the diarrhoea is the most prominent feature. A slow dehydiation occurs with the usual signs of dry inelastic skin, sunken, dull eyes and sunken anterior fontanelle in the infant. At this stage the pulse is soft and frequent and the urine is concentrated, scanty and may contain a trace of albumen. In the case which does not respond to treatment, the dehydration becomes gross until finally the infant presents the picture of a little shrivelled monkey-like creature becoming unconscious and dying. In the last few days the temperature is often elevated, as with the onset the course of this type of enteritis is gradual and death occurs at the end pf the third week of illness. In this series the average day of death was the 20th-23rd day, death in some cases being definitely due to a superadded complication such as broncho-pneumonia. Recovery is slow and it is often impossible to pronounce the child out of danger until the fifth week of illness. In this series the average time was

37.7. days. The course of the disease is notable for the liability of the children to relapses and in every case which recovered, there was at least one occasion, usually in the third week, when the child appeared about to become as ill as on admission. It appears, however, that, if these cases are recognised early enough and suitably treated, the progress of the illness can be arrested. This gives point to the argument in favour of immediate treatment of all suspected cases without waiting for the typical clinical picture to develop.

A general survey of the illness then shows a slow course with diarrhoea, and accompanying dehydration as the prominent clinical features. There is no toxaemia as in the acute toxic type and all the signs point to an upset of the lower part of the alimentary tract. This being the case, one would expect a fair response to symptomatic treatment and this expectation is justified in the results. In this series the number of deaths was 10 (12.9%)

The question of the chemistry and cellular changes in dehydration will be dealt with fully in the discussion of the treatment of both types of enteritis.

COMPLICATIONS. When I originally encountered this form of enteritis, a large proportion of the cases developed complications such as pyelitis, otitis media and broncho-pheumonia. I believed at that time that these were due to secondary infecting organisms. It is well known that the complications of measles are due in the

majority of cases to invasion of the subject by organism which are introduced into the ward either by other patients or by the medical or nursing staff. This idea is also advanced by Mitchell (118) and should hold good for enteritis as well as measles. If this theory is correct, one would expect to have a reduced incidence of complications in cases nursed in separate rooms. This has proved to be the case because in this series of 77 cases, all nursed in separate rooms, the number of complications was remarkably small. Three cases developed broncho-pneumonia and two of these died, death undoubtedly being hastened by the complication. Two cases developed acute otitis media, one with spontaneous rupture of the drumhead and subsequent recovery without operative interference. The other case which developed otitis media two days before death was not interfered with. Although there was slight marginal infection no bulging was observed. No case of urinary infection occurred. It is thus seen that isolation in separate rooms serves several purposes. It prevents children with different forms of enteritis from being herded together with consequent risk of cross infection. It also prevents secondary infection to some extent.

DIAGNOSIS. Bacteriological and serological examinations reveal nothing of any significance. The further points in diagnosis will be discussed in the section devoted to differential diagnosis of the different members of the different forms of enteritis.

PROGNOSIS. Table 16 has been drawn up to emphasise points in prognosis. These poimts are similar to those already mentioned in connection with acute toxic enteritis.

GENERAL CONDITION ON ADMISSION. It will be seen that the general condition on admission gives a fair indication of the course which the disease will follow but, of necessity, as the disease is considerably less lethal than the acute toxic type, the indication is not so dramatic. Of cases classified as "fair" on admission 46 (92%) recovered while of those classified as "poor" 21 (77.7%) recovered.

WEIGHT ON ADMISSION. The majority of cases are in the "at expected weight" category. Weight does not appear to bear any very definite relationship to fatality.

PYREXIA. It will be noted that 9 (90%) of cases dying were pyrexial in the late stages of the illness while 21 (31.3%) only of those recovering had a late pyrexia.

VOMITING. As already stated this was infrequent in all cases and actually occurred in 62 cases. It is of little significance as a prognostic sign.

DEHYDRATION ON ADMISSION. This is of considerable importance as is indicated by Table 16. 6 (60%) of the cases dying had

dehydration on admission while only 17 (25.3%) of those recovering were dehydrated on admission. To some extent this is an indication, in the acute choleraic type of enteritis, of the severity of the illness as the average duration of illness before admission of cases dying was 2.7 days while of those recovering it was 4.7 days. In other words the earlier the dehydration the graver the prognosis.

AGE. (See Table 15)

Once again the heaviest mortality and incidence falls upon the youngest infants. 30 cases (38.9%) occurred between the ages of one and four months. Of this number death occurred in 6 cases (20% of the number at age 1-4 months or 7.8% of total cases). In the age group 5-9 months of 22 cases, 2 died (9.1% of the number at age 5-9 months or 2.6% of total cases). Between 9 and 24 months of age 19 cases occurred and of these one case died (5.2% of cases at age 9-24 months or 1.3% of total cases). It can therefore be seen that the prognosis is worst at the ages 1-4 months and becomes increasingly better as the age of the infant increases. Age, therefore, is a valuable aid in prognosis; probably the most reliable. Table 15 is arranged to show the relationship between age and incidence and age and mortality. The marked difference between this relationship in the acute toxic type of enteritis and that in the acute choleraic type is easily seen on comparison of Table 15 with Table 12.

TABLE 16

GENERAL FACTS REGARDING 77 CASES OF THE CHOLERAIC TYPE OF ENTERITIS.

	CASES RECOVERING	CASES DYING.
TOTAL	67	10
HISTORY OF PREVIOUS ATTACK	6	-
FEEDING.		
(a) BREAST	1	-
(b) COMBINED	1	-
(c) ARTIFICIAL	65 (97%)	10 (100%)
WEIGHT ON ADMISSION (52 CASES)		
(1) ABOVE EXPECTED WEIGHT	12 } 46 cases	1 } Only
(2) AT EXPECTED WEIGHT	20 } weighed	4 } Six cases
(3) BELOW EXPECTED WEIGHT	14 } only	1 } weighed.
GENERAL CONDITION ON ADMISSION		
(1) FAIR	46 (92%)	4 (8%)
(2) POOR	21 (77.7%)	6 (22.3%)
ONSET (a) SEVERE	54	9
(b) MILD.	13	1
PYREXIA.		
(1) EARLY	-	-
(2) LATE	21	9
VOMITING	52	10
DEHYDRATION (ON ADMISSION)	17 (25.3%)	6 (60%)
AVERAGE DAYS ILL	37.7	20.3

POST-MORTEM FINDINGS.

Owing to the great difficulty of obtaining permission for post-mortem examinations in this district such examinations could only be carried out in two out of the ten fatal cases. The findings, however, are worth recording because they demonstrate certain differences between the acute toxic and choleraic type of enteritis.

GENERAL APPEARANCES. In both cases the body showed signs of gross emaciation and dehydration. While both these findings are also present in the acute toxic type they are not, in my experience, so gross in that type. This is probably in the main due to the fact that the illness is more prolonged in the choleraic type.

HEART. Cloudy swelling of the myocardium.

LUNGS. Broncho-pneumonia was present in both cases.

LIVER. Microscopic evidence of fatty change. This was not so obvious to the naked eye as in the acute toxic type. In one case there was severe "heart failure" change in the liver.

SPLEEN. The spleen in both cases was oedematous and congested. The change was more marked than that noted in the acute toxic type.

KIDNEYS. Congestion and cloudy swelling in both cases. No evidence of pyelites was found in either case.

SUPRA-RENALS. Cortex pale and thinner than normal. Slight congestion microscopically.

STOMACH. Mucous catarrh was present in both cases.

INTESTINES. The contents were yellow and slimy. Peyer's patches were hypertrophied and pink. To the naked eye, there was definite ulceration of lower jejunum and the ileum. Microscopically acute inflammation of the mucosa with areas of necrosis was found. This feature is uncommon in the acute toxic type and forms one of the main features of difference between the two types of enteritis. It also tends to bear out the impression, already recorded, that the main brunt of infection falls upon the gut and that it might be bacillary.

CENTRAL NERVOUS SYSTEM. No abnormality was found in either case.

MIDDLE EARS. Pus was present in the middle ears of one case. This case has already been discussed and I shall only repeat that the otitis developed only two days before death. There was no antral involvement in this case.

GENERAL TREATMENT OF THE ACUTE TYPES OF ENTERITIS OF UNKNOWN ETIOLOGY.

Possible empirical methods of dealing with the toxaemia of the acute toxic form of enteritis and the results of such treatment have already been discussed. This point seems a suitable one at which to discuss further forms of treatment which can be employed

in dealing with both the acute toxic and the choleraic types of enteritis. This may conveniently be done under two main headings, namely dehydration and its treatment and dietary treatment.

DEHYDRATION. Dehydration is a prominent feature, especially in the choleraic type of enteritis which is responsible for a large number of the clinical signs. Treatment of this dehydration is important, especially in the choleraic type. In the toxic type treatment of the dehydration may tide the infant through the toxaemia. This latter in my experience, however, is not often the case.

An understanding of the details of the physiology of water metabolism is an essential if the treatment of dehydration is to be carried out properly. The following is a summary of the important points in water and salt metabolism.

70% of the infant's body weight is due to water, some two-thords is within the cells and one-third is found in the circulating fluids. The blood volume according to body weight is much lighter in infants than in adults. In health there is a delicate balance between water intake and output so that the body content remains remarkably constant. The intake of water comprises the water drunk, the water content of the food and water resulting from oxidation of food within the body. The output is through the kidneys, lungs, skin and bowels. That water lost by the skin and lungs is least subject to variation and remains practically constant irrespective of fluid intake. The amount of water lost in the urine is subject to very wide variations and other things being equal, the urinary

output reflects accurately the water intake. In cases of severe deprivation of water, urine excretion may practically cease for a time but loss may continue to some extent through the skin and lungs. On the other hand, excessive fluid intake produces great augmentation of urine output without appreciable increase by vaporisation. Signs of serious dehydration appear when the body has lost 10% of its water content which is approximately 6% of the total body weight. Loss of 20% of the body water will result in death, (44, 31, 32).

A healthy infant requires 2½ ounces of water per pound of body weight per day to maintain water balance. The body of a healthy baby weighing 12 pounds contains some 6½ pints of water, and for the maintenance of normal water metabolism, the daily intake will have to be 30 ounces. If this infant becomes obviously dehydrated it will only have lost ¾ pounds in weight, the equivalent of 12 ounces of water. The signs of dry tongue, depressed fontanelles, dry wrinkled skin and sunken eyes are due to loss of intra - and extra - cellular fluids, the volume of blood plasma being maintained at the expense of the interstitial fluid. To combat these physical signs of dehydration, the volumes of both interstitial and intra-cellular fluid must be restored to normal. The intake, then, for 24 hours of this infant which has lost about 12 ounces of fluid will be 42 ounces. As long as the infant shows signs of dehydration, its daily water requirements may be estimated to be about 3½-4 ounces per pound body weight, (11, 52, 67, 68, 93, 100, 114, 140, 153).

Infants with gastro-enteritis lose considerable amounts of

salt as well as water and replacement of the salts forms a very important part of the treatment. The human body cannot store sodium chloride so that if the amount excreted rises for any reason a corresponding increase in intake must take place if a deficiency of salt is to be avoided. Widely varying values of blood chlorides have been put on record some being above and others at or below the average normal figure. But the general opinion appears to be that in the average case of enteritis, the blood chlorides are higher than normal.

Cooper found serum chloride high in cases of acute gastro-enteritis while it was low in chronic diarrhoea and he suggested that, in acute gastro-enteritis, there was a loss of fluid from the circulating blood and that the water left the blood stream without its full quota of chlorine. Aldridge found high serum chlorides in 21 infants suffering from acute gastro-enteritis and this was increased after the administration of saline in some cases.

Hartman suggested that administration of sodium chloride was deleterious in that it lead to a further retention of chlorides. Smellie supports this view. (3, 26, 27, 28, 29, 67, 68, 101, 102, 114, 131, 153).

METHODS OF ADMINISTERING FLUIDS. With all the above facts in mind, it becomes obvious that the correct treatment of dehydration is not a simple procedure. Theoretically exact treatment is only possible with the aid of frequent blood analysis as carried out by Csapo and Wollek, Csapo and Kerpel - Fronius and Ribadeau - Dumas

but in my opinion this is unjustified when dealing with ill babies. Where vomiting is not a prominent feature, I give fluids by mouth. Like Ellis the fluid I use is 5% glucose in half-normal saline. Where this is rejected I give 5% glucose in sterile water. As I have found these two glucose solutions satisfactory I have not tried the various solutions of weak sweetened tea, Ringers solution as suggested by Czerny, Langstein, Rominger, Strausky, Marriott and Kleinschmidt. (35, 36, 38, 43, 87, 88, 96, 97, 108, 148, 150, 160).

The nurse is instructed to present the glucose solution to the child every hour for a period of ten minutes, the child taking as much as he wishes in this time. As the condition improves milk is substituted for each alternate saline or water feed and the feeds slowly built up to the amount and times suitable for the particular age of the patient.

Where vomiting is troublesome and dehydration and acidosis severe other routes must be tried either in association with feeding by mouth or alone.

The intraperitoneal route is favoured by some.
(17, 41, 120, 125, 134, 147, 159, 167)

Personally I do not like this route and gave it up after only using it on three occasions. The shock produced and the over disturbance of an ill baby are my main objections to this method. Marfan, Tezner and Abel and Aron have reported certain difficulties and complications due to this method of injection.
(123, 161, 7,)

The intravenous administration of normal saline, dextrose solution, Hartman's solution has many devotees, (25, 29, 43, 60, 67, 87, 88, 96, 97, 108, 109, 123, 132, 150, 160) but personally I have not found this to be any more effective in infants than the subcutaneous method.

The disturbance of the patient together with the impracticability of keeping more than two cases going at one time without having an extremely large nursing staff was an important factor, militating against the use of this method. It also has its dangers because a definite strain is imposed upon the heart in an already ill child with the consequent risk of cardiac collapse which must obviously be very great in a toxaemia, and of pulmonary oedema.

Maddock et al hold this view. In my opinion this method of giving fluid is definitely contra-indicated in the acute toxic type of enteritis as these cases are already suffering from pulmonary congestion and an increase in circulating fluid of the nature of saline would definitely tend to produce a broncho-pneumonia. Wilmer's figures showing 30% of the deaths as due to broncho-pneumonia support this view. I also entirely agree with Maitland-Jones and Wilmer that, in the toxic type. although the dehydrati on may be successfully dealt with, the toxaemia remains unaffected. I cannot support the views of Hartman, Hoag and Marples, Cooper, Cohen that intravenous fluid is of any more value than subcutaneous fluid. Transfusion of whole blood has been given some attention but opinion is uniformily against its use.

In any case the introduction of whole blood into these cases

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unphysiological. In the dehydrated child the haemoglobin percentage and the red cell count are increased. This is all an indication of increased concentration of the blood due to loss of fluid. These children are not in need of haemoglobin or of erythrocytes but of fluid to reduce the concentration and to act as a vehicle for excretion of accumulated waste products. I do not consider that the results obtained by the introduction of normal saline into the blood stream justify the manipulation of the ill infant involved. It would seem that plasma might be suitable. Aldridge records some success with plasma. Laurent tells me that he has also used plasma but that he was not convinced of its value. I have not tried this method myself and so I cannot speak as to its efficacy. Hartman, Ellis and Cooper state that transfusion of whole blood is of value in the debilitated child recovering from the choleraic type of enteritis with an associated anaeima. (3, 25, 29, 43, 45, 67, 68, 100, 91, 104, 172).

As I have already indicated, I prefer the subcutaneous route. It is simple and easily managed and the fluid can be given in one dose or by the drip method. The fluid which I use is 5% glucose in normal saline and after a trial of both methods of administration, I have come to the conclusion that the single dose method at four-hourly intervals gives just as good results as the continuous drip. It has the decided advantage that a minimum of supervision is required. I would like to stress now the point that strict asepsis must be observed and for that reason I do not allow

the nurses to give the subcutaneous saline but do so myself. The extremely debilitated infants are very susceptible to infection and a minute number of organisms introduced subcutaneously can easily cause sloughing of the overlying skin surface. I usually give 80-100 c.c. of 5% glucose saline into the abdominal wall or either axilla every four hours. Contrary to expectation, the discomfort caused is minimal, due probably to the laxness of the subcutaneous tissues in the dehydrated infant and on no occasion has the glucose acted as an irritant. Occasionally the fluid is not completely absorbed. Reduction of the four-hourly dose until the glucose saline is completely absorbed in 2-3 hours overcomes this difficulty. Administration of subcutaneous glucose saline may save a child if it is given in the early stages of the acute toxic type of enteritis; given when the child is severely toxic and considerably dehydrated, it may overcome the dehydration but otherwise it does not affect the usually fatal course of the disease. Definite improvement follows the administration of subcutaneous glucose saline in cases of the choleraic type of enteritis.

FEEDING. I have used the method which is so popular among persons interested in enteritis, namely, a preliminary period of starvation of from 24-48 hours during which time the infant is given only 5% glucose in half-normal saline or glucose no sterile water every hour for a period of ten minutes. Where vomiting is troublesome, absolute starvation is imposed, the mouth being moistened with a swab soaked in glycerine and boracic lotion or simply in water. In the type

of case which can be fed by mouth but which is showing signs of dehydration, subcutaneous fluid is given also and, of course, in the type with persistent vomiting the subcutaneous method only is adopted. (25,60, 104, 108, 109, 132, 153, 172).

In some cases, very few, a definite reaction to this treatment takes place in that the vomiting ceases and the diarrhoea becomes less in 24-48 hours, but in the majority of cases this is not the case. Although Marriott, Marfan and Lelong consider that the period of starvation should be extended until four or five days I usually start feeding after 48 hours of starvation as suggested by Rominger, Czerny, Strausky, Kleinschmidt and Langstein, (38, 87, 88, 96, 97, 99, 123, 124, 150, 160).

When the period of starvation is over, an attempt must be made to get the infant back on to the food to which he has been accustomed. In no case must a breast fed infant be weaned but the breast feeds should be gradually introduced beginning with feeds twice daily and slowly building up until the child is back to his normal feeds again. (43). Children who have been artificially fed should be started on feeds of the type to which they are accustomed in a diluted form beginning with two feeds daily and gradually building up until the child is back on his normal feeding times, then the strength is gradually increased to normal. This of course, is the ideal but in practice the process is not so easy and many attempts must be made before the child is on his normal diet. In a large number of cases the feeding by mouth becomes almost an impossibility and the child has to be given what he will take. This often entails

running through the whole gamut of the artificial foods. It is for this transition stage from starvation to normal diet that many authors are in favour of a milk free diet. Czerny and Rominger recommended the use of whey or Moll's calium lactate whey, Beumer and Kleinschmidt prefer to start on rice gruel only and French author's such as Ribadeau-Dumas and Lelong prefer the use of vegetable soups or cereal decoctions. (15, 38, 87, 88, 99, 126, 127, 128, 148, 149). Dried buttermilk preparations have been successfully used by Langstein, Engel and Feer. Marriott, Utheim, Schiffe and Mosse, Bischoff, Beumer and others recommended the use of lactic acid milk mixtures instead of buttermilk soup while Weissenberg, Behrens, Gouce and Templeton have used citric acid milk. Personally, I have found bengerised milk to be the most useful feed in difficult cases.

The question of banks of mother's milk requires investigation but many administrative difficulties have to be overcome before it becomes practicable. (12, 14, 16, 44, 56, 65, 96, 97, 124, 158, 164, 168, 169, 170).

FRUIT DIETS. Some ten years ago Moro and Heisler introduced the apple diet in the treatment of diarrhoea. Feer also reported on treatment of diarrhoea with fruit juices and bananas. Since then a great deal of investigation has been done on this subject. (56, 74, 130,).

Heisler and Kohlbrugge consider that the acids, which inhibit bacterial growth are specific while Malyoth believed that the pectins, which have a high adsorbing power, act like charcoal. Favourable results have been published by many authors (10, 37, 43, 57, 64, 75, 18, 58, 74, 89, 98, 55, 106, 107, 121, 122, 129, 130, 135, 137. 126, 127, 128, 153, 163, 171,) Like Smellie, however, I was quite unable to convince myself that apples or bananas were of any real value in the treatment of acute enteritis of either type.

SUPRA-RENAL CORTEX. Harrop (66) considers that administration of supra-renal cortical hormone may be of value in cases of dehydration. I have tried this form of treatment in a few cases but I have not been impressed by the results. Laurent (91), who has used supra-renal cortical extract fairly extensively states that he has now given it up as he does not consider it to be of value in treatment.

DRUGS. In the earliest stages castor oil may be beneficial but in the later stages with a debilitated child it is contra-indicated. Opium and Belladonna may be used with some benefit where colic appears to be causing pain. Although Czerny and Keller (38) consider alcohol to be contra-indicated I have found brandy to be of value because it is a food and a sedative and to my mind is the safest and most serviceable medicine to use.

As far as chemotherapy goes, I can only state that in my experience it is of no value as a prophylactic and that the few

cases which have been treated in this way have not been improved.

VITAMINS. When the child is established on its normal diet, vitamins ought to be introduced into the diet. Orange juice and cod-liver-oil or one of the concentrated vitamin products are slowly added to the diet. Too early administration of orange juice has been found to upset some of the infants.

PROPHYLAXIS. The most important factor in prophylaxis is breast feeding. This fact has been recognised for many years but in spite of this the incidence of breast feeding is falling. Spence estimates that at the present time, in most of our big towns, 20%-30% of our babies are artifically fed from birth and that not more than a third of the mothers of these towns are fully feeding their babies up to the sixth month. It is quite possible, to my mind, that an immunity is conveyed to the infant in the mother's milk. In any case there cannot be the slightest doubt that the breast fed infant is relatively immune from enteritis. Spence is quite convinced that breast fed infants show a greater freedom from disease than artifically fed infants and Smellie (153) strongly supports this belief, My own figures are quite definite, as only three of the children of nine months and under with acute enteritis of unknown etiology were entirely breast fed up to the onset of the illness. If artifical food must be given, properly clean preparation of the feeds by an individual who is at least masked and whose hands have been carefully cleaned will do much to prevent infection of the feeds.

In the early stages much can be accomplished by withdrawal of milk feeds and the giving of 5% glucose in ½ normalsaline for 24 hours by mouth. It has been my experience in wards in which there has been an outbreak of enteritis that prompt treatment in this way of an infant who has passed a relaxed stool will often abort the attack. (153, 173).

C H A P T E R III.

UNCLASSIFIABLE TYPES OF ENTERITIS.

These cases were namely very mild attacks of diarrhoea and numbered 42 in all. 9 (21.4%) were under one year and 25 children (59.5%) were between one and three years of age. The remainder were evenly distributed between the ages of 4 and 14 years. It will be noticed that only one fifth of the cases were below one year of age. No deaths occurred among this group of cases.

On going over the case papers after two years, I noticed that the majority of the cases which could not be classified occurred in the first year of the investigation. On fresh consideration and in the light of increased experience I tried to re-assess these cases. Ten (23.8%) on clinical grounds could have been included in the bacterial group and one among the symptomatic group. It should be noted that all of the ten cases just mentioned occurred before serological examination was undertaken. The bacteriological findings from stool examinations were as follows:-

B. Morgan I, in 5 cases.

B. protens , in 2 cases.

In the remaining 35 cases no abnormal organisms were found in the stool.

C H A P T E R IV.

ROUTINE INVESTIGATION OF CASES OF ENTERITIS.

This is a suitable point at which to recapitulate briefly the salient points in investigation of all cases of enteritis in children. A complete history must be taken with special reference to feeding including recent changes of diet, recent illnesses and contact with other children suffering from enteritis. Clinical investigation must be thorough and should include examination of the tympanitic membranes at frequent intervals and microscopic examination of the urine. The stools should be examined bacteriologically on two successive occasions before accepting a negative result. Serological examination should be carried out in every case after the fourteenth day of illness.

The mechanical types can quickly be segregated and helped by good history, taking the dietetic and symptomatic forms recognised, but difficulties arise in assessing cases falling into the other groups. In a large number of cases the infective type of known etiology can be separated from the infective type of uncertain etiology if it is remembered that the former is commonest in children in over one year, that it is excepting typhoid and para-

typhoid infections, a relatively mild disease and that the stools of the dysenteric cases, especially Sonne dysentery, often contain blood and mucus. The course of the dysenteric illness is usually short. In contrast the type of uncertain etiology is oftenest found in infants of under one year, the illness is severe and becomes progressively more severe the younger the victim and the stools never contain blood and/or mucus. In the typical cases, then, the separation of the two types on clinical grounds is moderately simple. However, when atypical and mild cases are encountered it becomes extremely difficult to separate the types into the main groups. When this is the case it becomes necessary to diagnose in retrospect, relying upon the course of the illness and the results of bacteriological examination of the faeces and on serological examination. As regards the acute toxic and choleraic forms of enteritis of uncertain etiology certain points should be kept in mind. In the former toxaemia is prominent, with frequent vomiting and only slight dehydration. Cough is present in the earliest stages and is a point of some significance in the diagnosis. Death is a too frequent temination. In the latter type diarrhoea and dehydration are prominent and vomiting and cough are not troublesome. Response to symptomatic treatment is fairly good in the acute choleraic form but rarely occurs in the acute toxic form.

Usually it requires a period of 48-72 hours before one can place a fair number of the cases and often even a longer period is required as some of the dysentery group must await the result of

serological examination. The speed of arrival at a correct diagnosis is not of great moment from the patients' point of view with the present rough and ready methods of treatment. But from the point of view of other children, this delay is of considerable importance, and forms one of the major problems of the administration of an hospital dealing mainly with young children. All cases with enteritis must be nursed in separate cells. The fact that it is so often impossible to arrive at a diagnosis on first examination makes it imperative that these children should not be nursed in open wards, even with "barrier" nursing" on account of the grave risk of cross-infection.

C H A P T E R V.

THE EASTERN HOSPITAL ENTERITIS UNIT.

This thesis would not be complete without a description of the enteritis unit which housed the majority of the cases discussed in these pages.

This umit consists of nineteen cells (with complete structural separation) banked on either side of a central corridor but without ventilation into the corridors. There is, in addition, a side ward accommodating two patients. A duty room with bowl and instrument sterilisers is provided together with one bath-room and one sluice room containing an automatic single bed-pan steriliser. Ordinary dietary is served from a kitchen which also acts as a receiving room and sterilisation room for used feeding bottles.

After sterilisation the bottles are passed through a hatchway into an adjoining milk room which is reserved solely for the preparation of infants' feeds under an aseptic technique. Prior to admission of the first case to the enteritis unit, certain limitations were agreed upon as to its use. In the first place the upper age limit of children treated was placed at five years. This was done, primarily, to avoid difficulty with lavatory accommodation but, as can be seen from my figures, the demand for admission in the older age groups is very limited and these cases when they do occur are mild, if enteric fever cases are excluded. In order to facilitate nursing an attempt was made, also to limit the accommodation provided for bottle babies to one third of the total accommodation - the reserved cells being placed in the immediate vicinity of the duty room. In practice it was very soon found impossible to adhere to this - the demand for admission in the age group under one year being so great as to require on occasion considerably more than half the accommodation. A general aseptic nursing technique is employed similar to that used in the barrier and other isolation wards of the hospital. This involves the wearing of a mask in attending to the patients. As has already been mentioned, at first there were one or two cases of cross infection but since the introduction of masks this has entirely ceased. A large staff is employed (in the ratio of two nurses to three patients,) and a sharp separation of nursing duties is made into:-

A. The preparation of feeds.

B. The giving of feeds, and

C. Washing and changing of infants.

No interchange of duties is permitted but, after a period of fourteen days, duties are changed round to allow junior staff particularly to obtain experience in all aspects of the work.

PREPARATION OF FEEDS. Feeds are prepared in the milk room by a senior member of the nursing staff - normally the ward sister or her deputy. During the preparation of feeds the nurse is gowned, gloved and masked and remains in the milk room until all the feeds have been prepared for the ensuing 24 hours. The requisite number of feeds for individual patients is then sterilised in a Soxhlet apparatus, cooled and stored in the refrigerator in a wire-crate - individual crates being marked with a metal label to indicate the cell number of the particular patient. Single feeds are withdrawn as required, warmed and given by one of the staff specially allocated to feeding duty.

Particular attention has been paid to the administration of feeds and, with the staff available it is possible to ensure that babies, are taken out of bed and fed in a proper nursing position without hurry or fuss. This is desirable in any case but particularly in the severe forms of gastro-enteritis when prolonged nursing in the recumbent position is likely to aggravate any respiratory infection by encouraging hypostatic congestion of the lungs.

During the time of the investigation, it was occasionally necessary to re-admit enteritis cases because diarrhoea had recurred shortly after discharge from hospital. This was found on investigation to be due invariably to unsuitable feeding. In order to avoid this possibility a simple form was evolved which is given to the parents on discharge of the child and which describes precisely the character, quantity and frequency of feeds which have been found suitable as well as the details of other items deemed necessary such as cod-liver-oil and orange juice.

It will be noted that the hospitalisation technique is similar to that quoted by Maitland-Jones from the reports of Gladys Dick and to that recommended by Felsen (46,104,)

ENTERITIS OF CHILDHOOD.

SUMMARY AND CONCLUSIONS.

SUMMARY. The main object of the thesis is to clear up the uncertainty of classification of the various forms of enteritis of childhood which, of course, is a reflection of the great difficulty in diagnosis. However, while the subjects of classification and diagnosis were being investigated many other points of interest emerged which were followed up to a certain extent and some conclusions drawn.

A short historical review from the time of Hippocrates to the nineteenth century is given. This is designed to show how certain basic facts such as the relationship between diarrhoea and teething, hot weather, artificial feeding and overcrowding came to be recognised. There then follows a discussion of the various classifications based upon clinical, pathological, bacteriological and biochemical findings and which have not proved to be very satisfactory.

General etiological factors of enteritis of childhood then come under discussion with special emphasis upon the value of progressive public health legislation in causing a reduction in the death rate from enteritis as a whole.

The suggestion is then made that one of the reasons for the difficulty in classification lies in the fact that the clinical

basis which forms the foundation of every investigation is at fault. This, it is suggested, might be due to the true state of affairs being lost in a maze of biochemistry and bacteriology.

The classification adopted in the investigation of the 275 cases of enteritis is as follows:-

A. NON-INFECTIVE ENTERITIS.

(a) DYSPEPTIC ENTERITIS

(b) MECHANICAL ENTERITIS

(c) SYMPTOMATIC ENTERITIS.

B. INFECTIVE ENTERITIS.

(a) ENTERITIS OF KNOWN ETIOLOGY

(b) ENTERITIS OF UNCERTAIN ETIOLOGY

(1) ACUTE TOXIC ENTERITIS

(2) ACUTE CHOLERAIC ENTERITIS.

C. UNCLASSIFIABLE TYPES OF ENTERITIS.

The above classification was adopted at the commencement of the investigation and was based upon previous experience. It was not found necessary to alter this classification, but a few of the cases originally included in the unclassifiable group were subsequently thought to be more suitably included in other groups.

CHAPTER I, deals with the non-infective forms of enteritis and shows that this group is not of great importance. The dyspeptic form of enteritis is shown to be easily treated. The mechanical form may include certain serious conditions such as hypertrophic pyloric stenosis and intussusception but is rare. The symtomatic form is discussed at length and it is suggested that swallowed sputum and excretion of toxin through the intestinal wall might well be the two most likely causes. The association of enteritis with otitis media is noted and discussed fully. The conclusion is reached that undue importance has been attached to the symptomatic form of enteritis. An analysis of 492 cases of measles is used to support this view.

CHAPTER II discusses the subject of infective enteritis of known etiology but is confused mainly to Sonne dysentery. Seasonal incidence, incubation period, mode of spread, clinical features and complications are touched upon and finally the subject of diagnosis. Here I try to show that the only reliable method of diagnosis is by serum agglutination and figures are produced in support of this. This leads up to a discussion on the best routine method of dealing with a ward outbreak of some dysentery and the finding of mild cases and carriers. Treatment and pathology are also mentioned.

Infective enteritis of uncertain etiology is then discussed and the division into two types:- (1) Acute Toxic Enteritis

and (2) Acute Choleraic Enteritis

upon clinical and pathological grounds justified. Both types are

fully discussed and theories advanced as to their etiology. It is suggested that the acute toxic type is a virus infection and that the choleraic form is of bacterial origin.

Seasonal incidence, clinical picture, complications, pathology, diagnosis and treatment are dealt with fully. The question of the value of the various forms of treatment is gone into and personal experience of certain of these noted.

CHAPTER III includes the unclassifiable forms of enteritis. These are probably mild forms of the dysentery group and of the dyspeptic group.

CHAPTER IV deals with the routine investigation of cases of enteritis and makes certain suggestions which are in the main that serological examination must be carried out, clinical examination must include examination of ears and urine together with a most thorough investigation of the respiratory tract.

CHAPTER V describes briefly the enteritis unit at the Eastern Hospital in which the work recorded was carried out.

CONCLUSIONS.

A. The suggested classification is satisfactory.

B. Non-infectious enteritis is relatively unimportant and this includes true symptomatic enteritis.

C. The diagnosis of the dysenteries, especially of Sonne dysentery, can be rendered much more accurate if serum agglutination reactions are tested.

D. Infective enteritis of uncertain etiology is of two types one probably due to a virus and the other to a bacillus.

E. Treatment of all forms of enteritis is largely empirical and is not satisfactory in the case of enteritis of uncertain etiology. It is suggested that adult serum might contain antibodies.

F. All cases of enteritis must be isolated and a true 'barrier' technique adopted by nursing and medical staff.

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