OBSERVATIONS ON INFANTILE DIARRHOEA

WITH NOTES ON TREATMENT.

by

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 Thesis presented
for the Degree of M.D.
SECTION I.
INTRODUCTION.
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Infantile diarrhoea is known to have been rampant from earliest times, and to have been associated with a very high mortality. Even now, in spite of the tremendous reduction in the incidence of the disease in comparatively recent times, the mortality is relatively great.

The earliest writings on the subject of infant hygiene were those by Soranus of Ephesus (2nd Century A.D.). These writings were stamped by the mark of a master. It may be mentioned in passing, that there was a recognisable account of rickets, in these treatises. Susruta (5th Century A.D.), a Brahmin physician, in a book on infant hygiene, which bore his name, commented on the purgative effect of honey (sugar diarrhoea) in the newborn infant. It was not until the 20th Century that any other contribution on the influence of the constituents of the infant's food, as a possible cause of diarrhoea, was made.

Paul of Aegina (625-690 A.D.) gave a valuable account of pediatric learning up to, and including, his time. In fact, the Pauline pediatrics held the field unchallenged till the Renaissance. The first contribution of the Renaissance to pediatrics, appeared in 1472, under the title of 'De aegritudinibus infantum', by/
by Bagellardo of Padua. This tract was closely followed by similar ones by Metlinger of Augsburg in 1473, and by Roelants of Mechlin in 1483 or 1484.

The earliest English contribution to infant hygiene appeared in 1546, as part of the contents of a book by Thomas Phayre, called 'The Regiment of Life'. The section dealing with infantile disorders in this book was given the separate title of 'The Boke of Children'. Phayre, it is interesting to note, opposed wet-nursing, in spite of a very high infantile mortality at that time. This high rate was largely due to a complete lack of hygiene, personal, public and domestic.

From 1669-1671, epidemic infantile diarrhoea raged in London, and in some 8-10 weeks, 2000 infants succumbed. In fact, two-fifths (2/5) of the total deaths at that time were in infants under 2 years of age. Sanitation, in any form, was then, to all intents and purposes, unknown and the unfortunate newborn infants were wrapped up in swaddling clothes and allowed little or no freedom of movement. Discharges from the ears, and eczemas were left untouched and untreated, as part of the orthodox regime, then in faveur. Even the King's physician, Walter Harris condoned this state of affairs. Artificial feeding in lieu of breast feeding, was then unknown. Harris, who was physician to both Charles II. and William III. wrote a book entitled 'De morbis acutis infantum', which appeared in 1689.

Jean-Jacques Rousseau, in his novel 'Emile', published in 1762, condemned, in no uncertain tones, /
tones, the increasing tendency of the French mother not to feed her infant at the breast. At that time the infantile mortality was appallingly high. In England, Sir Hans Sloane stated that the mortality of dry-nursed to breast-nursed infants was as 3:1. Babies, which were not breast fed, were given water-pap - bread moistened in water, - or baked flour slightly moistened. At the British Lying-in Hospital, compulsory breast feeding of the infants, resulted in a fall in the infant mortality by 60%. As a result of the excessively high death-rate among the dry-fed infants, the employment of wet-nurses became popular. Many were the ways and means adopted by women of all types to obtain employment as wet nurses, considering they were rewarded for their pains by a remuneration, at the rate of 25 guineas per annum. In fact, the latter part of the 18th Century marked the peak point of the wet nurse.

In 1784, Underwood, in this country, recommended the use of boiled cow's milk diluted with barley water, as an efficient substitute for breast milk. Thus he laid the foundation of artificial feeding of infants by cow's milk and water mixtures. Underwood also curtailed the duration of breast feeding to 12 months. With this development of artificial feeding with milk and water mixtures, the feeding bottle passed through the various stages from a cow's horn to a glass bottle, and so on to the present day so-called 'hygienic' bottle.
Benjamin Rush, an American pediatrician of outstanding merit, was the first writer ever to give anything like a proper description of Cholera Infantum. This he did in 1789. He noted the regularity of its appearance in the summer months. Rush was also the first to correlate the incidence of the disease with the occurrence of the hot-weather months. The description which he gave was one of exceptional accuracy, and he refuted the prevailing theory that the condition was due to teething. He held that Cholera Infantum was a form of adult cholera and remitting fever, though it occurred earlier than the adult cases. Rush demonstrated very clearly that the condition occurred at one season of the year only. Consequently, it would seem that Cholera Infantum was prevalent in America at that time, but unknown on the Continent of Europe. In fact, when it appeared in Europe at a later date, the disease was termed Rush's Disease.

With the birth of new sciences and their development, in the 19th Century, attempts were made to isolate the various causes of infantile diarrhoea, and to classify them according to the prevailing medical opinion. Thus, when Morbid Anatomy was engaging the attention of the profession, Wiederhofer, in 1880 suggested a classification according to the post-mortem findings. Various other attempts were made to do so in this direction, but they were attended by no great success. This lack of success was in no small measure due to the fact that often the most severe clinical cases of infantile diarrhoea presented very few signs/
signs after death, and vice versa.

Charles West, the main founder of the Great Ormond Street Hospital for children, wrote at great length on the subject of infantile diarrhoea, without, however, elucidating much new information. Ballard, in 1885, made the important observation that summer diarrhoea began when the ground temperature, as registered at a level of 4 feet, reached 56 F. or more. He also noted that the curve of incidence of the disease followed more closely the temperature curve of the soil at 4 feet, than it did the atmospheric temperature curve.

The following year, 1886, Escherich, after a very extensive investigation into the bacteriology of the intestinal flora in infants, attempted to classify infantile diarrhoea according to the organism found in the bowel of such infants, as were suffering from the disease. Similar work has been going on more or less continuously ever since.

An entirely new light on the subject of the aetiology of infantile diarrhoea, was shed by Czerny in 1906. This German worker laid particular stress on the importance of certain constituents of the infant's food as being primary factors in the production of the condition. It will be remembered that Susruta, in the 6th Century, had commented on the purgative effect of honey in the newborn infant. Czerny was closely followed in this field by Finkelstein, also a German.

This/
This worker put forward the theory that the minerals in the artificial food of the infant, might be the cause of the diarrhoea. He described a form of modified cow's milk which seemed to meet the requirements of his theory. Latterly, however, Finkelstein abandoned his theory in part.

Since these two observers produced their momentous works other pediatricians have entered the field of investigation, and a considerable amount of work has been carried out in Britain, America and the Continent in an effort to elucidate the cause of much of the diarrhoea affecting infantile life. The names of Marriott, Alan Brown, Marfan and Nobecourt, to mention only a few of these well known research workers, may be cited.

It will be seen from the foregoing brief historical review, that only in the past 60 years or so, has much been done in the determination of the cause of infantile diarrhoea. The respective theories will be considered in detail in the section dealing with Aetiology and Pathology.

I have specially prepared the following table, reproduced in graphic form also, (Table I.) to show the mortality rate in Scotland and in Edinburgh, from Diarrhoeal diseases in children under 2 years of age, from 1871-1937, inclusive. For purposes of clarity, I have grouped the rates from 1871-1900, inclusive, into 5 year periods, but thereafter, I have given the rates for each individual year from 1901-1937, inclusive.
### TABLE I.

Infantile diarrhoeal diseases.

Death Rates for Scotland and Edinburgh. 1871-1937.

Death Rates of children under 2 years of age, per 10,000 population.

<table>
<thead>
<tr>
<th>Year</th>
<th>Scotland</th>
<th>Edinburgh</th>
</tr>
</thead>
<tbody>
<tr>
<td>1871-75</td>
<td>5.3</td>
<td>5.2</td>
</tr>
<tr>
<td>1876-80</td>
<td>4.8</td>
<td>4.7</td>
</tr>
<tr>
<td>1881-85</td>
<td>4.5</td>
<td>4.1</td>
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<tr>
<td>1886-90</td>
<td>4.4</td>
<td>4.5</td>
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<tr>
<td>1891-95</td>
<td>5.3</td>
<td>4.2</td>
</tr>
<tr>
<td>1896-1900</td>
<td>6.4</td>
<td>5.8</td>
</tr>
<tr>
<td>1901</td>
<td>7.5</td>
<td>5.3</td>
</tr>
<tr>
<td>1902</td>
<td>5.8</td>
<td>2.7</td>
</tr>
<tr>
<td>1903</td>
<td>5.0</td>
<td>2.7</td>
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<tr>
<td>1904</td>
<td>5.5</td>
<td>3.4</td>
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<td>1905</td>
<td>4.9</td>
<td>3.9</td>
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<td>1906</td>
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<td>5.4</td>
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<td>1910</td>
<td>4.4</td>
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<td>1911</td>
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<td>3.0</td>
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<td>1912</td>
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<td>4.4</td>
<td>2.1</td>
</tr>
<tr>
<td>1914</td>
<td>4.6</td>
<td>2.9</td>
</tr>
<tr>
<td>1915</td>
<td>5.6</td>
<td>2.1</td>
</tr>
<tr>
<td>1916</td>
<td>3.4</td>
<td>2.3</td>
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<tr>
<td>1917</td>
<td>2.5</td>
<td>1.3</td>
</tr>
<tr>
<td>1918</td>
<td>2.1</td>
<td>1.2</td>
</tr>
<tr>
<td>1919</td>
<td>2.1</td>
<td>0.8</td>
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<tr>
<td>1920</td>
<td>2.6</td>
<td>1.7</td>
</tr>
<tr>
<td>1921</td>
<td>3.1</td>
<td>2.0</td>
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<tr>
<td>1922</td>
<td>1.8</td>
<td>1.3</td>
</tr>
<tr>
<td>1923</td>
<td>1.8</td>
<td>1.5</td>
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<td>1924</td>
<td>1.7</td>
<td>1.3</td>
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<td>1.8</td>
<td>1.8</td>
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<td>1926</td>
<td>1.8</td>
<td>1.2</td>
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<td>1927</td>
<td>1.4</td>
<td>1.0</td>
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<tr>
<td>1928</td>
<td>1.5</td>
<td>1.0</td>
</tr>
<tr>
<td>1929</td>
<td>1.4</td>
<td>1.2</td>
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<tr>
<td>1930</td>
<td>1.2</td>
<td>0.9</td>
</tr>
<tr>
<td>1931</td>
<td>1.4</td>
<td>0.7</td>
</tr>
<tr>
<td>1932</td>
<td>1.7</td>
<td>0.7</td>
</tr>
<tr>
<td>1933</td>
<td>1.6</td>
<td>0.7</td>
</tr>
<tr>
<td>1934</td>
<td>1.2</td>
<td>0.9</td>
</tr>
<tr>
<td>1935</td>
<td>1.3</td>
<td>1.1</td>
</tr>
<tr>
<td>1936</td>
<td>1.9</td>
<td>0.7</td>
</tr>
<tr>
<td>1937</td>
<td>1.6</td>
<td>0.8</td>
</tr>
</tbody>
</table>
It will be seen that for Scotland, from 1871-1890, there was a tendency for the rate to fall, but to do so only slowly. From 1891-1900, there was a rapid rise to a peak in 1901. A sudden drop followed in 1902. Thereafter, until 1915, the rates tended to swing rather widely. Since 1915, however, there has been a much steadier, and more progressive fall, due doubtless in no small measure to the institution of Infant Welfare Centres throughout the country in 1917, and succeeding years. The rates for Edinburgh have followed rather similar lines, but in the 5 years period, 1886-1890, the rate was actually greater than that for the whole of Scotland during the same period. In the following 5 years period, 1891-1895, the rate had dropped to well below the general rate for Scotland for the same period. In both Scotland and Edinburgh, after both had reached a peak in 1896-1901 period, there has been a tendency to progressive fall. In the case of the rate for Scotland, this fall has been associated with a degree of 'swing'.

In 1906, Voluntary Visitation of children in their homes was undertaken in Edinburgh. By that time, however, the progressive fall had begun. In 1917, the Infant Welfare Department of the City of Edinburgh was instituted. Since then, there has been a general tendency to further fall in the mortality from diarrhoeal diseases in infants under 2 years of age. Though
infantile diarrhoea still carries off many infants, the rate has dropped progressively from 1901, until now the rate is almost \( \frac{1}{4} \) the rate of that year.

Unfortunately, these figures are not strictly comparable with similar statistics for England and Wales. The main reason for this is the difference in the methods of calculation. In Scotland, the Registrar General calculates the rate as the number of infants, under 1 year of age, dying from diarrhoeal diseases per 10,000 population. In England and Wales, the method adopted by the Registrar General, is to calculate the rate as the number of deaths of infants under 1 year, dying from diarrhoeal diseases, per 1,000 live births. Now that still-births are being registered in Scotland, we may hope for more uniformity in the calculation of those rates, so that, inter alia, some comparison may be made between the rates obtaining in Scotland, and in England and Wales.

In the table produced above, I calculated the rates for Scotland and for Edinburgh, for infants under 2 years of age. I felt that this mortality rate gave a better indication of the number of deaths from diarrhoea, than would be given by considering only infants under 1 year of age.

However, I have also reproduced a table from the Returns of the Registrar General for England and Wales in 1927. This table gives the Mortality Rate from Diarrhoeal/
Diarrhoeal Diseases of infants under 1 year, per 1,000 live births. The period under review extended from 1861-1927 inclusive. This table will also serve to demonstrate the steady decrease in mortality from diarrhoeal diseases, during that 70 years period. It will be noted, that there is also a peak for the 5 year period 1901-1905. This is rather similar to that shown in more detail for Scotland for the same period.

**TABLE 2.**

**Infantile Diarrhoeal Diseases.**

**Death Rates for England and Wales.**

1861-1927.

Rates given are deaths under 1 year per 1,000 live births.

<table>
<thead>
<tr>
<th>Year</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1861-65.</td>
<td>15.</td>
</tr>
<tr>
<td>1901-05.</td>
<td>23.</td>
</tr>
<tr>
<td>1911-15.</td>
<td>19.</td>
</tr>
<tr>
<td>1916-20.</td>
<td>9.</td>
</tr>
<tr>
<td>1921-25.</td>
<td>8.</td>
</tr>
<tr>
<td>1925.</td>
<td>7.</td>
</tr>
<tr>
<td>1926.</td>
<td>8.</td>
</tr>
<tr>
<td>1927.</td>
<td>6.</td>
</tr>
</tbody>
</table>

Not only has the mortality rate from diarrhoeal diseases dropped in both countries, North and South of the Border, but diarrhoea has fallen as a cause of death from second to now about fifth place.

The epidemic form of infantile diarrhoea, called Summer Diarrhoea, is now, fortunately, a thing of the past. Nevertheless, diarrhoeal conditions are a common cause of death in the infant. Diarrhoea is also less noticeably/
noticeably prevalent in the warm summer months, and is more evenly distributed throughout the year. In 1936 and 1937, however, it was found that the peak of the mortality for infants under 2 years of age, dying from diarrhoeal diseases in Edinburgh, was attained in both years around the month of March. This peak coincided with a rather warmer turn in the weather than was usual for that period of the year, and curiously enough this meteorological phenomenon was repeated in both these years.

The explosive onset, high infectivity, and high mortality of this acute form of Infantile diarrhoea, is now but rarely seen. This is due, I believe, in no small measure, to the remarkable advances in Infant Hygiene, but not to that alone, but also to the advances in Preventive Medicine and Public Health generally. The improved sanitation in cities and in rural areas, the raised standard of general education, and especially the education of the mothers and future mothers, in infant hygiene and nutrition, have all contributed very materially to this reduction of the severer forms of infantile diarrhoea. The better education of medical students and doctors in infant life and health has been a not inconsiderable factor in assisting to lower the incidence of, and mortality from, this once dreaded extinguisher of infant life.

Improved milk supplies, and the greater care taken in/
in the examination and control of dairy premises, as a result of Public Health legislation, have been notable advances in the prevention of infantile diarrhoea.

Though pasteurisation of all milk supplies has not yet become universal, the insistence on the boiling of all milks, whether designated or not, before administration to the infant, has been a measure of no mean importance in prevention. Until, however, we can obtain control over all like supplies, and can have the milk supplied direct from producer to consumer, and can educate the latter to the dangers attendant on the leaving of milk exposed to the atmosphere of a dusty, warm, and often overcrowded room, the spectre of the epidemic form of diarrhoea will ever remain, ready to return to life.

The introduction of dried, condensed and evaporated milks marked very real advances in infant hygiene. The purity of the milk could now scarcely be questioned. Though bacteria were not all killed in the process of drying or condensing, their virulence was practically reduced to nil. Injurious preservatives are also prohibited in such foods. The risk of infection was, therefore, considerably decreased. Again, however, the possibility of contamination in the home, if carelessness existed, still remained. (Jones and Wright, 1936, Jones, 1937).

Better home conditions, higher standards of general education, the gradual decrease in the incidence of the Musca Domestica, coincident with the decrease in horse traffic.
traffic, and the penetration of the health visitor into the home, are all factors tending to lessen the incidence of diarrhoeal conditions in infants. The introduction of Infant Welfare Centres some 20 years ago, has contributed to the reduction of the disease. Early departures from the normal health of the infant are promptly investigated, and treated in such centres. In fact, by careful supervision of the feeding and general hygiene, diarrhoeal upsets may be largely prevented. I have prepared the following table to show the progressive increase in the new cases under 1 year, and the total attendances per 1 year, at the various Infant Welfare Centres in Edinburgh, from 1919-1937 inclusive. Records for the 2 latter years of the World War were unobtainable.

**TABLE 3.**

Infant Welfare Centres (Preventive).

Record of New Cases and Total Attendances. 1919-37.

<table>
<thead>
<tr>
<th>Year</th>
<th>New Cases under 1 year of age</th>
<th>Total Attendances</th>
</tr>
</thead>
<tbody>
<tr>
<td>1919</td>
<td>1027</td>
<td>11,516</td>
</tr>
<tr>
<td>1920</td>
<td>1401</td>
<td>16,669</td>
</tr>
<tr>
<td>1921</td>
<td>1769</td>
<td>21,375</td>
</tr>
<tr>
<td>1922</td>
<td>1383</td>
<td>17,868</td>
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<td>28,978</td>
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<td>1573</td>
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<td>1625</td>
<td>29,979</td>
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<td>1929</td>
<td>1556</td>
<td>27,866</td>
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<td>1930</td>
<td>1707</td>
<td>31,733</td>
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<td>1931</td>
<td>2037</td>
<td>35,354</td>
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<td>1932</td>
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<td>40,262</td>
</tr>
<tr>
<td>1936</td>
<td>2727</td>
<td>43,011</td>
</tr>
<tr>
<td>1937</td>
<td>2961</td>
<td>45,363</td>
</tr>
</tbody>
</table>

Child Welfare Department instituted in May 1917.
The inclusion in the school curriculum, of Mothercraft classes for girls between the ages of 13-16 years, who attend the day schools, has been a regular feature in Edinburgh for some years past. Mothercraft classes, for expectant and nursing mothers, usually held at the Infant Welfare clinics, and conducted by the health visitors, are powerful means in our hands to educate the mothers of to-day and to-morrow, in the basic principles of infant feeding and hygiene. Naturally, at these classes, the vital and all-important subject of breast feeding is, and must be stressed. The doctor, whether in private or in public practice, must insist more strongly than is at present done, on the perseverance of breast feeding. To illustrate the increased interest in mothercraft, displayed by the mothers attending the centres, I have extracted from the Annual Report of the Medical Officer of Health of the City of Edinburgh, the following table, showing the figures of attendance at the mothercraft meetings, 1924-1937, inclusive. These classes were instituted in 1924, largely due to the encouragement and enthusiasm of the then Lord Provost of the City, the late Sir Thomas Hutchison. He presented a shield, the Hutchison Shield, for competition by written examination, among the mothers attending the classes.

Table 4./
It will be seen from this table, the increased interest and enthusiasm of the women, which yearly seems to be increasing. The knowledge displayed by these women is indeed remarkable and encouraging.

Medical students should receive a longer training in Infant Hygiene, and the harvest reaped at a later date from such a training, would, I am sure, be greater than that reaped from the present sowing among the stones of the specialties of less importance to the public weal. The recent introduction of a Diploma in Child Health, by the two sister English Colleges, is a step in the right direction, so far as post-graduate training is concerned.

All those around the mother during her nursing period, and in especial, the health visitor and physician, must do all that they can to encourage her to feed/
feed her child herself. Too often does one hear, "My milk does not agree with baby", when, in point of fact, there is usually only a small defect in the technique of the feeding. The rectification of these defects invariably leads to a contented infant, and concurrently, a happy and contented mother. The economic factor is, of course, an important one, and, if this was more often pointed out to the women, a greater desire to feed their infants would probably be displayed. The risks of artificial feeding are also considerable, and ever present, even to the most careful. Spence (1938) declares beyond any doubt, that breast fed infants show greater freedom from disease and greater powers of recovery, than artificially fed babies. He calculates that 20-30% of babies are artificially fed from birth, in many, if not in most, of our big cities.

Research into the causes of infantile diarrhoea is being continually carried on. It seems to me very doubtful if any one agent will ever be found for the so-called infective type of diarrhoea, the definite dysentery cases excluded, unless perchance, the cause remains enshrouded in the mists of obscurity among the ultra-microscopic viruses, waiting to be isolated from the present darkness. The more one meets with cases of diarrhoea in infants the more one is puzzled by them. History has shown, in this disease as in others, that nothing/
nothing is to be lost, and all to be gained, by applying the age old adage 'Prevention is better than cure'.

Thus the supervision of the mother, and of the infant during the early days of its life, and the encouragement of breast feeding, will still further reduce the incidence, and consequently the mortality from this disease. In the case of artificial feeding, as when the mother is ill, or otherwise unable to feed her child, strict supervision of the preparation of the feeds, will contribute towards preserving the health and well-being of the child.

Carter (1927) described an admirable scheme for the home treatment of those children presenting the early symptoms of the summer form of infantile diarrhoea. He has demonstrated clearly the value of early, prompt and efficient treatment of these cases. His paper impressed me considerably, and stimulated me to pursue a somewhat similar scheme, though the type of case was different.

I have collected a series of 250 cases of diarrhoea in infants under 2 years of age, whom I examined and treated at various infant welfare centres throughout Edinburgh, during the past 2 years. They have been culled from all parts of the city, and have been treated at home, with one or two exceptions. They were all under my own supervision, and the health visitors visited and assisted in the carrying out of the prescribed treatment, encouraging the parents to continue with the measures advised. Recently, I have experimented/
experimented with the Apple Diet, in some of the cases later to be reviewed, and the results have been most encouraging and extremely interesting.
SECTION 2.

AETIOLOGY AND PATHOLOGY.
There seem to be probably at least, two causes which render the child particularly liable to diarrhoea. In the first place, the hydrochloric acid secreted by the stomach of the infant is relatively small in amount. Swallowed organisms, therefore, may get more easily past this already weak barrier. In the second place, infants, and in particular, artificially fed children, are nourished almost exclusively on cow's milk. Now this milk is by no means ever a sterile fluid. Even the certified forms are not germ free (Ledingham, 1931). Pasteurised milk has on occasion, been found to contain pathogenic organisms (McIntosh and Whitby, 1931).

These statements would appear, at first sight, to make the aetiology of diarrhoea in infants, clear. The problem is not nearly so easy as thus suggested. Up to the present time, no single cause has been found which can be definitely stated to be the exciting factor in every single case of diarrhoea. Modern work on the aetiology of diarrhoea in infants, points to three different groups of case at least. These three groups have each a definite and distinctive aetiological basis. These three groups are as follows:

(1) Those cases due to infection in the bowel—Enteral infection.

(2) Those cases due to dietetic causes.

(3)/
(3) Those cases occurring in the presence of infection outside the bowel - Parenteral infection.

This is the classification adopted by Graham (1938), and it has met with general acceptance.

In the past, the various theories of the causation of infantile diarrhoea have reflected, to a large extent, the current medical progress at the time. The purely historical aspect of this has been briefly reviewed in the Introduction.

Since Escherich, in 1886, published his researches into the bacteriology of the intestinal canal of infants, work on the bacteriological aspect of the subject has been pursued. As a result of the various activities in this sphere of research, it has been established, beyond question, that in certain instances, the diarrhoea is due to bacterial action. In some of these instances, the action is due to specific pathogenic organisms, in others, to non-specific organisms. In this latter type of case, the diarrhoea appears to be due to the abnormal activity of non-pathogenic bacteria in a region of the alimentary tract which is normally free from organisms.

The first serious attempt on the bacteriological aspect of infantile diarrhoea was published by the Rockefeller Institute (1904). This was a monumental report, and a first class demonstration of the value of teamwork. The cases investigated were of the epidemic/
epidemic summer form of infantile diarrhoea. The type of organism preponderating in this series was a member of the dysentery group of bacteria— the Flexner-Harris organism. Only exceptionally was the Shiga form found. When it was found, it was in association with B. Flexner-Harris. The conclusion reached by these workers was to the effect that "the central fact was the occurrence frequently, of a specific organism in the diarrhoeal diseases of children which had, up to that time, been regarded as of specific pathogenic action in human beings, and further, that this organism was considered to be the cause of the form of dysentery of adults and children associated with necrotic and pseudo-membranous lesions of the intestinal mucosa, and marked by high infectivity."

Charlton and Jehle (1904) considered that summer diarrhoea in infants might be caused by various organisms of a non-dysenteric type, especially B. Coli Communis. They also noted the frequent occurrence of "vast numbers" of streptococci in the green stools, a finding which had also been recorded by the Rockefeller workers. In the case of the latter team of investigators, however, the streptococci were more especially observed with the dysentery organisms.

In this country, work on the bacteriological side of summer diarrhoea, was carried out by Morgan (1906). He described a hitherto unknown bacterium, and suggested that/
that it might be the probable cause of the diarrhoea. This organism, designated the B. Morgan No.1., was isolated by Morgan from 28 out of 58 cases investigated. Of these 28 cases, B. Morgan No.1 was found in 11 out of 28 cases clinically diagnosed as acute infective diarrhoea, and in 17 cases out of 30 clinically diagnosed as catarrhal enteritis. Morgan also recovered another organism - B. Morgan No. 3 - from 5 out of the 58 cases. He found that this No. 3 bacillus was agglutinated by the patient's blood, and that it was pathogenic to rats. Morgan concluded that this No.3 organism was also a probable cause of infantile diarrhoea.

A further bacillus - B. Morgan No. 4 - was isolated in these researches, from 3 out of the 58 cases. This bacillus was shown to resemble the Flexner group of B. Dysenteriae. Morgan also suggested that this organism might be a probable cause of infantile diarrhoea. He is careful to point out, however, that the cause of epidemic infantile diarrhoea in America, differed from that found in this country.

Morgan (1907) continuing his work, published a further paper on B. Morgan No.1. He found that it preponderated over all other types of non-lactose fermenting bacilli, and that it was present in just under 50% of a further series of 34 cases which he had investigated. This organism was demonstrated to be pathogenic/
pathogenic to animals, producing diarrhoea and death in young rabbits, rats and monkeys. In diarrhoeal states in general, Morgan observed that the lactose-fermenting organisms, so characteristic of normal faeces, were less abundant, and tended to be replaced by non-lactose fermenting types.

Ledingham and Morgan (1910) continued the researches of the latter, confirmed the previous findings, and enlarged thereon. Jones (1937) believes that the Morgan bacilli are common causes of dysenteric outbreaks among children. This is not, however, the general consensus of current opinion.

Mellanby (1916) attempted to determine the maximal and minimal conditions under which toxins could be absorbed from the alimentary tract. He suggested that the toxic factor might be an amine base, but he was unable to show any relationship between any special toxin and infantile diarrhoea, especially the epidemic form. Brown and Boyd (1923) believed that they had experimental proof that a toxin could actually be produced in the intestinal mucosa itself. Boyd (1923) found that the portal blood, in fatal cases of infantile diarrhoea, contained a toxic substance, which, on injection into animals, caused rapid collapse. This toxic substance was closely allied to histamine.

Paterson and Nabarro (1922) carried out bacteriological examinations in 41 out of 84 fatal cases of epidemic/
epidemic summer diarrhoea. The organism most frequently isolated was the B. Coli Communis. B. Coli Anaerogenes was also frequently met with, but Nabarro expressed doubt as to whether this organism might have any aetiological significance in the outbreak. The organism was also found to occur in several of the survivors, and numerous strains were isolated. These strains were not agglutinated by dysentery serum. Paterson and Nabarro also observed that the infectivity of their series was slight. They pointed out, nevertheless, that the occasional high infectivity shown by some cases might be due, in all probability, to a true typhoid or dysentery infection. Furthermore, typhoid infection in the young child is often far from being typical in its clinical manifestations, as compared with those presented by the adult. The absence of the rash may be quoted as an example of atypical typhoid fever as shown in children. Such cases are easily missed, and conditions consequently become optimum for further spread from lack of precautionary measures.

It is interesting that Nabarro failed to isolate B. Morgan No. 1 from any of these 41 cases. Findlay (1933) believes that this organism is a secondary invader in cases of infantile ileo-colitis. With this conclusion I cannot agree. I have watched an outbreak of a B. Morgan No. 1 infection in a ward of infants convalescing from whooping cough. This infection spread throughout/
throughout the ward, and was associated with 4 deaths out of some 30 children affected. In this particular instance, there was no question of delay in carrying out the bacteriological examinations of the stools. Specimens were plated and incubated forthwith, and also independently examined in the local municipal laboratory. A feature of this outbreak was the prominence of vomiting, which, in some cases, overshadowed the diarrhoea. The question was accordingly raised as to whether there was also a gastric as well as an intestinal form of the disease. The occurrence of vomiting will be commented upon later.

Brown et al (1930) carried out an investigation in Toronto, into the epidemiology of the summer outbreaks of infantile diarrhoea occurring in that city. They proved that a large number of the cases which occurred in these epidemics, were due to infection by the dysentery group of bacilli. Several types were found. B. Dysenteriae Sonne was found frequently in this investigation, and several workers have since confirmed Brown's findings.

Nabarro and Signy (1932) have suggested that chronic carriers of the Sonne organism exist, and that these carriers are a potent source in disseminating the disease. Full proof of this is lacking, however. These two workers maintain, and I believe, rightly so, that Sonne infections are by no means as uncommon as was/
was at one time thought. B. Dysenteriae Sonne was isolated from 5 cases in this series under consideration here.

Smith (1931) has put forward the theory that there is a form of Sonne infection, which appears to affect the gastric mucosa more than the intestinal mucus membrane. In this form of infection, there is severe, and often intractable, vomiting. This suggestion requires further investigation. It is generally agreed that the vomiting is a toxic phenomenon (Turner, 1938).

Miller (1938) described an outbreak of Sonne dysentery in London. He stated that danger to life from this type of infection was small, and only occurred in weakly infants. Observations in Edinburgh during 1937 and 1938 would not tend to bear this out. During these two years, especially during the spring months of both years, severe attacks of Sonne dysentery occurred in certain districts of the city. Several of the youngest affected were profoundly toxic from the very outset of the disease, and not a few died within the first 24-48 hours of illness. Bowes (1938) and Trimble and Brothwood (1938) have described attacks of Sonne dysentery due to infected milk supplies. Bloch (1938) described a large outbreak of dysentery in Glasgow, in which, out of 86 positive findings in stool examinations, 78 cases were due to the Sonne organism. It is worthy of note that Bloch mentions that 8 of these 78 cases were/
were asymptomatic. He also commented on the general increase in Sonne infections in this country. Sheldon and Evans (1940) also commented on the frequency of Sonne dysentery as a cause of diarrhoea among evacuated children.

A Ministry of Health Report (1938) described in great detail, an outbreak of gastro-enteritis in Wiltshire in 1936. This outbreak was due to the ingestion of contaminated milk. Some 100 children were affected and all recovered. In every case, bacteriological examination of the stools yielded negative results. A sample of the milk, however, yielded the 'Dublin' type of Salmonella organism. Confirmatory evidence of this finding was obtained when the blood of the affected children was examined. It was found in every case, to agglutinate the specific organism in high titre. The Report hints that summer diarrhoea may sometimes be attributed to milk or other foods contaminated in such a way as occurred in this instance, when a mechanical milker was at fault. The main conclusion reached was to the effect that all milk should be efficiently pasteurised, before being passed on to the consumer. Bendixen et al (1937) gave it as their considered and emphatic opinion that all liquid milk for human consumption should be adequately pasteurised or boiled. Guthrie and Montgomery (1939) described a series of cases of B. enteritidis Gaertner, in infants under 2 years/
years, in which the outstanding feature was the frequency of septicaemia which caused death in 6 out of 28 cases.

Sansby (1938), in an interesting study of milk bottles and caps, pointed out that the sealing of the bottles was not, in itself, sufficient to prevent contamination of the milk, unless the whole of the pouring surface of the bottle was also covered by the cap. The cap, once loosened or removed, should not be capable of manual replacement. This observation serves to demonstrate that the simple process of pouring milk from one vessel to another is not devoid of danger. As pointed out previously, even certified milks can carry infection, and pasteurised milk can retain, undamaged, pathogenic organisms.

But pathogenic organisms are not the only type of infective agents capable of producing diarrhoea in the infant. Bloch (1920) is of the opinion that in most cases of the epidemic form of diarrhoea, coliform bacilli, non-pathogenic to adults and children over a certain age, are probably the cause of the disease. There is considerable evidence to show that many of the infantile diarrhoeas are due to the growth of organisms in the upper alimentary tract, which are normally resident in the lower gut. Bacteriological examinations of the stomach and upper bowel of infants with severe diarrhoea, have often revealed organisms of the/
the B. Coli group in large numbers. The significance of this finding is difficult of explanation. Is the presence of these organisms in sites normally sterile, the cause or the effect of the disease? The tendency seems to be to regard their presence as effect, producing, as a result of their multiplication, either an actual septicaemia from absorption from the gut, as has occasionally been demonstrated, or else, by the manufacture of toxins, which on absorption, are capable of producing serious effects on the body. Plantenga (1935) has shown that the administration of filtrates of broth cultures of B. Coli by mouth, can initiate diarrhoea and vomiting. Similar results are not obtained when the same cultures are introduced into the rectum. An endotoxin does not seem to be the cause, as killed cultures of B. Coli, on injection, do not produce symptoms. The symptoms may be due to decomposition products of coliform bacilli.

Bacteriological examinations of the stools of infants suffering from diarrhoea, have produced disappointing results. There is little uniformity in the findings, but all investigators are agreed that a small group exists in which a pathogenic organism is the definite cause of the diarrhoea. This organism is usually of the dysentery group, and the B. Dysenteriae Sonne and B. Dysenteriae Flexner are the commonest. B. Typhosus is occasionally encountered, as are members of/
of the Salmonella group of food poisoning bacteria. These pathogens cause the disease to have a high infectivity, and explosive outbursts are therefore liable to occur. Before one can say that a particular case is not due to any of these pathogens, several specimens of stool must be examined with negative results, as one examination is valueless. Similarly agglutination tests should be carried out whenever possible, as agglutination reactions may be found positive even when no specific pathogenic organism is found in the stools.

Mention must be made of a severe form of diarrhoea due to infection with the fungus Oidium albicans. The infection usually spreads to the stomach and intestine from the mouth. Often the oral signs have cleared up where the intestinal symptoms make their appearance. It is not uncommon about the third week of life, though it may occur later. The intestine presents a remarkable appearance, and often appears to be dripping with blood, and, of course the typical thrush patches may be seen on the intestinal mucosa. This is a particularly severe form of infantile diarrhoea, and has a high mortality.

In the majority of these infections of the alimentary tract, no specific pathogenic organism is found, and there are no agglutination reactions. The cause of these cases remains obscure. B. Coli is generally accepted as the probable cause of the disease, but again/
again, no specific strain of the coliform bacillus has been incriminated. Bloch (1920) concluded that the clinical picture, pathological findings and epidemicity are the same whether pathogenic bacteria are present in the stools or not. Gastro-enteritis in infancy, he attributes to infection - not to any specific infection - nevertheless to infection. In most cases, coliform organisms, non-pathogenic to adults and children over a certain age, are the probable cause of the disease. In a minority of the enteral cases, the infection is due to organisms pathogenic to adults, e.g. B. Dysenteriae. Paterson (1922) agreed that this is the fairest summing-up of the evidence we have up to the present. This view is also held generally, but only to a minority of the infantile diarrhoeas.

Such, then, is a brief review of the causes of the enteral group of diarrhoea in infants. An attempt has been made to trace the evolution of the bacteriological aspect of the subject.

The diet of the infant has also, from time to time, been suspected as being a cause of outbreaks of infantile diarrhoea. Czerny (1906) in collaboration with Keller, published his researches into nutritional disorders of infancy, which cast an entirely new light on the prevalent ideas of that time. He pointed out that disorders of nutrition were not only associated with the gastro-intestinal tract, but that the infant as a whole must be studied and surveyed. Czerny stated that these/
these disorders of nutrition might be classified under 4 headings: (1) Those caused by food. (2) Those caused by infection. (3) Those due to constitutional anomalies. (4) Those due to congenital malformations. From these various causes, Czerny stated that 2 states of disordered nutrition might arise. These 2 states he called Milchnahrschaden and Melnahrschaden.

In the former state, Czerny saw the ill effects of too much protein and fat, and too little sugar. The infant thus affected was fretful and restless, the weight was stationary or falling, and the stools were large, pale, dry and alkaline. In particular did Czerny see the ill-effects of too much fat, and in fact, he later exonerated protein from any blame as a cause of the condition. This latter finding was in keeping with opinion in this country.

In Melnahrschaden, too much sugar was considered by Czerny to be the cause of the condition. The protein and fat were usually normal in amount. In this state of disturbed nutrition, the infant suffered from diarrhoea, with the passage of loose, large, frothy stools, acid in reaction. These stools burned and excoriated the buttocks. There was also loss of weight and often vomiting.

The essence of Czerny's theory was, that, while infection, constitutional anomalies and other conditions in which normal food disagrees, might give rise to nutritional/
nutritional upsets, the real and important cause was that the components of the diet, especially the fats and sugar were given in the wrong proportions.

Czerny was closely followed in this new field of investigation by Finkelstein (1909). This worker, in contradistinction to Czerny, saw, not the proximate principles of the diet at fault, but the mineral salts. As the mineral contents of the milks of the various species of mammal differed, it seemed logical to assume that the intestinal mucosa would function par excellency, when the fluid medium was as Nature intended it. Meyer (1908) has shown that when cow's milk curd and human whey, and human curd and cow whey mixtures were fed to two different groups of infants, the results were almost identical. In fact, many of the infants fed on the cow-curd and human-whey mixture did better than those fed on the human-curd and cow-whey mixture. These latter infants often had green undigested motions. From these experiments, it seemed that Finkelstein's theory was based on sound principles.

Like Czerny, Finkelstein saw certain stages in the development of nutritional disorders. One stage might pass into the next, suddenly or slowly, depending on the circumstances peculiar to each infant. 4 stages were described by Finkelstein. (1) Disturbance of Equilibrium. This corresponded to Czerny's Milchnahrschaden. The infant's weight remained stationary, or tended to fall, the stools were dry and infrequent, and the baby more liable to secondary infections. (2) Dyspepsie/
(2) Dyspepsia. In this stage, definite loss of weight occurred, the stools were loose with slight diarrhoea, and the infant peevish and irritable, but not actually ill. This stage corresponded to Melaunarschaden.

(3) Alimentary Decomposition. There was usually diarrhoea in this stage, with great and progressive loss of weight and a subnormal temperature. Finally (4) Intoxication, in which there was great loss of weight, severe diarrhoea and frequent vomiting. Disturbances of nervous function were also present, with muscular twitchings, attacks of unconsciousness, from which, at first, the infant could be aroused, to lapse again after removal of the stimulus. Death finally stole over the scene.

These observations of Czerny and Finkelstein were great landmarks in the advance of knowledge in infant nutrition. Their theories are now, in large measure, abandoned. The importance of their observations lies in the fact that they emphasised, as never before, the importance of considering the child as a whole, and not merely focussing attention on the gastro-intestinal tract.

The constitutional make-up of the infant is a factor which cannot be disregarded when considering the aetiology of infantile diarrhoea. Certain constitutional anomalies lead to lessened food tolerance, and in particular is this shown in the so-called exudative diathesis. The infants with this anomaly are particularly liable to bouts of diarrhoea, precipitated by/
by minor dietetic errors or indiscretions. A noteworthy feature of these cases, e.g. those with eczema, is that during the acute stage of the diarrhoea, the skin condition clears up as if some magic balm had been applied to the skin, to return so soon as the diarrhoea ceases. This phenomenon I have witnessed in two bottle fed infants. One of these cases is included in the present series. A form of sensitivity or allergy to certain articles or constituents of articles, of food, in particular, cow's milk, must be a very important factor in these constitutional cases (Ratner and Gruehl, 1935). I have observed a female infant who, on weaning from the breast to cow's milk, at 8 months of age, showed a very severe reaction to its first feed of boiled cow's milk. The reaction took the form of severe sweating and subnormal temperature. The child only slowly recovered from this shock, but later, even 1 teaspoonful of cow's milk produced diarrhoea and abdominal discomfort. It was interesting to find on enquiry, that the child's mother could not take milk in any form without suffering from an attack of asthma.

In another case, also not included in this series, a healthy breast-fed male infant of 6 months of age, was suddenly weaned on account of illness in the mother. This infant also showed an intolerance to cow's milk and had to be weaned on Almata. Later cow's milk was again introduced into the diet in small amounts, with no/
no apparent ill effects. When 11 months old, however, progress became retarded, and the weight steadily fell for the ensuing 6 weeks, in spite of every effort to stop the fall. Suddenly, in the early days of the 14th month, the child was stricken with a convulsion from which it never recovered. No abnormality was found, either ante-mortem or post-mortem. This case seemed to be one of either an acute fulminating infection of unknown type or more likely, was a condition of constitutional imbalance of unknown type.

Hypertonicity and Hypermotility are also alleged to occur in certain infants. Infections also seem to upset the balance of the Autonomic Nervous System (Pearson and Wyllie, 1935). In much the same way, constitutional disturbances such as rickets, are often associated with diarrhoea with or without vomiting. Such upsets clear up on the exhibition of suitable doses of the appropriate vitamins, and rectification of the diet. A parenteral infection is often the actual precipitating factor in these cases of diarrhoea.

How far the commonly stated predisposing factors in diarrhoea in infants, may be the actual causal agents, is very difficult to say. The effects of hot weather, high humidity, poor ventilation and overclothing are all recognised as well defined predisposing factors. In particular do they predispose to the summer form of diarrhoea. The extent of their influence in precipitating or causing the actual onset of symptoms, is not yet/
yet known.

Overheating, whether from clothing, or due to meteorological conditions, as noted by Ballard in 1885 and confirmed by Paterson and Nabarro (1922), has a well defined influence on the gastric and intestinal secretions of the infant. The general effect produced by overheating, is a depression of these secretions. Hydrochloric acid in the stomach is decreased, as is also the total volume of gastric juice secreted. This may allow of infection, contained in the milk, to pass unimpeded into the normally sterile duodenum and upper jejunum, and produce an infection there. Similarly, by depression of the secretion of the hydrochloric acid in other cases under similar meteorological conditions, the normal acidity of the duodenum and upper jejunum is decreased, or may even become alkaline. Organisms normally resident in the ileum and lower jejunum, may therefore spread upwards, and by their multiplication in this abnormal site, produce effects on the body. Such effects may be either absorption into the blood stream of these organisms, causing a septicaemia, or else absorption of the toxins produced by the bacilli, with resulting toxaemia. This results in the occurrence of both local and general effects, for it will be noted that in the areas of bowel where absorption is maximal, bacterial activity is minimal or absent.

Parsons (1933) has shown that anacidity and achlorhydria commonly occur in the artificially fed infant, which/
which is puny or suffering from some infection. The production of this anacidity is due to either infection, be it enteral or parenteral, or to too rapid emptying of the stomach. Usually, however, in such cases where infection is present, hydrochloric acid can be shown to be present by injection of histamine (a procedure not free from risk in such cases). Measuring total chlorides of the gastric contents is a safer method. Neale (1930) showed that in no case did he ever fail to obtain hydrochloric acid after injection of histamine, in cases which had previously shown no acid. Most observers, save Hurst and others, agree that both anacidity and achylia are not congenital.

Marriott (1930), in his researches into gastric digestion, found that cow's milk had a high buffer action. He noted that the stomach of the infant was often incapable of producing sufficient acid for optimum digestion. A further observation, stressed by Marriott, was that micro-organisms in the ingested milk might escape destruction, because of insufficient concentration of acid. In this way, he suggested, gastrointestinal disturbances might be initiated. Marriott introduced his lactic acid milk to combat this high buffer action of cow's milk. Experience has amply demonstrated the efficacy of this milk in nutritional disorders of infancy.

It/
It is probably because the gastric juice of infants contains less hydrochloric acid than older children, that they are more liable to develop diarrhoea. When human milk is given to an infant, an acidity still remains in the upper part of the small intestine, and this acidity is maintained, thereby keeping this section of the bowel free from organisms. This very largely explains why breast fed infants rarely suffer from the severer forms of diarrhoea, in particular the infective enteral type. Infection of the intestine may, however, occur in the breast fed child, if the mother is careless in respect of the cleanliness of her nipples, or in the use of dirty contaminated comforters. In these cases, if harmful germs are swallowed, especially if the infant is weak or improperly fed, they may get past the weak acid barrier of the stomach, and flourish and produce their effects in the bowel. The danger in these children is great, since under such circumstances, the acid secreted is diminished.

Overfeeding, by overtaxing the digestive organs or feeding with excessive quantities of indigestible material, may also produce diarrhoea. Such foods as contain excess sugar and/or fat, are particularly liable to cause an intestinal upset with diarrhoea and often vomiting. These symptoms are due to fermentation, with the production of abnormal acids which irritate the intestinal mucus membrane. Experience, however, shows that/
that an excessive acidity, or abnormal acids in sufficient amount are necessary for the production of the diarrhoea.

A dietetic fault can be found in a large number of cases of infantile diarrhoea. This fault may be due to underfeeding, to overfeeding, either of normal foods, or of improper foods. The cases of diarrhoea due to overfeeding with normal foods, are most liable to occur in the warmer months. At that time, the calorie intake of the infant requires to be lessened, and, coincidently, the intestinal juices are also secreted in less quantity. In particular, is the fat of the diet difficult of digestion, and the continuance of a high fat intake, which can be well borne in the winter, will, if continued for long in warm weather, result in a breakdown in the digestion. This breakdown will be more marked and more rapid in the less robust infant than in the sturdier one. I firmly believe that feeding on rich cow's milk mixtures in warm weather, is a cause of diarrhoea. Proof of this contention is, I suggest, to be found in the improvement brought about by using skimmed milk mixtures in such weather. I have employed weakened feeds both prophylactically and curatively with satisfactory results.

Mention has already been made on the effect of increased temperature on intestinal secretions. Infections in any part of the body, associated with a temperature, often cause a bout of diarrhoea. This brings/
brings us to a consideration of the third group of cases of diarrhoea, viz., those cases associated with the presence of infection outwith the bowel - the so-called Parenteral group.

Certain of these infections have a greater effect on the functions of the alimentary canal than others. Thus otitis media, rhinopharyngitis and tonsillitis, produce more profound disturbances of digestion than e.g. pneumonia, though the temperature attained by these conditions may be similar. Renaud (1921) demonstrated the frequent finding of pus-like material in the ears of infants dying from diarrhoeal conditions. While a few of these fluid collections were due to post-mortem degeneration was admitted, (Alexander, 1927) in most instances there was actual infection of the ear cavities. That an infection was indeed present was shown by the finding of pus cells, and often haemolytic streptococci.

These observations aroused great interest, and stimulated research into the relationship, if any, between infections, aural in particular, and infantile diarrhoea. In other words, there was a recussitation of the old observation, mentioned incidentally by Rush, that the removal of a septic focus cured another existing disease. Rush had noted the beneficial effect of removal of septic teeth in rheumatic cases. Attention was accordingly directed to determine the influence of infection in other parts of the body, on the production of diarrhoea. The literature which has now appeared since/
since Renaud made his observation, is almost forbidding in its extent. Obviously only a few of these opinions can be reviewed.

Floyd (1925), Jeans and Floyd (1926) and Holsclaw, Boehm and Bierman (1930) considered, after very careful investigation of their cases, that gastro-intestinal disturbances were related to otitis media, and were due to toxaemia from infection of the mastoid air cells. Asherton (1932) described the post-mortem findings of a case of infantile diarrhoea, associated with bilateral otitis media. The post-mortem revealed that the mastoid air cells were also filled with pus. Le Mee (1937) pointed out that otitis media in infants is always associated with a mastoid infection. He accordingly prefers the term oto-mastoiditis rather than otitis media.

Marriott (1928) stated "Otitis media is the most frequent infection responsible for nutritional disturbances in infants". In the work of this investigator, and also in that of Jeans and Floyd, the streptococcus seemed to have been the organism most frequently found in the otitis media associated with their cases. Rominger (1935) stated that of 500 cases of nutritional disturbance in Kiel Children's Clinic, 61% were due to parenteral infection of some sort, and Anderson (1938) drew attention to the frequency of respiratory infections as a cause of gastro-intestinal symptoms. Ebbs (1937) believed that an infection anywhere in the body/
body of an infant with low powers of resistance, is capable of producing the diarrhoeal syndrome. Ebbs, in an analysis of 880 cases post-mortem, revealed that 238 children under the age of 2 years, suffered from diarrhoea and vomiting, which was either the major complaint, or was a complicating factor in the cause of death. 91.6% of these cases had a parenteral infection, which was in some instances, otitis media. Blacklock, Guthrie and Macpherson (1937) gave the percentage as over 90% in their cases of gastro-intestinal upset as due to otitis media.

Ebbs, in his very valuable paper, stated that otitis media may not always be the causal condition, but may be the factor deciding life or death. Adams (1937) believed that the common cold was often the initial cause of a major disaster in infancy. He showed, in his paper, a most interesting chart of the cases of otitis media requiring paracentesis in the Children's Hospital, Birmingham, from January 1935 to March 1937 inclusive. Though these figures of Adams include all those of children up to the age of 12 years, more than one-third represented infants under 2 years. He found that the period of maximum incidence of otitis media in his cases over that 2 years' period, was the first quarter of the year, viz. January to March, in both years observed. The same finding seemed to be in the process of demonstration in 1937. This observation is
of great significance, in that it shows that if otitis media is such a potent factor in the production of diarrhoea in infants, as it is alleged to be, then it should show also its maximum incidence at the same time as infantile diarrhoea is at its maximum, viz, the summer months, July-September. Statistics on the incidence of diarrhoea in infants, still show a slightly greater prevalence of the condition in the warmer months, than in the colder, wetter months. Within recent years, however, the tendency of the diarrhoea-vomiting syndrome has been seen to be less marked in the summer months. The result of this observation is that the curve of incidence is continuing to become flatter. It still holds good that the so-called infectious type of diarrhoea is more prevalent in the warmer months, than in the colder ones. This tends very strongly to disprove that all cases of infantile diarrhoeas, the proved specific infective type excepted, are caused by parenteral infection, especially upper respiratory infection.

McConkey and Couper (1938), in an analysis of their cases of mastoiditis and otitis media in infancy, found confirmation for the theory held by other workers, that the diarrhoea and vomiting syndrome was frequently due to otitis media and latent mastoiditis. The death rate of their series, thus affected, was 84.8%, the highest rate being evident in the age group, 1-3 months. Baber (1939) was convinced that the more thorough was the search for a focus of infection in cases of diarrhoea in/
in infants, the more frequently will such be found to be responsible for the condition. Baber found that 67% of her cases were associated with parenteral infections.

As has been hinted above, not all observers are agreed that parenteral infections are the major causes of infantile diarrhoea. The opinions of these investigators seem to be called for. Again only the views of a few of them can be given here.

Wishart (1926 and 1930) thought that otitis media and mastoid infection were secondary developments, and not the primary causes of the condition. Brown et al (1930) found, in a series of 171 cases of intestinal intoxication examined on admission to hospital, that 44 had otitis media, while 65 others developed otitis during the course of their illness. In all some 63.7% of diarrhoeal cases had otitis. They also found that 63.1% of 84 control cases had otitis, and that there was a higher percentage of streptococci in these, than in the cases with diarrhoea. They came to the very definite conclusion, that neither otitis media nor streptococci were responsible for the infantile diarrhoeal syndrome. A similar conclusion was reached by the same workers later (1933). Maybaum (1932) and Poole and Cooley (1932) felt that otitis media and mastoiditis were not important aetiological factors in the production of the diarrhoeal syndrome in infants. Maizels/
Maizels and Smith (1934) thought that otitis media was rarely a primary cause of intestinal intoxication.

Yampolsky (1933), in a survey of 500 cases, came to a similar conclusion, viz, that otitis media was not a cause of the infectious diarrhoea and vomiting of infants. Findlay (1932) was also very sceptical of the relationship, if any, between the diarrhoea-vomiting syndrome and otitis media and mastoiditis. He reached the conclusion that parenteral infection, such as an area of focal sepsis, especially otitis media, was not a serious responsible factor. In fact, Findlay went so far as to say that surgical intervention in some of his cases, had definitely prejudiced their chances of survival. The death rate, however, from infantile diarrhoea is still so relatively high, even in uncomplicated cases, that the suggestion that surgical intervention in complicated instances had prejudiced the chances of recovery, seems to me to be very doubtful, both as a statement to make and as a conclusion to reach. The question of when to operate in such cases is the really fundamental point, the essential feature being early interference, and not, as so often happens, a last desperate measure to save life.

Marriott (1933) noted that parenteral infections complicated the clinical picture in 66% of cases of true dysentery. The mortality rate in such cases with secondary infections was 28%, but only 10% in those uncomplicated/
uncomplicated by such infections.

It does seem, however, that parenteral infection is, by general concensus of opinion, a potent factor in the production of infantile diarrhoea. It is not the only cause of the disease as we have seen. Dietetic faults, and intestinal infections, whether from pathogenic organisms or saprophytes living in abnormal areas of the bowel, are recognised to be causes of the condition.

Classifications of the causes of infantile diarrhoea are based on these three factors. Many classifications have been made (Carmack, 1930, Ebbs, 1937 and Smellie, 1939), but the one indicated by Graham (1938) seems to me to be the most suitable, as indicating the factors at work. This classification was that given at the commencement of this section.

The first group, consisting of those cases due to infection in the bowel, is a relatively small one. In the majority of these cases, the infection is unknown. In a small proportion, however, well defined pathogenic organisms can be isolated. These microorganisms are usually of the dysentery group of bacilli, but the typhoid group have been isolated on occasion. The dysenteric cases produce a rather typical clinical and pathological picture, to which the name of Ileocolitis has been given. The infectivity of these dysenteric, typhoid and food-poisoning group of cases is high in comparison with the low infectivity shown by the cases of non-specific infection in the bowel.

Mention must be made, when considering this small group /
group of cases due to non-specific or specific infections, of those instances where the causal organism has been the Staphylococcus. The diagnosis of this form of infective diarrhoea is very difficult, as the bacteriological tests are beset with considerable difficulties in their interpretation. Brooks and Tiedeman (1937) referred to this form of infective diarrhoea, and discuss the possibilities of such an infection as a cause of outbreaks of enteritis in both adults and children. Shaughnessy and Grubb (1937) have described the investigation of outbreaks of Staphylococcal diarrhoea due to milk infected from cows suffering from mastitis of staphylococcal origin.

In my own series of cases, only 25 were judged as being infective in origin. Of these 25 instances, in only 6 cases was a pathogenic organism isolated, the B. Dysenteriae Sonne in 5, and B. Dysenteriae Flexner in 1. In the remaining 19 cases, no pathogenic organism could be isolated, the organism most frequently present being the B. Coli Communis. In no instance in these 25 cases was the infant breast fed at the time of infection.

The acute summer form of infantile diarrhoea was conspicuous by its absence in this series, which, I feel, is fairly representative of the cases attending the Infant Welfare Centre. This observation does not imply that the condition does not exist. It undoubtedly does, but/
but it would seem that the form now appearing is much milder than formerly, and incidentally more amenable to treatment. The Cholera Infantum of the older writers has, fortunately, practically ceased to exist.

The second group, consisting of those cases due to dietetic causes, is larger than the enteral group. A dietetic error or indiscretion can usually be found in this group. Such faults may be overfeeding, or less commonly, underfeeding, or the feeding of infants on unsuitable articles of diet. Overfeeding is certainly more common in the breast-fed child than in the artificially fed one. In my series, I did not find either overfeeding or underfeeding in breast fed infants a common cause of diarrhoea. The giving of unsuitable foods or articles of diet was the commonest cause in the cases I encountered, and this applies almost exclusively to the artificially fed infants.

Intolerance to cow's milk - an uncommon event - and other constitutional anomalies may conveniently be classified under this dietetic group.

In 82 of this series of cases, a dietetic cause was considered to be the aetiological factor in the production of the diarrhoea. Infection was absent from all, and a feeding upset was the only cause which could be found to account for the diarrhoea.

The third group, containing those instances of diarrhoea/
diarrhoea associated with a parenteral focus of infection, is but imperfectly understood. The incidence of this type of case is increasing. This increase is either real or apparent as a consequence of the fall in the enteral and dietetic groups, which has been observed within comparatively recent years. The reason why an infection outside the bowel should produce diarrhoea, is unknown. From the mass of evidence available, it is impossible to escape the conclusion that a parenteral infection is a common cause of diarrhoea in infants. Infections in certain parts of the body seem to produce this intestinal upset more readily than infections on other areas. In particular does an upper respiratory infection, with or without otitis media, tend to predispose to the onset of diarrhoea. In such cases, the diarrhoea is simply an indication that the baby has 'caught a cold'. As we would expect, this class of parenteral infection occurs more noticeably in the young infant and in the colder, wetter months. Dolce (1937) and Macgregor (1939) have produced evidence that these infections of the respiratory tract are most frequently contracted from the mother, and point out the dangers resulting from such close contact between mother and child.

Toxaemia, severer in one type of infection than in another, has been suggested as a cause of the diarrhoea. The constitutional make-up of the child is probably also important/
important, as helping to explain why one child should be affected with diarrhoea, and another not so affected, though both are suffering from a similar infection. The swallowing of infected material from the nasopharynx may also act as an intestinal irritant. The diarrhoea resulting from parenteral infections, in general, is usually milder, and dehydration less acute than obtains in those cases of primary bowel infections, and in certain of the dietetic cases.

The breast fed baby is not nearly so immune to this form of diarrhoea, as it is in the other groups. The infection is usually 'caught' from the mother or other person in close attendance upon the infant. 20 infants in this series of mine were breast fed and suffering from some form of parenteral infection, which was judged to be the cause of the diarrhoea. In a further 8 cases, the feeding was partly breast with a complementary milk feed.

The frequency of otitis media in infants seems to be due to the child spending most of its time in the recumbent position. The Eustachian tube is short, has a larger calibre than in the adult, and the isthmus is non-existent. The absence of the latter results in the contractions of the muscles of the soft palate having an influence on the whole tube. The mastoid air cell, middle ear and Eustachian tube form one single cavity (le Mee 1937). Hence any infection in the nasopharynx with the child in the recumbent position, predisposes/
predisposes to infection spreading upwards to the ear. This latter event results in an oto-mastoiditis, or as it is more commonly called, simply otitis media.

In all, 143 of this series of 250 cases of infantile diarrhoea were classified as belonging to the parenteral group. Upper respiratory infection accounted for 68 cases. In these 68 instances there were never any indications of aural involvement, but otitis media was present in a further 22 cases. These latter cases usually showed some evidence of naso-pharyngitis. In 11 of the cases of otitis media, the infection was bilateral.

Other infections, e.g. pyelonephritis, meningitis, erysipelas and osteomyelitis, to mention only a few, may similarly cause diarrhoea. Again the mode of production of the diarrhoea is unknown. These infections constitute but a very small proportion of the diarrhoea-producing infections. There is general agreement that upper respiratory, with or without otitis media, is the commonest of the parenteral infections causing diarrhoea in infants.

In the present series under consideration, the main incidence of all cases was found to occur in the first quarter of the year. There was a tailing off in the summer months, and a slow rise to peak level again in the first quarter of the following year. This observation would tend to confirm that parenteral infections are the commonest cause of the diarrhoeas as met with at an Infant Welfare Centre.
PATHOLOGY OF INFANTILE DIARRHOEA.

Diarrhoea, if severe or prolonged, may result in serious damage to the body, or in death. In all diarrhoeal states, there is an excess loss of base in the motions. This depletes the store of Alkali available to neutralise the acid substances produced in the ordinary course of metabolism. In the combined total secretion of the gastro-intestinal tract, bases predominate over acids in the proportion of 3:2. Failure, therefore, to reabsorb the intestinal secretions will result in this excess loss of base from the body.

There is also diminished absorption of food which results in a state of starvation. Starvation in its turn, produces an excess of ketone bodies. Toxins absorbed from the intestinal canal may play a part in damaging the tissue cells. Fever, when present, disturbs the catabolic processes, so that excessive tissue breakdown takes place, which further increases the acid substances in the tissues. Most important of all the ill effects of diarrhoea, however, is the loss of water from the body, a condition commonly called Anhydraemia.

Water absorption in diarrhoeal states is poor. This accounts for the looseness of the motions. The amount of water lost in this way may be very large, and at times, equal or even exceed the total fluid intake. The reduction of the body fluid below a certain minimum, results in the body making strenuous efforts to correct the/
The chief result in the dehydrated state, is the loss of tissue fluids, with a lowering of the total blood volume. The essential change takes place in the plasma fraction, the cell volume remains constant. The reduction of plasma volume may reach one-third of the original total amount.

The symptoms of dehydration or anhydremia, are largely dependent on this decrease in the volume of the blood. There is loss of body weight, which may be extreme and often rapid. The features of the infant become sharpened, and the eyes sunken, and in severe cases, staring. The conjunctivae lose their normal lustre. The fontanelle becomes depressed, and the skin becomes grey and loses its normal elasticity. There is arteriolar constriction, with consequent accumulation of blood corpuscles in the capillaries. The capillary blood count is therefore often high. The arteriolar constriction is due to the diminished blood volume. The mucous membranes of the lips and mouth become dry and marched, the mouth is held open, and the tongue dry and often red and raw looking.

The blood is thickened and does not flow easily. This results in a slowing down of the circulation, though the pulse may be rapid, and often irregular. The plasma protein is high, and the leucocytes increased, i.e. a leucocytosis may be present. There may also be an increase in the red blood cells, but this increase is/
is only relative, and is due to the concentration of the blood. However, a true anaemia may be present.

Consequent upon the poor circulation, especially through the kidneys, the urine becomes scanty concentrated, contains albumen in varying amounts. Sometimes casts are found, and the granular variety is the most frequent type met with. This is in keeping with the frequent finding, post-mortem, of parenchymatous changes in the kidney closely simulating a nephritis. Occasionally the urine reduces Fehling's solution.

The Non-Protein Nitrogen (N.P.N.) is increased, and uraemic figures may be found. Blood Chlorides are raised in many instances, more particularly in the severest cases. (Hartmann, 1928, Schoenthal, 1929, Hamilton et al, 1929).

The blood bicarbonate is decreased in almost every case, and the reduction is often extreme. Values as low as one-fifth of the normal amount have been recorded. With this lowering of blood bicarbonate, the symptoms of acidosis make their appearance. The breathing becomes deep, regular, and the accessory muscles of respiration come into play. Thoracico-abdominal breathing takes the place of the normal abdomino-thoracic respiration.

Nervous phenomena are often terminal, ushering in death. Convulsions and muscular twitchings are the commonest manifestations.

When diarrhoea is severe, acidosis is constantly present/
The occurrence of acidosis is more rapidly produced if there is much dehydration. Owing to the excess loss of base compared with acid, in the stools, there is a lowering of the basic ions in the blood plasma, as noted supra. The kidneys are the only organs capable of excreting fixed acids. When there is much anhydraemia, the secretion of urine is considerably decreased. Consequently, less acid is removed from the body. This acid accumulates in the blood, causing further depletion of the alkali reserve. As a result of the poor circulation through the kidney, the normal function of that organ to secrete these fixed acids combined with ammonia, as ammonium salts, is greatly impaired. Further demands are therefore made on the alkali reserve.

Because of tissue anoxia, of the stagnant type, consequent upon the impaired circulation, lactic acid and the other organic acids formed in the tissues during catabolism, are set free. They still further deplete the bicarbonate reserves.

Lastly, ketosis may be superimposed on the acidosis. This ketosis is due to the partial starvation resulting from impaired absorption of food, or from lowered food intake imposed as a therapeutic measure. Aceto-acetic acid and Beta Hydroxy-butyric acid are produced and more plasma bicarbonate is required. The ketosis, however, is not a very important factor
in contributing to the acidosis associated with diarrhoea.

In certain diarrhoeas, especially those associated with a profound toxaemia such as may occur in Shiga dysenteric infections, little or no diarrhoea may occur. The severe toxaemia produces in such cases, a fatal outcome ere diarrhoea becomes very manifest.

We may summarise the foregoing brief review thus. Acidosis is present in most cases, to a greater or lesser degree. The acidosis is due to a lowering of plasma bicarbonate. Several factors are probably involved in the production of the acidosis:

(1) Owing to the decreased water content of the blood, the circulation through the kidneys is slowed down. Renal function becomes, as a consequence, imperfect, and fixed acids accumulate in the blood and deplete the alkali reserve. Phosphate retention may occur.

(2) Because of tissue anoxia (stagnant type) lactic acid and other organic acids accumulate in the blood. These acids are derived from incomplete oxidation in the tissues.

(3) A certain degree of ketosis is present due to starvation.

(4) An excess of base, compared with acid, is lost in the stools.
MORBID ANATOMY OF INFANTILE DIARRHOEA.

The time elapsing from death, especially in warm weather, to the examination on the post-mortem table is of considerable importance, since post-mortem changes take place rapidly between death and examination. Hence the examinations must be carried out as soon as possible after death.

Intestinal Lesions.

In some cases, the intestines appear perfectly normal. In others, often those with a previous history of indigestion, a condition of catarrhal enteritis is present, with much mucus. Many cases show simple congestion of the mucous membrane, with or without petechial haemorrhages. Definite ulcer formation is quite common, the ulcers being either very small, often situated along the mesenteric attachment, or larger, punched out ulcers, or in a few instances, shallow, irregular ones. So-called Membranous Colitis may be found, when the superficial layer of the colonic mucous membrane appears to be sloughing in large patches; these sloughs may be grey, or bile stained. Frequently, in the small bowel, the surface of the mucous membrane is roughened, with numerous small erosions and petechial haemorrhages. As the caecum is approached in these cases, the changes may become more gross, with quite large haemorrhages present, the lymphoid aggregates being prominent and often/
often eroded. Such a change may also be present in the large gut, but may be absent, or only present in lesser degree, and confined to the first part of the colon.

Microscopically, in the milder cases, the changes are confined to the mucosa and submucosa, which are infiltrated with polymorphonuclear leucocytes. Oedema may be present, but this is most prominent in the dysenteric cases. In the membranous variety, the membrane is made up of necrotic mucosa, in which all structure is lost, together with a varying amount of fibrin and inflammatory cells. The submucosa is thickened, hyperaemic, often oedematous, and usually shows small haemorrhages. The sloughs, and the tissue immediately underlying the sloughs, are packed with bacteria of various kinds. In the dysenteric forms, the colon is more affected as a rule, than the lower ileum, but both are affected. Here oedema is quite a marked feature, and is responsible for the great swelling of the submucosa. Large members of bacilli can be demonstrated in the necrotic sloughs, and even in the submucosa and muscularis.

Mesenteric Glands.

In many cases, these glands show none, or at most, very slight enlargement. In most cases, however, of the dysenteric type with ulceration and colitis, the glands are enlarged and packed with inflammatory cells. Bacteria may even be demonstrated in these glands on occasion.

Liver.
Liver.

This organ is very often the one which shows the most obvious changes. The organ is not usually enlarged. It varies in colour from a pale red to a bright yellow colour, the latter being either uniform or irregular in distribution. When the yellow colouring is extensive, it is rather characteristic of the acute infectious type of infantile diarrhoea. Section reveals a firm organ, with colour changes as noted.

Microscopically. The liver cells show, in the mild cases, a degree of cloudy swelling and fatty degeneration. In the severer forms, the liver cells are distended with fat globules, so that the cells tend to be spherical in shape, with the nuclei flattened against the cell membrane. The fat is commonly evenly distributed throughout the cell, but in the less severe instances, the fat may be found mainly in the centre of the liver cell. In some cases, the liver cells are themselves degenerated, and both cell membrane and nucleus stain poorly.

The cause of these grosser changes in the liver is still obscure. It does not appear to be due to the starvation regime instituted in the first 12-24 hours of treatment, as glucose is usually administered during that period. Further such changes are not observed in the livers of infants dying from pyloric stenosis, where starvation may be extreme. A toxic theory seems to/
to be the only one which would explain the changes, but from what, or from where, the toxin is manufactured, is unknown.

Kidneys.

These organs often reflect the damage done to the liver, and when the latter is severely damaged, the kidneys show similar changes. The change is at first, one of cloudy swelling, noted in the milder instances. This may go on to the severer degeneration, viz. fatty degeneration. In some cases, the parenchyma is damaged to such an extent that a true parenchymatous nephritis is simulated, with swelling and desquamation of the cells of the tubules, the cytoplasm and nuclei taking on the stain badly or not at all. Small haemorrhages may be noted microscopically. Urine examination often bears out the pathological picture, as albumen and epithelial casts, and even fatty casts may be found.

Spleen.

This organ may show very little change. In some instances it is enlarged, and deep red in colour.

Microscopically. There may be marked proliferation of the endothelial cells lining the sinuses.

Heart.

The change noted here is mainly one of cloudy swelling. Rarely there may be evidence of fatty degeneration. If the case has been one of chronic wasting with/
with superadded acute diarrhoea, the typical picture of a 'marasmic' heart may be found - tortuous vessels, and markedly wasted muscle. Small petechial haemorrhages may be noted under the pericardium.

**Lungs.**

These organs usually show some change. Petechial haemorrhages are especially common in the severely toxic cases. Congestion of varying degree is very common, and a broncho-pneumonia is a not uncommon finding.

**Brain.**

A normal appearance is usual. In some cases, there is evidence of congestion with the surface of the brain covered with small petechial haemorrhages. Gross damage to the brain is uncommon. Thrombosis is probably the commonest of these gross signs. There is usually associated with these thromboses, haemorrhage into the adjacent brain substance.

Finally, there may be evidence of infection in the ears or in the sinuses, as has been emphasised in the discussion on aetiology.
SECTION III.

SYMPTOMS AND SIGNS.
Before beginning a discussion on the clinical features of Infantile Diarrhoea, it would probably be wise to recapitulate the classification adopted in this thesis. It was the classification made by Graham (1938), and it seemed to meet the requirements of all types of case likely to be encountered. The three groups of cases were (1) Enteral, (2) Dietetic and (3) Parenteral.

The parenteral group constituted, by far, the largest proportion of the cases under discussion in this thesis. Consequently, it is probably desirable that the clinical features presented by this most important group should be described at the outset. Though, as the name of this group implies, infections outwith the alimentary tract are the actual causes of the diarrhoea, it must not be forgotten that a careful enquiry should be made in all cases of diarrhoea in infants, as to the presence of acute catarrhal infections especially of the upper respiratory tract, in the attendants of the infant.

**PARENTERAL DIARRHOEA.**

Onset. This is, as a rule, sudden, but occasionally it is more gradual and no definite time can be stated as to the actual commencement of the illness.

Vomiting. Vomiting is one of the first symptoms usually noted. It is not frequent, in fact it may occur on only one occasion, viz. at the very onset of the condition. It is, however, more marked and more frequent/
frequent and troublesome in those cases of manifest otitis media and tonsillitis, associated as these conditions usually are, with a degree of pyrexia ranging from 101-104°F, or more.

In the mild cases of this group, vomiting may be entirely absent, and never noted throughout the whole course of the illness. The vomiting, when present, is usually followed by evidence of an infection, such as a rhinitis, with watery discharge from the nose, snuffling and occasionally a short, dry, harsh and barking cough.

Convulsions. One or more convulsions may usher in the illness, when the latter is of sudden onset. This mode of onset is not very common. Noted most in cases of otitis media.

Anorexia. Loss of appetite is a common, in fact constant, feature, associated noticeably in those cases with a fair degree of fever. The infant refuses all feeds, whether breast or bottle, but takes water well. In the milder forms of the disease, the appetite may be merely impaired.

Tongue. The tongue is usually furred, often thickly so. This furring is noted especially in the posterior third. The mouth is not, as a rule, dry or parched unless temperature is very high or dehydration is severe.

Diarrhoea. The onset of diarrhoea often occurs coincidently with the development of anorexia. It is to/
to be especially noted that the diarrhoea commences after the infant has begun to show signs of a feverish illness. The diarrhoea usually begins gradually, and the mother may not draw attention to the slight increase in the number of the stools, but rather to their increasing greenness. At first, these stools are a little greenish-yellow, with here and there a whitish lump of fatty curd. Many cases remain at this stage and progress no further, the diarrhoea beginning to improve on the institution of treatment.

In other cases, and in the more severe ones, the diarrhoea progresses, the stools become more frequent, the colour a darker green, the fatty curds more conspicuous against the darker background, and the odour more offensive. Whether the baby is breast fed or artificially fed, the motions are usually acid. The acidity in the artificially fed infant is probably due to the fermentation of carbohydrate contained in the feeds.

The diarrhoea may progress still further, in the more severe cases, till the motions rapidly become fluid, brown in colour, odourless and dehydration becomes an urgent feature of the condition.

**Buttocks.** The skin of this area tends, even in a short time, to become reddened and irritated. Excoriation may also occur, especially if scrupulous cleanliness is not the forte of the mother. The inflammation around/
around the buttocks is due to the irritating effect of the abnormal stools.

**Weight.** This always shows some aberration. There may be merely a failure to put on weight, but no actual or apparent loss. This occurs in the very mild cases only. Actual loss of weight is more commonly present, the degree of loss varying considerably, being largely dependent on the severity of the infection, but also on the duration of the illness prior to being seen. The loss of weight is due to a combination of factors. These factors include, loss of fluid in the motions, reduced intake of food and fluids, and to the toxaemia of the infection.

**Dehydration.** This is not greatly marked in the mild cases, but slight degrees of this state are often present on clinical examination. The fontanelle becomes, at first, less tense than normal, and if the dehydration be more advanced, the fontanelle becomes actually depressed, loses its normal consistence and pulsation. This latter state of the fontanelle is evidence that the dehydration is severe. Such a state of the fontanelle may occur in cases, originally mild, but neglected, or in those which have a very acute onset, with high fever, vomiting and diarrhoea.

Signs of dehydration are not, however, confined to the fontanelle. The eyes may be sunken and staring, and the skin on the inner sides of the thighs and of the abdomen/
abdomen may show an impairment of its normal elasticity. This impaired elasticity is best demonstrated by pinching up the skin between the finger and thumb, when it will return but slowly to its normal contour.

The abdomen may be soft and collapsed. It also appears to be often tender on pressure.

**Temperature.** The temperature varies in degree, ranging from 97 - 104 F. or higher. The subnormal temperature occurs in the marantic or physically poor child. Fever, however, is present in most cases, and frequently the higher levels of pyrexia occur in cases of otitis media, pyelo-nephritis, etc. Apparent simple naso-pharyngitis, with no obvious involvement of the ears, often produces a remarkably high degree of fever.

**Pulse and Heart.** There is tachycardia in all cases, the pulse rate ranging from 120-160 beats per minute. The rate depends on the severity of the condition. In the severe cases, the pulse may be irregular, scarcely perceptible, and the infant manifesting signs of collapse. The heart sounds in these latter cases tend to be tic-tac in character. In the mild and moderately severe cases, the heart sounds are usually normal, only taking on the characters of the graver form when the case is progressing adversely.

**Urine.** This is usually diminished in quantity. On the institution of proper treatment, however, the urine should not be greatly diminished since free fluid administration/
administration is one of the primary essentials of successful treatment. When the urine is diminished, it usually contains a trace of albumen, probably due to a toxaemia of the renal tubules, in the nature of a nephrosis. In severer instances, the urine will be markedly reduced in quantity, will be highly coloured, and contain albumen. Microscopical examination of the urine must always be carried out. This essential procedure in any case of infantile illness must never be omitted.

General Signs and Symptoms. The child is often cross, fretful and irritable. It sleeps badly. Occasionally the baby is troubled by colicky pains in the abdomen. These pains occur especially at night. The attacks cause the infant to scream out periodically and to draw up its legs. Flatus may be passed, with or without a green motion, towards the end of one of these attacks, with apparent relief of the paroxysm.

A very thorough examination of all these parenteral cases must be made. Particular attention must be paid to the ears, upper respiratory tract and urine. In only one case of this present series of parenteral diarrhoea, was pus found on microscopical examination. The exhibition of alkalis in suitable doses resulted in rapid improvement, both of the urinary and general condition.
Much has been written on the relationship between otitis media and diarrhoea. This has been very fully treated in the previous section. Emerson (1927) states that otitis media is the most frequently overlooked affection of infancy and childhood. Guthrie (1927) considers that otitis media is especially common in under-nourished children.

In a few of the parenteral cases of diarrhoea, but a very few indeed, is attention drawn to the ears by the occurrence of periodic flushings of the pinna and adjacent cheek, or by head rolling, the putting of the fingers into the ears, screaming attacks, or pain on pressure over the meatus (le Mée, 1937). In the vast majority of cases, evidence of ear infection is only obtained on actual auriscopic examination.

Rodger (1937) points out that bilateral infection of the ears is common. Of 185 cases under 1 year, 40% were bilateral, and of 200 cases between one year and two years of age, 43% were bilateral. He stresses the importance of always examining both ears. Further, he states, the onset of otorrhoea or of the necessity of myringotomy in one ear, may precede by several days, or even two or three weeks, the development of otorrhoea, or of the necessity of performing myringotomy in the other ear. As a result of this observation, constant examination of both ears is of vital importance.

As to the changes in the ear drums in cases of otitis media we are up against a very real difficulty almost at once. In many instances, even to the experienced/
experienced eye of the otologist, the ear drum appears perfectly normal in every way, yet very soon afterwards the ear is discharging freely. Of the evident signs of a middle ear infection in infants, a variety of appearances of the ear drum will be noted. In the milder, catarrhal type of otitis, the membrane preserves its normal contour, and may appear moist and glistening. The colour is often more pink than usual, but in other cases it is yellow. The handle of the malleus is well defined and a few vessels may be seen to radiate from it. If there is any scarring or thickening of the membrane from previous disease, the appearance of the drum may be very confusing.

In the more severe form of otitis, which may go on to suppuration, the membrane is red and injected, it has lost its normal lustre and with it the light reflex. The whole appearance of the drum is like red velvet. The handle of the malleus is also, in these cases, indistinct. Though bulging of the membrane is less liable to occur in infants than adults, on account of its thickness (Alexander 1927), the normal contour of the drum is usually increased. This bulging is most in evidence in the posterior segment, which tends to overhang, and even hide the handle of the malleus. Sheldon (1938) points out that the posterior wall of the external meatus is often unnaturally reddened, and slight/
slight oedema may also be observed.

Le Meé (1937) describes a frosted appearance of the drum, the latter being dotted with little white grains. This appearance I have never seen, and Guthrie (1937) thinks that the condition is probably more a characteristic of older children. That the condition, when present, is indicative of an infection in the middle ear seems undoubted.

If perforation of the drum threatens, an area of the membrane begins to necrose and becomes yellow in colour. Pulsation of this yellow patch may be noted. When perforation has occurred, a yellow bead of pus is usually noted at the site, pulsating as it wells out. The margins of the perforation are reddish in colour, fairly regular, and the opening usually oval or circular. Many otologists believe that a perforation through Shrapnell's Membrane always indicates a mastoid infection which requires very careful watching. Operative interference may be required should further signs develop or the otorrhoea not clear up within a short time.

In view of the frequent absence of any characteristic signs of middle ear infection, it is more than probable that otitis media is commoner than we suppose, and that many cases may resolve without any symptoms or signs being manifest. This may partly explain why many of my cases appeared to be only simple rhino-pharyngitis, as the possibility of a middle ear condition could not be/
be definitely ruled out. The findings of the post-mortem examinations of Ebbs (1937) bring this very forcibly to one's attention.

The occurrence of infections in other parts of the body do not seem to bring about the same reaction as does the occurrence of upper respiratory and ear infections. Meningitis, pyelo-nephritis, abscesses, erysipelas, osteomyelitis are other conditions which may precipitate an attack of diarrhoea. These conditions, however, constitute a very small group in comparison with the upper respiratory type of case. Pneumonia, whether of the alveolar or bronchial variety, is another more common condition associated often with diarrhoea. No such type of case was encountered in this series. I have, however, seen both forms of pneumonia accompanied by very troublesome and obstinate diarrhoea.

**Dietetic Diarrhoea.**

**Onset.** The onset may be gradual or sudden. The gradual onset is most commonly noted in those cases where the feeding has been unsatisfactory for some time previous to the occurrence of diarrhoea. The sudden onset is associated with the more severe form of dietetic diarrhoea.

**Vomiting.** This symptom is frequently one of the early features of the cases with a sudden onset. It is frequent, often projectile, and the vomited material peculiarly/
peculiarly sour smelling and offensive. The vomiting may continue for some time, and be a source of trouble in the carrying out of proper treatment. In the milder cases with a gradual onset, vomiting is usually absent, or only occurs on occasion, especially if the mother in her anxiety tries to force the child to feed. Only rarely does it interfere with the carrying out of suitable treatment. On occasion, the vomiting is entirely absent in the severest cases, though the infant is none the less critically ill.

As was noted in the section dealing with treatment the institution of the Raw Apple Diet usually effectively controls any vomiting that may be present.

Convulsions. These are frequent in the severer forms of the dietetic type of diarrhoea. Usually only one or at most two, occur at the commencement of the illness. Exceptionally they may be more numerous.

Anorexia. Anorexia is a constant feature of this form of the diarrhoeal syndrome. In the mild forms with a history of unsatisfactory feeding, a perversion of the appetite is often present. This is particularly noticed in the child of eighteen months or over. In other cases, the appetite is merely diminished. The severer types, with a sudden onset, show a complete loss of appetite. There is no desire for food, and what little may be forced on the unwilling child is promptly rejected.

Thirst/
Thirst. This is marked in both the mild and severe forms. The baby takes water avidly, and unless too much is taken at a time it is well retained.

Tongue. The tongue is heavily coated, both in the cases with a gradual and a sudden onset. The breath is heavy, and if the temperature is high, the mouth is dry, the gums are red and sore, and bleed easily.

Diarrhoea. Diarrhoea may commence slowly or suddenly. In the former instance, the stools gradually become more frequent, green, undigested and very offensive. This change in the character of the stools is, as a rule, preceded by a degree of unsatisfactoriness in the infant's general health, associated with an irregular weight chart and the occurrence from time to time of motions of an abnormal type, though the child is not really ill. This form of onset is especially noted in the chronically improperly fed child, who is subject to frequent bouts of more acute diarrhoea.

If unattended, the motions become more frequent, darker green in colour, more offensive, and one frequently finds that this state of affairs has been going on for some time ere advice is sought. If still the child does not receive attention, the diarrhoea progresses, and assumes the characters of the severer form. These characters, occurring in the neglected, milder cases, are noticed within a few hours of the onset in the more acute cases.
In this latter class, the stools are frequent, up to 12 or more being passed per diem. At first, green, the motions become undigested and offensive. These characters, are, however, soon lost and the stools become thin, pale brown or brown-yellow and watery. The offensive odour is usually lost. Lumps or shreds of mucus are frequent in the watery motions, and streaks of blood are not uncommon. The presence of the latter is due to the congestion of the alimentary mucosa.

Buttocks. The same changes are noted as for the parenteral cases.

Weight. This may be stationary or slowly falling in the mild cases, but is rapid in the more sudden and severely acute forms.

Dehydration. Signs of dehydration are usually present and well marked in the cases of sudden, acute onset. In those instances the signs become manifest within a few hours of the onset of the illness. In the neglected, mild cases, several days may elapse ere the signs become obvious. All the usual phenomena, as described under the heading of the parenteral group, are present. In the mild case, treated early and adequately, dehydration does not become a noteworthy or even noticeable feature.

The abdomen is occasionally tender on palpation.

Temperature. In the slight cases, a mild degree of pyrexia/
pyrexia is commonly present, usually 99°-101°F, but seldom higher. In the more acute type, temperatures of 101°-104°F may be recorded.

**Pulse and Heart.** The pulse rate is increased in all cases, whether mild or severe. The increase of the rate is dependent on the degree of toxæmia. In the acutely toxic cases, the pulse rate is between 140-160, often irregular, and of low tension. The milder instances show only an increase in the rate.

The heart sounds are not, as a rule, affected, unless toxæmia is marked. When such is present the quality of the first sound may be impaired. There is a softening of the first sound, which is most noted in the apical area. Tic-tac rhythm may be present in such instances.

**Urine.** This is reduced in quantity, is stronger, and more highly coloured. It is acid, and only occasionally contains albumen. When the latter is present the case is commonly acutely ill. The strong urine irritates the skin of the napkin area, and this adds to the discomfort of the infant.

**General Signs and Symptoms.** The baby is restless and irritable. It is also pale. Sleep is disturbed, and the child fretful. Attacks of colic are frequent. The eyes are bright, even though the child may be severely ill.
ENTERAL DIARRHOEA.

This class of case also includes the dysenteric forms - or specific forms - which cause the so-called Ileo-Colitis.

Onset. The onset is usually brisk in both the non-specific and specific varieties. Only occasionally is the onset gradual.

Vomiting. Non-specific cases. Vomiting is a very frequent and early symptom of this form of Enteral Diarrhoea. It is often severe and continuous, and treatment by mouth may be extremely difficult, necessitating the employment of other methods of fluid administration.

Specific cases. Vomiting is not a constant nor a persistent feature of this variety of Enteral Diarrhoea. When it occurs it is often associated with a Sonné infection. In the badly toxic case e.g. Shiga and Flexner forms, vomiting may occur at the commencement of the disease, but soon stops even though the prostration and toxaemia are bad.

Anorexia. Non-specific. Loss of appetite is invariably present, being but slightly impaired in the mild case, whereas in the severe case it is a prominent feature.

Specific. A similar state of affairs exists with regard to the appetite as holds for the non-specific variety.

Tongue. Non-specific. The tongue is thickly coated/
coated and dry. The mouth is also dry and parched. Thirst is marked, and fluid given is promptly rejected.

Specific. In this variety the tongue is often remarkably clean. This is especially noted when the general signs of toxaemia are not a dominating feature of the condition.

Diarrhoea. In both the non-specific and specific forms, diarrhoea is an early and very important symptom.

Non-specific. The motions soon lose their faecal characteristics. They become fluid and consist of intestinal debris, pus and occasionally blood in varying quantities. Tenesmus is not a feature of this type of case.

Specific. The diarrhoea is urgent. The stools are, at first, small, green and undigested, with some mucus, and maybe a streak of blood. The mild cases may progress no further, and the motions clear up either spontaneously or on the institution of treatment. In the severer forms, the faecal characters of the stools are soon lost. The motions rapidly come to consist of mucus, pus and blood, with intestinal débris. Tenesmus is frequently a prominent feature of the disease. Prolapse of the rectum may occur in such instances.

Weight. Non-specific. Weight is lost rapidly in this form of enteral diarrhoea, as a rule. As much as one pound may be lost in the twenty-four hours. The loss of weight, of course, depends on the acuteness and severity/
severity of the illness, being less rapid and less marked in the milder forms.

Specific. In these cases, the loss of weight is not, as a rule, so marked nor so rapid as in the non-specific variety. Nevertheless it may be considerable, especially in those cases with marked tox-aemia and frequent evacuations.

Dehydration. Non-specific. This is a prominent feature of all cases of this variety of enteral diarrhoea. It is of slight degree in the milder forms, but may be extreme in the severer cases. All the usual signs will then be present.

Specific. As a general rule, dehydration is not such a marked feature as in the non-specific form. In the toxic cases, however, it may be prominent and rapid in occurrence. Such cases demand immediate treatment. This is not particularly common, fortunately.

Abdominal Pain and Tenderness. Non-specific. Abdominal pain is not a feature of this variety. Attacks of colic may occur occasionally.

Specific. Abdominal pain is an outstanding feature of this form of diarrhoea. Tenderness is most often present over the caecum and colon regions, the child wincing when these areas are palpated. The abdomen is also usually distended.

Temperature. Non-specific. The temperature is high. Occasionally there may be only a slight rise or even/
even a normal reading. This is not, however, usual. Subnormal readings are of bad import. Such may occur in underweight, puny or marantic infants.

Specific. The temperature is again high. Only on occasion is it normal. Even the severely toxic cases as a rule show a high temperature.

Pulse and Heart. In both non-specific and specific forms the pulse rate is increased. The severe cases show often an irregularity in the pulse. These cases may be associated with a tic-tac rhythm of the heart.

Urine. Both forms show a diminished output of urine. It is more concentrated, and may contain a little albumen. On occasions when the urine is very concentrated, the albumen content may be considerable. Actual anuria is uncommon.

General Signs and Symptoms. In both forms, the child is acutely ill, pale and irritable. Apathy often alternates with irritability. Actual coma may supervene in the severely toxic cases. There is a tendency for convulsions to occur in such cases. In these instances where the dehydration is marked, the prostration may develop rapidly. Delirium is occasionally present in those cases with a high temperature.

In all three groups, whether Parenteral, Dietetic or Enteral, the last and severest stage of the illness is/
is common to all. When this stage is reached, it may be impossible to decide the original cause of the disease. It may be reached if the early case has been neglected and untreated, or it may be reached quickly if the disease is severe and the toxaemia profound. In the former instance, several days may elapse ere the stage is reached. In the latter case, it may be reached in a few hours. In the, fortunately rare, condition of Cholera Infantum, the time elapsing from the onset of the disease to its final toxaemic stage, often appears to be reckoned, not in hours but almost in minutes.

The child is pale and collapsed, the nose and lobes of the ears, and the extremities are cyanosed. The temperature is either very high or subnormal. Dehydration is extreme, and the infant is comatose or delirious.

The urine is grossly diminished, and heavily laden with albumen. Anuria may actually supervene, with muscular twitchings, and convulsions may occur. These latter often usher in death, or death may silently steal over the infant.

In this extreme terminal stage, the diarrhoea may, and often does, cease. The eyes are staring, and beads of muco-pus accumulate in the canthi of the eyes. The child does not recognise its parent.

The pulse is uncountable, often irregular, and the action of the heart irregular also. The heart sounds are scarcely audible. The respirations are very shallow and/
and rapid, but now and again, a sigh may be emitted, often the only sign to show that the child is still alive.

The skin completely loses its elasticity, and remains wrinkled after pinching up. Sclerematous changes often take place in the subcutaneous tissues e.g. the lower limbs, and give to the examining fingers the sensation of handling wood.

This final stage is, very fortunately, much less common than it used to be, but it is still, nevertheless, all too common.
SECTION IV.

TREATMENT.
SECTION IV.

TREATMENT.

Infantile Diarrhoea is essentially preventable, and breast feeding is one of the greatest safeguards. This does not imply that breast-fed infants do not suffer from diarrhoea. They do, but the condition is usually much milder, and the chances of survival much greater than in the artificially-fed infants. Weaning should not be carried out in warm weather, unless under exceptional circumstances, such as sudden, acute illness in the mother or some similar misfortune. This warning is not so important as it has been in the past, but, nevertheless, it holds good still, and is one of the Golden Rules in weaning. The other Golden Rule in the weaning of infants, is to make all changes gradually (Watkins 1939).

All cases of diarrhoea, no matter how apparently mild they may at first appear, are potentially serious. Treatment, therefore, must be immediate and energetic. Proper treatment in the early stages will give far more effective results, than will intensive treatment in the late or neglected case.

Certain general principles underlie the treatment of all forms of diarrhoea. They may be given in tabular form, in order of importance, thus:

(1) The fluid balance of the body must be restored and maintained.

(2)/
(2) The mineral balance of the body must be restored and maintained.

(3) The recognition of parenteral infection and the appropriate treatment of such when found.

(4) Rest must be afforded to the gastro-intestinal tract.

(5) Food in easily assimilable form must be given.

Unless sufficient fluid is given, other forms of treatment will be ineffective. Paterson and Smith (1934) calculate that for every lb of body weight, a normal infant requires $2\frac{1}{2}$ ounces of fluid per diem. Smellie (1939) believes that $3\frac{1}{2}$ ounces of fluid per 1 lb of body weight per diem is required in the presence of marked dehydration.

The water content of the infant is some 70% of its weight (McQuarrie 1933). Of this $2/3$ are contained in the body cells, the remainder constitutes the circulating fluids, blood, lymph, etc. (McCance 1938). Infants seem to have a blood volume too large for their weight, but too small for their body surface (Rowntree and Brown 1929). The power of the body to compensate for water loss is strictly limited. The loss of 10% of the water content of the body produces serious disorder of function and manifest signs of dehydration become noticeable. The loss of 20% is fatal (Rubner 1929). As fluid loss tends to be such an outstanding and important feature of infantile diarrhoea, a sufficiency/
sufficiency of water must be given to restore and maintain a normal water balance.

Loss of mineral salts, especially the fixed bases, occurs to a greater or lesser extent in all diarrhoeas. In particular is this loss most manifest in the severer forms of the disease syndrome. Restoration of these lost salts is, therefore, a further and important necessity in treatment. The solutions in common use, for attaining these ends are (1) Normal Saline (0.9% NaCl). (2) Normal Saline (0.45%). (3) Ringer's Solution. (4) Hartmann's Physiological Buffer Salt Solution. In the milder forms of diarrhoea, where the mineral loss is but slight, restoration of the fluid content of the body and its maintenance, are all that is required and this can be done by giving per oram plain boiled water.

In any case of diarrhoea, an essential step is to determine where and when possible, the underlying cause. Upper respiratory infection and especially aural infection are probably the most common causes of the parenteral type of diarrhoea. Microscopic and macroscopic examination of the urine must never be omitted. The diarrhoea resulting from such infections will respond but poorly to treatment directed merely to alleviation of the intestinal symptoms, unless at the same time measures are taken to deal with the parenteral focus.

Digestion is also impaired to a varying degree. The intestinal tract is irritated, either by the tox-aemia/
toxaemia emanating from the focus of infection, or else is itself the seat of abnormal bacterial activity.
Consequently, food will be but imperfectly digested and assimilated, and unabsorbed excess will ferment or decompose with resulting aggravation of the intestinal symptoms. Rest to the bowel is, therefore, also an important feature of the treatment, and easily digested food only later administered in small and cautiously increased amounts.

The duration of this period of rest varies, being dependent on the severity of the illness, its duration, the age of the child and its nutrition. On the Continent of Europe, especially in Germany, long periods of rest have been advocated, ere a return to a modified diet is attempted. In this country and in America, on the other hand, opinion is in favour of shorter periods, rarely longer than 24-48 hours. The usual method of rest to the alimentary tract is by "starvation", but with liberal administration of fluids, with or without glucose, by mouth or parenterally.

After the period of rest is accomplished, food is gradually given to the infant. Marriott (1935) lays down certain conditions regarding the withholding of food from the infant. These are when the infant is grey, dehydrated and apathetic, has a high temperature and appears "toxic". The food must be (1) easily digested/
digested, (2) non-irritating to the alimentary tract, (3) a poor culture medium for bacteria and (4) of a low fat content. The fat delays digestion and often provokes or aggravates vomiting. Breast milk fulfils these requirements admirably, especially the fore-milk which is poor in fat. Unfortunately it is seldom to be obtained, unless the child is breast-fed. The recent introduction of Breast-Milk Depots, such as are instituted at the Queen Charlotte Hospital in London, are steps in the right direction, not only as a preventive measure but also as a curative one for those unfortunate enough to be artificially-fed. Wilmers (1938) has recently described a fairly simple method of preserving breast milk. Such a method is in use at the Infants' Hospital, London. In artificially-fed children, for whom breast milk is not obtainable, a modified skim-milk formula must be employed. Fine curds must be produced in the gut to minimise irritation of the alimentary mucosa. The feeds in all cases must be given in small quantities, frequently and only cautiously increased in strength. The carbohydrate must be difficult of fermentation. The increase of the strength of the feeds is governed by the response of the child to treatment.

After these preliminary remarks on the essentials of treatment, it is probably advisable to discuss whether the treatment should be carried out in an institution or at home. This discussion will be considered under/
under various headings.

**Feeding.** Whenever possible, and home conditions permit, the breast-fed infant should be treated at home. The diarrhoea is likely to be mild and to respond satisfactorily to proper measures. The presence of a parenteral infection has to be considered and any severe one e.g. pyelo-nephritis, otitis media with symptoms suggestive of cranial involvement and similar signs demand hospital treatment. Should the child be critically ill, with dehydration, and symptoms of collapse or cyanosis, institutional treatment is necessary. During the period of starvation the mother must be instructed to empty her breasts at regular intervals, by breast pump or else come to the hospital at regular intervals and have her breasts exhausted. Whenever the preliminary period of starvation is over, the mother must come to the hospital regularly to feed her child. If it is impossible, as is often the case, to come every three hours then she may come at 6 hourly intervals, the breast not being utilised for the feed at that particular time being exhausted, and the milk stored for the infant's next feed.

In artificially-fed infants, the milder cases may be treated at home, under strict supervision, if the home conditions permit of such. The severer cases will do better in hospital. Further remarks are made under the headings of Environment and Parenteral Infection.

Dehydration/
Dehydration. Dehydration, if present to any degree calls for institutional treatment. Thus severe diarrhoea, intractable vomiting, the neglected case, the case with marked anorexia, or which is collapsed and cyanosed, will all show dehydration to a degree necessitating additional means of fluid administration, other than can be given by mouth, and hospital offers the only means of carrying out such urgent measures.

Age of child. The younger the infant, the more serious the condition, and unless the baby is not acutely ill, is in a suitable environment and can receive special attention, it may be treated at home, under the strictest supervision. Otherwise institutional treatment will be required.

Type of stool. If dysentery is suspected, immediate removal to an isolation hospital is demanded. The infectivity of the other forms of diarrhoeal stool, i.e. in the non-specific enteral infections, is not high, but in the presence of other young children in the home, it is advisable to remove the victim of the attack to hospital.

Environment. If unhealthy whether from overcrowding, or other insanitary states, removal to hospital is essential in all cases. An important point concerning environment, is the mental attitude of the parents, especially the mother. If such be co-operative, sensible and willing then the milder cases may be nursed at home/
home. But in all serious cases removal to hospital is necessary, as the environment will not be conducive to urgent treatment.

Duration of illness. The longer the illness has lasted, the more likely are the serious symptoms and signs to be present. Therefore removal to an institution will be necessary. Such cases will usually occur in families of dull-witted parents, or else the child may not have been receiving proper attention by virtue of the mother having too much on her hands, e.g. large family.

The acute case with sudden onset, toxaemia, high fever, and rapid dehydration coming on rapidly following the initial symptoms, will demand immediate removal to hospital.

Type of Parenteral Infection. Serious infections such as pyelo-nephritis, otitis media associated with nervous symptoms, or with a tense, bulging membrane requiring paracentesis, will need removal to hospital. Such conditions as erysipelas, a notoriously serious disease in infants, by virtue of its peculiarity to spread over the whole body, pneumonia, and similar affections, all demand institutional care.

We may sum up the question of institutional care or home treatment, by stating that one must be guided by the condition of the child and the circumstances at home.
The profound and complex changes in tissue metabolism, consequent upon the dehydration, acidosis, and toxaemia, make the treatment of infantile diarrhoea a matter of extreme urgency in many cases. The highest grade of nursing is of great importance, and the child must be very carefully handled. The child must be nursed in a warm (65°F), well ventilated room, or ward. The baby is nursed during its feeds. This is important.

Since dehydration is such an urgent feature of the more serious cases, fluids must be introduced into the body to replace the great loss occurring as a result of the diarrhoea. There is also a disturbance of the acid-base balance with resulting acidosis. The intestinal mucosa is intolerant of food and consequently must be rested as much as possible.

Treatment therefore, consists in stopping all food for 24-48 hours, until the acuteness of the diarrhoea has subsided. As much water as possible should be given by mouth. Often, however, persistent vomiting is present and this mitigates against any great quantity of water being given. Very small amounts of fluid, e.g. ½-1 ounce at ½-1 hourly intervals must be given. If larger quantities are administered, vomiting will be aggravated. Further if the fluid is given cold it may be retained better. If these measures fail, washing out the stomach once or twice daily with ½ N saline should be tried, and small quantities of the fluid left in/
in the stomach.

Colonic lavage is recommended by some authorities, but this procedure is not attended by any great success, and is probably best omitted. Astringents certainly have no place at this stage in the treatment.

All authorities are not agreed as to the necessity of incorporating mineral salts in the fluid given by mouth. All are agreed as to their incorporation when the fluid is administered by parenteral routes. However, it is always advisable to add mineral salts to the fluid given orally. Some (e.g. Graham 1935) believe that N saline should be given. Others (e.g. Moncrieff 1933) believe \( \frac{1}{2} \) N saline is better. In practice these are the two most commonly used solutions. Glucose should be added in 5% strength. A stronger solution of glucose, when given orally, is inclined to irritate the gut. Marriott (1935) advocates Ringer's solution in water in the proportions of 1:3, or a buffered lactic acid solution. He maintains that the latter serves two purposes. The sodium lactate provides base, and the lactic acid maintains an acidity in the bowel, which inhibits organismal growth.

The oral administration of fluids must be supplemented in the vast majority of cases of the severest type, by parenteral means. In not a few cases where it is impossible to administer any fluid orally, whether from weakness or vomiting, all the fluid requirements must/
must be met by giving it parenterally. These routes are, subcutaneous, intraperitoneal, and best and most rapid of all, intravenous. This latter method is the most popular and certainly justifies its popularity by the excellence of its results. It is the method I have always used in hospital.

In the intravenous method the injections of fluid may be either continuous or periodic, preferably the former. The choice of fluid to be given by this route is again a matter of dispute. Graham (1935) states that the provision of a plentiful supply of Sodium Chloride and water, given as N. saline or even as hypertonic saline furnishes all the base that is required. The chlorine ion appears to be excreted as an ammonium salt, leaving the sodium ion to replenish the store of alkali. Marriott (1935) and Payne (1938) point out that in some of those cases with a severe degree of dehydration, a high blood chloride is already present. Thus the injection of further chloride will increase this already high blood chloride, and aggravate the existing acidosis, instead of relieving it. The administration of alkalis is, at best, only a palliative measure, and may actually do harm, and its use should not be countenanced (Graham, 1935).

In actual practice, Normal saline with added glucose in a strength of 5%, is given for the first 24 hours, thereafter, \( \frac{1}{2} \) Normal saline is substituted.
When and where possible, chemical analysis of the blood is desirable, in order to ascertain what particular strength of solution is required in any particular case. In all cases the solutions used must be freshly prepared and, of course, sterile.

The continuous method of intravenous administration of fluid is the better of the two methods. A complicated apparatus is required. A 300 cc. graduated funnel of glass, or better, a thermost apparatus of similar capacity, drip connection, rubber tubing, screw clamps, cannula and a fine ureteric catheter, are the main necessary components of the apparatus. The great saphenous, or the median basilic, veins, are the sites of election for inserting the catheter.

The chosen vein is cut down upon, and its lower end is ligated. The catheter is inserted through a transverse incision into the vein for a distance of some 2-3 inches or even more, and tied in position with cat-gut ligature. During the insertion of the catheter, fluid must be dripping freely from its lower end, to avoid the introduction of air bubbles into the vein. By the use of the ureteric catheter, splinting of the limb is obviated, and the child is given a degree of movement which would be precluded by splinting if the cannula was used, as formerly.

The quantity of fluid given usually amounts to 2 ounces per one pound body weight (Karelitz and Schick, 1931). In those cases with marked dehydration, the first/
first 100-200 cc. may be given quickly, e.g. in 20 minutes. Thereafter, the rate of flow is reduced to 15-40 cc. per hour. The catheter usually remains patent for 12-36 hours, or even longer. The administration of fluid is continued so long as there is any toxaemia, and until fluid can be tolerated by mouth. As the amount of fluid taken orally increases, the amount given intravenously is decreased. Even when the child is taking the requisite 2 ounces per one pound body weight by mouth, it is advisable to continue the intravenous administration at a slow rate, e.g. 3 cc. per hour, for a further period of 12 hours. This method of continuous intravenous therapy is far and away the best at our disposal.

The other method of intravenous fluid administration is the intermittent or periodic way. In this method, the injections are given at intervals. The apparatus required consists of needle, glass funnel and rubber tubing. The site of election in this method is either the longitudinal sinus through the anterior fontanelle or the median basilic vein. The same precautions re having the fluid dripping freely from the end of the needle while entering the vein, holds good as for the continuous method. Administration is better carried out by the gravity method, than by the two-way syringe. 20-30 cc. per 1 kilo body weight are given. Such injections of fluid are given at 6 or 12 hours intervals/
intervals, and continued until improvement in the general condition is manifest, and the child taking well by mouth. I have had more experience of this intermittent method of fluid administration, than the more recent ureteric catheter method of continuous administration. Judging from the few instances in which I have employed the latter method, I have no hesitation in saying that the catheter method is the better. It has the advantage, also that it can be continued or stopped at will.

Intraperitoneal Injection. This is another way of rapid restoration of the tissue fluid. It is greatly favoured by many. It has the very serious drawback, however, of being attended by a great risk of infection. Normal and \( \frac{1}{3} \) Normal saline are used, with glucose added in 2% strength. Injections may be repeated every 6 hours. As much as 10 ounces of fluid may be given at one time. This route must never be used if there is any abdominal distension, or if, in a doubtful case, there is a possibility of laparotomy at a later date. The temperature of the fluid should be 100 F.

The bladder must first be emptied. A short bevelled needle, e.g. an intravenous needle, is introduced in the mid line, half way between the symphysis pubis and the umbilicus. The fluid may be given either by the gravity method, or by the two-way syringe, or better, by the thermos apparatus. After the operation, the needle/
needle puncture is sealed with collodion.

**Subcutaneous Injection.** This is the least satisfactory of the parenteral methods of fluid administration. It is unnecessarily painful, the risk of infection is considerable, and as it takes longer to perform, the exposure of the infant is increased - a serious drawback in the collapsed infant. The amount of fluid that can be given by this method at any one time, is less than can be given by the other routes. Absorption is slow and irregular. Not more than 4 ounces can be given at any particular site, as a rule. The solutions used are Normal and \( \frac{3}{4} \) Normal Salines, with 5% glucose. A stronger solution of glucose should not be employed. The injections may be given either by needle and syringe slowly, or better, by needle and funnel i.e. the gravity method. Too rapid injection may cause a violent tissue reaction. The injections are repeated every 6 hours, a close watch being kept on the rate of absorption. The common sites for injection are, the loose areolar tissues of the axillae, the flanks, thighs and lower abdomen. Paterson (1933) believes, though he states he has no scientific proof, that subcutaneous injection of fluid into the axillae predisposes to terminal pneumonia. One advantage of this method is that it can be carried out by a trained nurse. Apart from this, however, the subcutaneous route has little to commend it.

Blood/
Blood Transfusion. Mention of blood transfusion in these severe cases must be made. Findlay (1933) states the effects are not encouraging in his experience, and Moncrieff (1933) is of the same opinion. Neale (1931) and Marriott (1935), on the contrary, are enthusiastic over its use. Neale draws attention to the loss of protein in the fluid escaping from the body by way of the intestine. He states that the efficiency of blood transfusion may have a relationship to this. Marriott is of the opinion that the repeated transfusion of citrated whole blood is of the greatest value. In his opinion, transfusions, however, must not be given until the fluid balance of the body has first been restored. He also believes that the immune bodies contained in the transfused blood may have some effect in increasing the resistance of the infant to infection. From the slight experience I have had of blood transfusion in infantile diarrhoea, the results were not impressive.

Needless to say in all those operations for the introduction of either blood or fluid into the body, the most rigid aseptic and antiseptic precautions must be taken.

Feeding. How long should a child be kept on bland fluids before commencing feeding? This question can only be answered for each individual case. A generalisation is impossible. We must always remember that an/
an infant has no reserves of carbohydrate and that the protein so necessary for building up the body is being either stopped or restricted during the preliminary period of "starvation". In order to provide sugar for energy, glucose is always incorporated in the solutions employed to restore the body fluids. The use of glucose has also another function, viz. to prevent the breakdown of body protein to glucose in order to provide energy i.e. the glucose is a protein sparer.

In this country, feeding on modified milk mixtures is begun as soon as possible. In very mild cases of diarrhoea, the period of starvation usually rarely lasts longer than 12 hours, often 6 hours. During this period, as much fluid as the infant will take is given. This fluid is either boiled water with added glucose (5%), or Normal or $\frac{1}{2}$ Normal saline with glucose. In the mild cases in this series, glucose dissolved in boiled water was employed. It was given freely, frequently and in small quantities at a time to obviate the occurrence of vomiting. When vomiting was present, it often yielded when the fluid was given cold.

In the more severe cases, the period of starvation was increased to 24 hours, never longer. As for the mild cases, glucose in water was used. By the administration of small, frequent feeds of fluid, one can often get the infant to take as much as 1-1$\frac{1}{2}$ pints in the 24 hours. Actually this amply sufficed in all the cases/
cases treated in this series when the starvation regime was employed.

After the period of starvation is completed, the stage of return to a suitable diet is reached. This stage is often a very difficult one. Where possible, and always in the already breast-fed infant, a return to breast milk should be made.

**Mild Diarrhoea in the breast-fed baby.** Breast feeding is resumed at 4 hourly intervals, save in the very young infant, when 3 hourly intervals are available. Plain boiled water, or $\frac{1}{2}$ Normal saline is given before each feed at the breast. This helps to take the 'edge' off the infant's appetite, so that it only gets the fore milk since it will suck less vigorously after the water or saline. This has the advantage that the fore milk is fat poor. Such breast feedings should at first be given for only 2-3 minutes and gradually the duration of the feed increased as improvement takes place. After the breast feed, more fluid is offered to the infant. As progress is maintained, the infant takes less and less of the fluid and sucks longer at the breast until a return to normal is made. Should weaning have been in progress at the time of the onset of the diarrhoea, it should not be recommenced until the child has settled down and is digesting the breast milk well. Then and only then, should supplementary feeds be introduced, and these only gradually, carefully noting their effect on/
on the infant's progress.

Treatment of a parenteral focus is also carried out simultaneously.

**Breast milk feeding in the severe case.** Breast milk is the ideal food when a return to feeding is being made in the cases which have been severely ill. With the institution of Breast Milk Depots there is now much more chance of the critically ill infant making more rapid and steady progress when such a supply is available. If the milk can be obtained, 2 drachms made up to 1 ounce with boiled water should be given every 3 hours. The milk is increased daily by 1 drachm, reducing the water in the feed but still giving the infant as much water as it will take. In a short time the baby will be taking whole breast milk, and if progress is satisfactory, the feeding times will be prolonged till a return to normal is accomplished.

**Artificial Feeding in the mild case.** The bulk of the cases in this series were of this category. Skimmed milk and water mixtures, lactic skimmed milk and evaporated milk may be used. In this group these were the three forms of modified cows' milk employed. Feeding intervals depend on the nutrition of the child. If this is fairly good then the 4 hourly regime should be adopted, whereas if the infant is small or puny, then the 3 hourly regime is recommended.

After the preliminary period of starvation has been/
been accomplished, small feeds of any of these modified cows' milk mixtures should be started. A suitable quantity of skimmed milk, boiled, is $\frac{1}{2}$ ounce. This is diluted with a similar quantity of boiled water. No carbohydrate is added at this stage. Before and after feeds boiled water is given to the child, as much as it will take. Additions of $\frac{1}{2}$ ounce daily of skimmed milk and $\frac{1}{8}$ ounce of boiled water can be made until the child is getting the normal bulk of a feed proportionate to its age. As the feeds are increased, less water is taken in the intervals.

When the normal bulk of the infant's feed has been reached, carbohydrate is added. Dextri-maltose or glucose may be used. One or other of these carbohydrates is added to the feeds, at first $\frac{1}{3}$ drachm per feed, later increased to 1 drachm. Simultaneously with the addition of carbohydrate to the feed, the strength of the milk mixture is increased. $\frac{3}{4}$ ounce of milk is added daily, decreasing the water proportionately. By this means, after a varying time, governed by the response of the child the full strength feed is reached. During this time also, some cream may be introduced into the skimmed milk, thereby bringing the milk feed up to full complement.

**Lactic Acid Skimmed Milk Feeding.** This is a form of modified cows's milk which is strongly advocated by Marriott and Exchaquet (1935.) Several of the cases
in this series were treated with this milk. The initial feed is rather stronger than the milk-water mixture. 1 ounce of this milk is a good initial feed. Carbohydrate can be added at the beginning, in this type of milk modification. ½ drachm of glucose or dextrin-maltose is added to each feed. The feeds are steadily increased in strength daily; ½ ounce of lactic skimmed milk being added each day. By this feeding method, a more rapid return to full calorie diet can be made. The several cases which were treated with this milk responded very well.

**Evaporated Milk Feeding.** This is another form of modified cows' milk which has recently had a vogue. The brand used in this series was the Carnation Brand. This is given in the proportion of 1 part of evaporated milk to 3 parts of water. ½-1 ounce may be given 3 hourly or 4 hourly, with added carbohydrate. The progress noted in these children was disappointing, compared with those on skimmed milk-water mixtures and lactic milk. Skimming this form of milk was obviously out of the question as the milk is homogenised. Attempted acidification of the milk was carried out, using lactic acid. The administration of this acidified diluted milk mixture gave better results than when it was not acidified. Altogether, however, the results were disappointing in the few cases so treated.

**Protein Milk Feeding.** Little may be said concerning/
concerning this form of modified milk. It was not used in any of the cases in this series. Many advocate its use, but it is most suitable for hospital cases, where proper facilities are present for its preparation. It is not suitable for cases treated at home unless the mother is sufficiently intelligent to carry out efficiently its rather difficult preparation.

Artificial Feeding in the severe case. When feeding is started, the great essential is to commence with small quantities, make increases slowly, and be guided by the reaction of the child to food. Too prolonged starvation or underfeeding in infants already critically under-nourished may be fatal. On the other hand, too rapid increases of the food may result in an exacerbation of the symptoms which may prove fatal.

The previously mentioned modified milk mixtures may be used. Protein milk is especially useful. Whatever form of modified milk is used, the initial feed should not exceed 2-3 drachms of milk, made up to ½ ounce with boiled water, and given 2 hourly. The alternating hours, water is given, as much as the child will take without inducing sickness. The milk is increased daily by 1-2 drachms, until the bulk of the feed proportionate to the child's age has been reached. Thereafter, carbohydrate is added to each feed, ½ drachm at first, then 1 drachm. When the child has recovered, symptoms have disappeared, and the stools are normal, then/
then and only then, should whole milk be given undiluted. The addition of this whole milk may conveniently be done by replacing one diluted feed per diem by a whole milk feed. By this time also, the child will be able to be fed every 3 or 4 hours.

This severe form of infantile diarrhoea is, fortunately, not so common as formerly. None the less it is still too common. The artificially fed baby is especially liable to this acute, toxic type of diarrhoea. Johnston, Brown and Tisdall (1930) record that out of 146 cases of intestinal intoxication only 5 were breast fed. Smellie (1937) is quoted as having found only 3 breast-fed infants in a group of 60 cases of clinical gastro-enteritis. These figures serve to emphasize the value of breast feeding as a prophylactic against infantile diarrhoea, especially the severe and often fatal forms. The chances of survival of the breast-fed child on these rare occasions when such children are stricken with this dreadful disease, are very much better than obtain in the artificially fed children.

Mention must here be made of the variety of diarrhoea occurring in the undernourished child. In the breast-fed baby, measures should be taken to increase the supply of milk. During the application of these measures, a complementary feed of cows' milk, boiled, and suitably diluted, with added carbohydrate, should be given after the breast feeds, to make up for the deficiency/
deficiency in calories. A test feed is a great advantage in enabling one to calculate the required quantity of complementary feed. Regular feeding must be insisted upon, and weaning must not be undertaken.

In the artificially-fed child, a careful enquiry should be made into the composition of the feed. It will usually be found that the child is being given too weak a mixture of cows' milk and water. Consequently all that is required is an increase in the calorie intake of the child and this may be accomplished by strengthening the feed by addition of more milk, and/or carbohydrate.

A trial was given in some 55 cases of the present series, to the Raw Apple Diet. This method of therapy has only recently been introduced into orthodox medicine.

Attention to the therapeutic properties of raw apple pulp in the diarrhoeas of infancy and childhood was drawn by certain German authors. (Heisler 1929-1930) and Moro 1929). The diet is often, consequently, spoken of as the Moro-Heisler Diet.

Scraped raw apple pulp was administered as a home remedy for diarrhoea as early as 1775 (Council on Foods of the Amer. Med. Assoc. 1937). Heisler was first introduced to the treatment of diarrhoea by raw apples, when one of his adult patients, a long suffering victim of diarrhoea, tried out the regime with marked success himself. Though such a treatment had been in existence for/
for such a long time, it was not until these German authors described the beneficial effects accruing from its use, that any serious attention was given to it.

A vast amount of literature has appeared on this subject since these original publications in Germany. The results recorded by the various authors have been by no means uniform, for while some are enthusiastic, others are disappointed with the treatment. The enthusiasm of a small band knows no limits, and they acclaim the treatment as a cure for most forms of diarrhoea, whether in children or adults. The regimes adopted by the various authors differed in several smaller details, from the original recommended by Moro.

The rationale of the raw apple diet in diarrhoea is still not clearly understood. Some believe the beneficial effects are due to the Pectin contained in the apple, others to the astringent effect of the tannic acid contained in the apple pulp. Yet others ascribe the effect to the large amount of cellulose in the diet which acts in a purely mechanical way. These theories will be discussed in more detail in a later section.

My introduction to this method of treatment was made in an isolation hospital, where cases of both infantile and adult dysentery were submitted to the diet. The results were so encouraging that I attempted to put the lessons learned then into operation and to treat/
treat some 55 cases of infantile diarrhoea of all forms, save the dysenteric variety, at home, under my own supervision and that of the Health Visitors.

The method adopted in this small series, consisted in the administration of finely grated apple, raw and peeled of its skin. Some advocated that the skin should be incorporated in the grated pulp, but one felt that carelessness on the part of the mother might result in dirty, or uncleaned skin being introduced into the pulp, with probable undesirable results. The pulp is administered for three days. No other nourishment is given, but liberal quantities of boiled water were allowed. Moro (1929) advised against the concurrent administration of fluids, but most observers are agreed that the giving of fluid, together with the apple pulp, does in no way detract from the value of the diet.

The apples chosen must, of course, be ripe. Apples with a soft pulp are probably the best and most efficacious. Such species are, Jonathan, Mackintosh and Delicious. The harder varieties e.g. Newtons, can be used, and I found no ill effects resulting from their use. The apple must be finely grated or sieved. The raw apple pulp can be given to infants of all ages. The youngest in this group was 18 days old, the oldest was just under 2 years of age.

To a young baby under three months, one-two (1-2) teaspoonfuls of the grated apple pulp is given two hourly. Actually/
Actually more may be taken by the infant and no untoward effects noted. To an older infant, between 3 months and six months of age, two-six (2-6) teaspoonfuls every two hours may be given. For the older child, the pulp is best given three hourly, in quantities of two-four (2-4) tablespoonfuls or even more. It may be said without fear of contradiction, that as much apple pulp may be given as the child will take.

This diet is carried out during the day only, though some advocate that it should be continued throughout the night also. This is, of course, impracticable when treatment is being carried out at home. The duration of treatment is usually three days only. In some very mild instances the regime may be continued for only two days.

During the period of the apple administration, fluids are given freely. Plain boiled water may be given to those cases treated at home. Very weak tea is recommended by some, and this was given to those treated at the Day Nurseries. The tea is prepared from mild Indian tea, ⅛ teaspoonful to ⅛ pint of water, and allowed to infuse for not longer than two minutes. No milk or sugar is given with the tea. No more appreciable benefit, however, seemed to be derived from those cases given tea in addition to the apple diet, than was already being obtained from exhibition of the fruit along with plain boiled water.

Occasionally some difficulty is experienced in getting/
getting the infants to take the apple pulp. One usually finds that this difficulty is more manifest in the older children. In the case of the 18 day old baby, the pulp was taken as if the child had had it since birth. Those infants who have been weaned and who had acquired tastes for other foods, are those who tend to present the difficulty. When such instances arise, the sprinkling of a little sugar over the apple pulp, or better, the incorporation of a small quantity of ripe banana, well mashed, into the apple pulp suffices to tempt the refractory palate.

The presence of vomiting is not a bar to the use of the diet. When the vomiting is not severe, the administration of the pulp actually seems to help it. The reason for this is probably due to the stomach having something to contract down upon. However, if vomiting is severe and likely to prove difficult to control, it is improbable that the apple will be retained.

Under this regime, the stools in all cases, became less offensive, and were reduced in frequency. In some instances, this reduction in frequency was marked, in others it was less so. The reduction in frequency was least noticeable in the cases due to a parenteral infection. However, in all cases, the offensive odour had disappeared within 48 hours, usually much sooner. The rapid reduction in frequency and the abolition of offensive/
offensive smell was most noticed in the dietetic and non-specific enteral cases. Fretfulness and irritability, and attacks of abdominal pain soon disappeared on the institution of the diet. In a few cases, the improvement was so marked within 48 hours that the last day of the diet was omitted and cautious return to a milk feed allowed with no untoward result.

The last six cases of this series of 55 treated by the apple regime, were given a proprietary powdered apple preparation - Aplona, instead of the fresh fruit. The quantities of this powder used, varies with the age of the child. For children under six months of age, ½ teaspoonful of Aplona is added to 5 ounces of water, which has been boiled, and is miced with the powder when warm. For older children, 1-2 teaspoonfuls of the powder are mixed with 5 ounces of warm, boiled water. The mixture of Aplona and water is then stirred well, shaken and allowed to stand for 5-10 minutes. The mixture must not be boiled, and no sugar is required. 2-5 ounces of this mixture are given 2 or 3 hourly. Fluid, in the form of plain-boiled water, is given freely in the intervals between the Aplona feeds.

Certain advantages are claimed for this preparation. The most important of these claims seems to be that the powder is produced from a certain species (unnamed) of apple, in a certain stage (unstated) of maturation. Various species of apple are said to have varying therapeu-
therapeutic properties, but this small assay seemed to show that, provided the apples were ripe and were finely sieved, the species used did not influence the final results of the treatment, and the six cases treated with Aplona gave no better and no more rapid response than those treated by the raw pulp. An important and very practical disadvantage of this particular preparation, is its unpleasant taste.

In all the 56 cases, where the apple treatment was used, the continuation of the apple for a further period of 4 days, given at two feeds in the day, was carried out. The two feeds chosen to administer the apple were the 10 a.m. and 6 p.m. feeds. It had been found from a previous experiment with the apple diet, that occasionally the diet had to be resumed, because of a return of either the looseness or the offensive odour. With the continuance of the apple in two feeds per diem, for a further period of 4 days, in some instances even up to a week, after the intensive course of apple, the return of offensiveness was abolished almost entirely. The tendency to relapse into a state of looseness of the stools on the gradual resumption of a weakened milk feed was reduced to practically nil by thus continuing the apple twice daily.

No case of dysentery was treated in this series, all being transferred to an isolation hospital. When considering the treatment of this specific form of diarrhoea/
diarrhoea, later, mention will be made concerning certain modifications of the apple diet, often found to be necessary when treating these cases.

On the fourth day, resumption of the milk feeds is undertaken. These feeds must be small in amount at first, and given at 3 or 4 hourly intervals. Boiled cows' milk and water mixtures are probably the best for use in the home treatment of such cases. Lactic acid milk, skimmed or whole, and protein milk are also excellent. Carbohydrate should not be added at first. A suitable initial feed consists of 1 ounce of boiled milk and 1 ounce of boiled water. In the intervals between feeds, the child is given water, as much as it will take.

Additions to this weak feed must be made after due consideration has been given to the response of the child. If this is satisfactory, then the milk and water can be increased to two ounces of each per feed. Once normal bulk of feed has been reached for a child of any age, then sugar can be added, in the proportion of $\frac{1}{3}$ drachm per feed at first. Later it is increased to 1 drachm. Simultaneously with the addition of carbohydrate, the strength of the milk in the feed is increased. Such an increase of the milk may be carried out, by replacing $\frac{1}{2}$ ounce of water in the feed daily by $\frac{1}{2}$ ounce of milk. An interesting feature of the feeding is that the milk does not need to be skimmed in the great majority of these cases.
cases treated for the first three days of the illness by the apple diet.

**Nursing.** This is vitally important. Meticulous care and the most scrupulous cleanliness must be observed. Gentle handling of the infant is essential. All cases of diarrhoea, whether mild or severe, must have plenty of fresh air. In mild cases, it is permissible to take the child out into the open, provided that it is kept warm. Nursing at the feeding times is one of the primary essentials.

The temperature of the warm or room should be kept at 60-65°F. To keep the child warm, small hot water bottles, suitably protected, should be placed in the axillae and between the thighs. If the child is being nursed at home, the cot should be so placed as to be outwith the path of draughts between the door and windows. A fireplace, with or without a coal fire, is an added advantage, assisting as it does in the ventilation of the room.

Clothing must be light, porous and warm. Woollen garments are undoubtedly the best. The baby must be nursed at feeding times, on the mother's or nurse's lap. If there is much post-nasal secretion, the head is kept slightly raised, supported by one or two pillows. This permits of natural drainage of the nasal secretions, so that they do not accumulate in the posterior nasopharynx, where upward extension to the ears might occur.

Soiled/
Soiled napkins must be promptly removed, and placed in a basin containing some antiseptic solution. The buttocks must be washed gently and anointed with some simple bland ointment. Vaselin is probably the most satisfactory of the bland protective applications. Gentle handling of the infant is very necessary. Too long or too great exposure of the body must be avoided under all circumstances.

Bathing should be discontinued in all cases, during the illness. A warm sponge of the whole body night and morning is done instead. Attention to the mouth is essential, especially after feeds. For this, 1% Gentian Violet, gently swabbed inside the mouth, gums and palate is an efficient prophylactic of oral infections.

Gentle bathing of the eyes with normal saline or plain boiled water, twice or thrice daily, is to be recommended when beads of pus or muco-pus tend to accumulate in the canthi. The bathing is, of course, directed from inner to outer canthus. Boracic lotion, the time honoured remedy, is not altogether free from risk. Occasionally it sets up a nasty dermatitis. After bathing the eyes, a small quantity of vaselin or Ung. Hydrarg. Ox. Flav. (B.P.) should be smeared along the margins of the eyelids.

Drugs. These are of no great value in the treatment of infantile diarrhoea. An initial dose of castor oil has much to commend it, especially in the milder instances.
instances. It should not be given in the severe cases. The rationale of the action of the oil is that it effectively clears out the bowel, and tends to exert a constipating effect thereafter. The oil is especially valuable and effective in the mild dietetic and enteral forms. Some recommend small doses of Hydarg. c. Cret., instead of the oil. It is quite a good remedy in the parenteral cases, one dose being given only, viz. at the outset of treatment. Castor oil was the drug I favoured most in this series.

Astringents are valueless, and actually contraindicated in the acute stages of the disease. Some recommend their use in the subacute and chronic stages, but their value then is open to question also. Demulcent drugs, as chalk, Kaolin and bismuth have been recommended for the condition from time to time. They are of must value in the dysenteric type of case. They have but slight effect in the other forms of the disease. When they are given, large doses must be employed, and in the case of bismuth, pushed to the extent of producing constipation. Bismuth subcarbonate or salicylate, are the two preparations of bismuth of most use. Dosage varies from x-xx grs, 4 times daily. The subnitrate is contraindicated. The best drug in my experience, for controlling the looseness of the bowels, which is often troublesome in the subacute stage, is castor oil in small doses. Such a dose would be 5 minims/
120.

minims, made up with mucilage of acacia and peppermint water, and repeated every 5 hours, i.e. 5 times daily. This mixture was the one I used most frequently, especially in the parenteral cases, not treated by the apple diet when no looseness tended to occur.

Opium is recommended by many. Still (1915) speaks very highly of its value. Hutchison (1931) lays down certain rules regarding the use of opium. These rules are:

(1) Never to give opium at the outset of the illness.
(2) Never to give opium if signs of collapse are present or threatening.
(3) Never to give opium if the tongue is furred.

Hutchison goes on to give the indications for its use:

(1) Frequent stools with much tenesmus. Consequently opium in some form is most used in the dysenteric diarrhoeas. By the combined use of the apple diet and salines, the need for opium administration has, in my view, been lessened in this type of diarrhoea.
(2) A clean tongue, with the presence of frequent offensive stools.
(3) In those cases of functional - so-called lienteric - diarrhoea.

The forms in which one may administer opium are (1) liquid and (2) solid. The former is the better form/
form, and is to be recommended. Tr. Opii. is the best liquid preparation. Dosage varies with age. Thus 1/8 minim up to 3 months of age, 1/6 minim at 6 months, and 1/4 minim at 1 year. Dover's Powder or Pulv. Ipecac.c. Opio. is the best of the solid preparations. It may be given in doses of 1/8 gr. at 3 months, 1/6 gr. at 6 months, and 1/4 gr. at 1 year. I never had occasion to administer opium to any of the children treated in this series. However, when in hospital, I used Tr. Opii, but very seldom was its use called for.

A baby should never be awakened to be given a dose of opium.

Value of alcohol is still disputed. Its use is often expected by the parents. Still (1915) thought that alcohol, in the form of brandy, was valuable in certain cases, with collapse and exhaustion. He gave x-xx minims in 1 teaspoonful of cold water, repeated every 2 hours if necessary. He deprecated the use of larger doses. There seems to be no doubt that alcohol is very efficacious in certain carefully chosen cases. It is especially useful in those cases where vomiting is troublesome. Brandy and whisky are the two forms of alcohol to be recommended. Not only do they seem to 'settle' the gastric mucosa, but they also provide some fuel for combustion. I cannot agree with those who state that alcohol is of no use in the treatment of infantile diarrhoea. In many instances, its use seemed/
seemed to be life saving. Sherry whey is a particularly useful form of administering alcohol to a baby who is vomiting. It can only be given for a short time, however, and gradually replaced by milk feeds. Sherry whey was the food given to some of the infants for a short time, in the outbreak of Sonne dysentery occurring in the whooping cough convalescent ward, as described in the section dealing with aetiology.

Collapse should be met with by a hot mustard bath (1 tablespoonful of mustard in 1 gallon of water). The baby must be immersed in the bath. It must be completely undressed, and bathed in front of a fire. Allow the child to remain in the bath for 4-5 minutes, supported by the nurse's hands. Dry quickly with a warm towel, and wrap in a warmed blanket, and return to the cot, which should have been warmed by several hot bottles, suitably protected. Such hot water bottles may be improvised by utilising old Milk of Magnesia bottles, suitably covered, and placed in the axillae and between the thighs, i.e. adjacent to the great arterial trunks of the upper and lower limbs.

For hyperpyrexia, a tepid bath, rather than a cold one, is desirable. This together with brisk rubbing, will result in profuse sweating.

Coramine and Camphor in olive oil are both useful for collapse, (Chopra et al, 1936). Both may be given subcutaneously, though coramine may be given in other ways/
ways, e.g. orally. Adrenaline and strychnine hypodermically are not free from danger, and are not to be recommended as a routine measure against collapse (Lucas, 1914). It may be mentioned, however, that intra-cardiac adrenaline does seem to cause at least a temporary revival in some severe cases of diarrhoea.

To induce sleep, there is no better drug than Chloral hydrate. Antipyrine is especially useful in cases associated with pain, e.g. otitis.

Lastly mention may be made of the use of anti-coli serum in the cases of non-specific enteral infection. A polyvalent serum is recommended by Plantenga (1935), in doses of 20-30 cc. This form of therapy is not used in this country to any extent. Reports on its efficacy are conflicting.

In the dysenteric variety of infantile diarrhoea, treatment differs in some details from that outlined above for the other forms.

Dehydration is often not so severe. In the profoundly toxic cases it may be well marked, however. Restoration of the water and mineral balance is carried out as for the other types previously described, with the possible addition of anti-serum administration.

In the case of average severity, in the early stage of the disease, an initial dose of castor oil should be administered, and a period of starvation instituted, lasting from 12-24 hours. During this period/
period, glucose (5%) in boiled water, normal or \( \frac{1}{3} \) normal saline should be given, freely, and in small quantities at a time. In those cases where vomiting is troublesome, as occurs in some Flexner and Sonne infections, the glucose solution will be better retained if given cold. Gavage, however, may be necessary in obstinate instances.

At the end of this period of starvation, a return to a milk feed may be made. Milk and water mixtures, or lactic acid milk may be employed. In hospital, one got better results with the latter feeds. It is not nearly so necessary in this type of infection to restrict the quantity of the feed, as is so necessary in the non-specific and dietetic and parenteral forms of the disease, since the colon is the main seat of the pathological changes in the dysenteric variety.

The quantity of the feeds will vary, of course, with the age of the child. For the infant, 3 ounces of lactic milk, with added carbohydrate is a suitable initial feed. If milk and water mixtures are favoured, then 2 ounces of boiled cow's milk and 1 ounce of boiled water, with added sugar, is a satisfactory feed to commence with. Normal quantities may be allowed in the older child. Due consideration must be given in all these cases, as in the others, to the reaction of the child to food, and its progress in combating the infection.

The/
The principles of nursing are the same as for the other forms of diarrhoea, with the additional necessity of complete isolation, and strict 'barrier' nursing. Thorough disinfection of all excreta and fomites is called for. These dysenteric infections spread like wildfire through an infants' ward, unless the nursing is of a very high order.

Treatment of dysentery cases by the raw apple diet is an excellent method. All but the most severely dehydrated cases can be submitted to this form of therapy. The diet should be given as previously described, for three days, and the rapid abatement of the toxaemia and the improvement in the stools, are most marked. In a previous experiment with this diet in dysentery cases, it was found that though the diet relieved the toxaemia and greatly improved the stools, the tenesmus, so often present, was not so dramatically alleviated. Consequently, the simultaneous administration of salines such as Sod. or Mag. Sulphate was tried. This assay was very successful, the alleviation of this distressing symptom being remarkably rapid. The drug was administered every hour for three hours in the morning, the dosage varying with the age of the child, x-xx grs. being the usual dose. Such administration of a saline along with the apple diet should be continued for the three days as a rule, but occasionally, the saline may be discontinued after the second day, if by that time the tenesmus has been relieved. It is probably best however, to continue the saline for a further day to/
to obviate the risk of a return of the tenesmus. The apple is continued for the following week after the intensive course, being incorporated in the morning and evening feeds only, as previously described.

**Drugs.** The best and most useful are the salines, sod. or mag. sulphate. The one or the other may be given. Three doses of the saline are given at intervals of one hour in the mornings. As a rule, this causes two or more satisfactory evacuations from the bowel, per diem, and rapidly relieves the tenesmus. In many instances, two days' treatment with these drugs suffices, but in any case, the saline should be continued till the motions have become normal, and till blood and mucus have disappeared. The use of the saline obviates the necessity of saline or astringent irrigations of the bowel. The latter are more helpful in the chronic forms of dysentery.

Polyvalent sera have been recommended, especially in the severer cases with much toxæmia. The anti-dysenteric serum is given in doses of 5-10 cc. intramuscularly, once daily till improvement in the condition of the child becomes manifest. Reports on this form of therapy are very conflicting. Findlay (1933), Moncrieff (1933) and Pearson and Wyllie (1935) say that the anti-serum should be tried, but Josephs and Davison (1921) were not impressed with its value. To be effective, the serum must be specific for the causal organism, and until the organism has been identified, the anti-serum/
antiserum used must be polyvalent, and therefore treatment in the early stages before bacteriological examination has been completed, is more in the nature of a shot in the dark. From only slight experience, however, I thought that intravenous glucose in Normal saline for those toxic cases seemed to produce more rapid, and in the long run, more satisfactory end results than when anti-serum was employed.

Bacteriophage had a vogue in the treatment of dysenteric conditions at one time. It did not come up to the expectations of its sponsors, however. The criterion of cure taken by some authors, is the disappearance of the organism from the stools (Seidlmayer, 1939). Haler (1938) described an outbreak of dysentery in which bacteriophage was of considerable value. The use of bacteriophage seems to be restricted to the more troublesome cases, which are not responding to the more usual measures. I found a bacteriophage preparation, Enterofagos, to be a more than useful adjunct to treatment in several cases of non-specific dysentery, in children. Since employing the apple diet, however, there has been less call for such form of therapy.

Finally, in this discussion on the treatment of infantile diarrhoea, it may be wise to give an outline of the treatment adopted for the various parenteral infections encountered in this series of cases under consideration.

The/
The Common Cold. This was either a simple rhinitis or a rhino-pharyngitis. It was far and away the commonest infection encountered. The treatment adopted for this affection was that recently introduced in the Maternity and Child Welfare Department of Edinburgh.

Regularly before each feed, the nose is freed of any secretion. This is most easily achieved by inducing sneezing, by tickling the vestibule of the nostrils with a small pledget of cotton wool, well teased. After the muco-pus has been expelled, the nostrils are wiped dry, and a little Ung. Hydrarg. Ox. Flav. (B.P.) inserted as far into the nostrils as possible. Obviously only a small quantity of medicament can thus be introduced into the nasal cavities. Consequently a small reserve depot of the ointment is inserted into the vestibule of the nose, so that the warmth of the body, by melting the ointment, permits of the latter to spread itself more liberally over the nasal mucosa, during the period of sleep following the feeding. The results of such a simple treatment were excellent. It is probable, however, that the good results are due to the frequent cleansing of the nose, rather than to any specific action of the small amount of ointment used.

The instillation of menthol or camphor, dissolved in liquid paraffin was not carried out. Recent work seems to imply that such form of local therapy is not altogether free from danger (Ikeda, 1937). Douching of the/
the nose must not be countenanced under any circumstance. It would only tend to drive infection into the aural cavities.

For the harsh, dry and barking cough, which may be present, especially at night, and which may interfere with sleep, a simple sedative cough mixture may be given. A suitable prescription would be:-

\[
\begin{align*}
&\text{Tr. Camp. c.opii } 3\frac{1}{2} \\
&\text{Glycerin } 3\frac{1}{2} \\
&\text{Aqua ad } 3\frac{1}{2}\text{III.}
\end{align*}
\]

Sig.: \(\frac{1}{3}\) in warm water nocte.

Tonsillar infections are often troublesome to treat effectively in the young baby. Local applications are the only way of getting at the seat of the infection. The best drug for this local therapy is Gentian Violet, 1% aqueous solution, painted over the tonsils twice or thrice daily. If there is much pain on swallowing, or the associated adenitis is painful, then hot applications over the throat seem to afford relief. A very careful watch must be kept over such cases, as there is always the possibility of a retro-pharyngeal abscess developing. I have no experience in the use of the sulphonamide group of drugs in haemolytic streptococcal throat infections in these very young infants. Great care would have to be exercised in their administration.

For Stomatitis and Gingivitis, local applications of Gentian Violet, in solution as described above, act as a specific. Two or three applications per diem for
a few days rapidly clear up these conditions (Sutton, 1938).

Concerning the treatment of otitis media, there is considerable controversy. Many (Lyman, 1927, Odenal, 1928, Marriott, 1935, Deans and others) believe that as soon as such a focus of infection is diagnosed, paracentesis membrani should be performed without further delay. If necessary, a mastoidectomy might even have to be performed, if there were signs of mastoid involvement. They state that after a paracentesis has been carried out, little else may be required in the way of treatment. Others (Finkelstein, 1905, Wishart, 1930, Findlay, 1933) are against such procedures. Carmack (1930) is also of the opinion, and in a very critical review of the problem, stated that, especially in the case of mastoiditis, operation should not be attempted during the violent gastro-intestinal crises which commonly occur in such cases. In no case in the present series was operative interference of any type required. The aural conditions settled down under expectant treatment, or else, discharging freely after spontaneous rupture of the tympanic membrane.

The expectant treatment adopted here was the instillation of Glycerin Acid Carbol (⅛ B.P. strength) drops into the ears twice or thrice daily. The drops were warmed slightly before instillation, and afterwards the external meatus was lightly plugged with cotton wool. If/
If pain is severe, light packing of the meatus with gauze soaked in either 5% ichthyl and glycerin, or 10% Aluminium Acetate is effective. The latter drug is especially useful if there is any infection of the skin of the meatus, e.g. a boil, or even simple excoriation of the skin. If there is restlessness and pain, hot boracic fomentations applied over the ears, three hourly, together with the administration of antipyrine, ½-1 gr. is carried out. This relieves the pain, and frequently causes the ear drum to rupture spontaneously. If these simple measures do not give relief, and the child is not improving, then myringotomy should not be further delayed.

Otorrhoea, once established, should be treated by gentle syringing with normal saline, twice daily. This syringing must always be performed by a fully trained nurse, conversant with such treatment. After syringing, the ear is dried out, and a loose plug of cotton wool inserted at the entrance of the meatus. This plug merely acts as an absorbent of the discharge, and does not interfere in any way with proper drainage. So soon as the plug is moistened by the discharge, it is removed, and a fresh plug is inserted. Most of the cases in this series were treated thus, with particularly gratifying results. For the older child, and in the refractory infantile cases, insufflation of Iodine and Boracic acid powder, according to the formula of Sulzberger (Crooks, 1938/
1938) is an admirable method of treatment. In all cases, the Golden Rule in respect of otorrhoea, is to keep the ears clean and as dry as possible (Crooks, 1938). For those cases due to streptococcal or pneumococcal infections, help is to be expected from the drugs of the sulphonamide class (Blechmann, 1935, Douthwaite, 1937, Lucas, 1937, Crooks, 1938 and Hall, 1939). In streptococcal tonsillitis also, these drugs may be of service, but the organism must be streptococcus or pneumococcus (Douthwaite, 1937, Salama, 1937). The drug of this class of which I have had most experience, is Rubiazol. It is an excellent preparation, of low toxicity but I doubt whether as efficient as e.g. Sulphonamide-P (Burroughs and Wellcome).

Pyelonephritis should always be treated in hospital, but milder cases of pyuria may be treated at home, provided the home conditions are satisfactory and conducive to proper treatment. Alkalisation of the urine is the treatment par excellence in the acute stages. Mandelic acid and its derivatives should be reserved for later, when the acute stage has passed and the symptoms have subsided. Mandelates actually seem to aggravate the dysuria in the acute stage. In the one case of pyuria encountered in this series, response to alkalis alone, was excellent. Reports on the use of the sulphonamide series of drugs are encouraging (Huber, 1936, Pernice, 1936, Helmholtz, 1937, Payne, 1939, Cruikshank, 1939).

Finally/
Finally, it is always advisable to exhibit iron, during the period of convalescence from the diarrhoea (Smallwood, 1939). The preparation of iron which was used in the cases in this series was Ferri et Ammon. Cit. There seemed to be no contraindications to its use, in favour of any other iron preparation.
SECTION V.

CASES.
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CASES.

Case 1.

History.
Child was artificially fed on cow's milk and water mixtures from birth till 8 months of age. Since that time, the child had a very bad feeding history. Mother was a cook in service. Consequently she was unable to give the attention to the child which was necessary. As a result, feeding was irregular, and the food presented to the child often quite unsuitable for its age. The little girl had attacks of diarrhoea alternating with constipation, had a poor appetite, and there was a slow and progressive loss of weight.

As a consequence of the mother obtaining a post as cook, in a Day Nursery under the Corporation of Edinburgh, the child was admitted as an inmate to the Nursery. A careful watch could be kept on the child's diet, and note made of her progress.

Complaint on admission.
Frequent attacks of diarrhoea and loss of weight.

Examination on admission.
A small, thin, red-haired girl, bright eyed but listless and pale. Undernourished and soft. Walked well when persuaded to do so, but persuasion was difficult/

Alimentary System. Appetite: poor. There had been attacks of vomiting on occasion. Bowels: very irregular; frequent attacks of diarrhoea, during which the stools were loose, undigested, pale and extremely offensive. In the intervals between the bouts of diarrhoea the bowels tended to be constipated.

Teeth: \[\frac{102}{102} | \frac{201}{201}\], good. Tongue: heavily coated, especially over the posterior third. The fur was yellowish brown. Breath: heavy. Throat: clean.

Abdomen: rather full and tumid. There was some degree of tenderness over the right iliac fossa. Liver and spleen: not palpable.

Other Systems: no abnormalities were detected. No evidence of parenteral infection. Ears and urine were clear.

Laboratory Investigations.

Three specimens of stool were examined bacteriologically. No pathogenic organisms were isolated on any occasion.

Tuberculin Tests: the Mantoux test was carried out. Reactions were negative to tuberculin dilutions of 1:10,000, 1:1,000 and 1:100.

Wassermann Test - negative.

Diagnosis/
Diagnosis.

Dietetic Diarrhoea.

Progress Notes.

10.8.38. Admitted to Day Nursery. Child was put on ordinary toddlers' diet. This was continued until 26.9.38. There was no appreciable improvement during this period, the weight chart showing very erratic changes, and the diarrhoea still showed intermittent recurrences.

27.9.38. till 10.10.38. Was put on a diet such as is ordered for Chronic Intestinal Indigestion, together with a rhubarb and soda mixture. No improvement could be said to have taken place during this period.

11.10.38 till 13.10.38. Put on the raw apple diet. Child at first took only 2 tablespoonsful of the apple pulp. This quantity was rapidly increased to 4, then 5 tablespoonsful of the pulp, as the child's appetite improved. This diet was given 3 hourly, together with free administration of weak, unsweetened tea.

Immediate improvement was evidenced on the second day of this treatment. The stools became less offensive and less loose. The tongue began to clean, and the appetite improved. The colour seemed to be returning to the cheeks. The last stool passed on the second day of this treatment was fairly well formed, inoffensive, brownish in colour. Symptoms were still further improved on the third day. The loss of weight during this three/
three days' treatment was only \( \frac{3}{4} \) ounces.

14.10.38. The apple pulp was continued in two feeds per diem for this day and the following 6 days. A start was made with feeds of undiluted skimmed cow's milk. This was taken well.

16.10.38. Improvement maintained. Child now having two stools per day. These were well formed and practically odourless. Weight increasing. Child was bright. Taking a modified toddler's diet well. Additions being made daily to the diet.

24.10.38. Recovery was apparently complete. Now on full diet. Running about, happy and contented. Ferri et Ammon. Cit.grs v.t.i.d.


Case 2.

Dora A. Aged 2 months. Only child. Illegitimate.

History.

Child was full-time. Breast fed for 10 days while mother was in hospital. Thereafter child was artificially fed on a cow's milk and water mixture 3 hourly. 2 ounces milk and 1 ounce water, with 1 teaspoonful cane sugar. Admitted to an institution when one month of age, with a view to adoption. Mother out working. After admission, the child was kept on the same feed at/
at the same feeding intervals. Progress was satisfactory until 31.3.39, when at 9 weeks of age, child suddenly developed an acute attack of diarrhoea, with frequent loose, green and slightly offensive stools. There were occasional lumps of mucus, but no blood.

**Examination on Admission.**

A very healthy child. Anterior fontanelle: 2 fingers' breadth. All systems were normal.

31.3.39. Child obviously ill. Fretful and irritable. Off feeds. Attacks of screaming occurred, associated with drawing up of the legs. No flatus was passed after subsidence of these attacks of colic. Skin: hot and dry. Anterior fontanelle: rather tense. Temperature: 103.2°F.


**Abdomen.** Nothing to note, apart from slight distension. No tenderness.

**Respiratory System.** No symptoms. An occasional rhonchus was heard on auscultation of the chest.

**Aural examination.** Both ear drums very injected and red. Left drum bulging with rupture apparently imminent.
imminent in the posterior inferior quadrant. No discharge from either ear.

Other Systems. All appeared to be clear. Urine quite normal.

**Laboratory Investigations.**


Wassermann Test. Negative.

**Diagnosis.**

Parenteral Diarrhoea due to Double Otitis Media.

**Progress Notes.**

31.3.39. Glycerin and Carbolic drops instilled into ears twice daily, after slight warming. Hot fomentations to the left ear, 4 hourly. Antipyrine gr. 1/2 twice daily.

**Dietetic measures.** Apple diet administered. 2-3 teaspoonful 3 hourly, with free fluids in the form of boiled water without glucose.


Stools still green and rather loose, but offensiveness gone. Glycerin and carbolic drops continued to left ear, together with hot fomentations. Spirit and peroxide drops to the right ear.

2.4.39/
2.4.39. No change in aural conditions. Child somewhat better. Taking 3-4 teaspoonsful of apple now, with less persuasion. Temp. 100.0°F.

Stools definitely reduced in frequency, but still having some degree of frequency. Still quite green, but odourless.

3.4.39. Right otorrhoea still profuse. Left ear improving, still inflamed but not so tense and bulging. Temp. 98.8°F.

Stools remain green and still rather frequent. No odour. Apple increased to 1 dessertspoonful 3 hourly.

4.4.39. Left ear now resolving, not bulging and less red. Right otorrhoea profuse. Child brighter and sleeping well. No attacks of colic now. Temp. 98.8°F.

Stools much improved. Typically apple stools, viz. reddish-brown, formed and odourless. Reduced in frequency from 6 per diem to 2.

Apple mash continued noctemaneque, 1 dessertspoonful. Milk introduced into diet. 1 ounce of boiled, skimmed milk and 1 ounce of boiled water, given 3 hourly. No sugar added.


Stools: normal. Having 2½ ounces of milk with 1 ounce of water, and ½ drachm of dextrimaltose, 3 hourly. Weight steadily increasing. Apple pulp still being given night and morning. Gentle syringing of the right ear instituted twice daily, normal saline being used.

14.4.39. Very well now. Taking 3 ounces of milk and/
Case 3.

Alexander S. Aged 11 months. Third child.

History.

Full-time. Breast fed for 3 months. Thereafter, until 6½ months of age, was fed on Nestle's milk. Weaning was then commenced, and child was now having a mixed diet of soup, egg yolk, vegetables, fish, milk pudding and a cereal. Child had been very well till he was about 9 months of age. At this time he began to have frequent attacks of diarrhoea. These attacks were sudden in onset, lasted a few days, and just as suddenly stopped.

25.8.38. Was violently sick, vomiting up undigested food. This was immediately followed by a sharp bout of diarrhoea, with loose, green very offensive stools. No mucus nor blood. The vomiting abated after a few hours, but the diarrhoea continued intermittently for the succeeding week. The mother gave the child a dose of castor oil at the outset of the illness. It was without effect. The motions remained foul and offensive and loose until seen on 2.9.38.

Examination/
Examination on 2.9.38.


Alimentary System. Appetite: very poor. Taking only boiled milk at irregular intervals. Drinking well. Tongue: fairly clean. Breath: heavy and disagreeable. Throat: clean. Teeth \( \frac{102}{002} \) \( \frac{201}{100} \).

Abdomen. Soft, apparently not tender. Evidence of loss of subcutaneous tissue when skin picked up between finger and thumb. There was a peculiarly disagreeable odour about the child.


Laboratory Investigations.

One specimen of stool was examined bacteriologically. No pathogenic organisms were isolated.

Diagnosis.

Dietetic Diarrhoea, subacute in type, after an acute onset one week previously.

Progress Notes.

2.9.38. Child was promptly put on the starvation regime for a period of 24 hours, with free fluid administration. This was impressed upon the mother.

Thereafter, gradual introduction of skimmed milk and milk pudding was made. The Health Visitor made daily/
daily visits to ensure that the mother was carrying out the prescribed treatment. The appetite for the succeeding week was poor, however, and though the boy took water well, it was only with the greatest difficulty that he was persuaded to take any milk or milk pudding.

The diarrhoea abated, but did not cease, the stools remaining loose and offensive, but not green. The looseness of the bowels showed a tendency to be worse at nights, when it was associated with colicky pains.

9.9.38. Child seen again. Was still losing weight. Not looking at all well. Examination of the various systems again revealed no evidence of a parenteral infection. Child was put on the Apple diet, to commence on 10.9.38.

12.9.38. Seen again. Has been taking 4 tablespoonful of raw apple pulp every 4 hours. Drinking freely of boiled water. Looking much better. Stools now inoffensive, two per diem. Complete disappearance of nocturnal diarrhoea and colic. Stools also formed, brownish in colour. Breath 'sweeter'. Actually has put on 2 ounces of weight since seen on 9.9.38.

13.9.38. Apple increased to 5 tablespoonful 4 hourly. Improvement continued. Deemed advisable to extend the apple diet for a further 24 hours.

14.9.38. Start made with skimmed milk in small quantities. (3 ounces milk and 1 ounce boiled water) 4 hourly. Apple continued night and morning.

16.9.38. Continued improvement. Taking milk feeds/
feeds well. Stools now normal and remaining so. Whole milk started.

20.9.38. Diet increased, soup added, some cereal and a little boiled fish.

30.9.38. Taking a normal diet now. Apple night and morning discontinued. Colour back to cheeks. Ferri et Ammon. Cit. grs. x.t.i.d. Gained 1 lb since last visit, i.e. 10 days.

25.10.38. Has been very well. Now on full diet. Has cut 2 low-first molars since 30.9.38 without upset.

**Case 4.**


Breast fed until 6 months of age. Now on an all round diet. Had always been healthy save for an occasional 'cold'.

13.2.39. Child suddenly taken ill with vomiting, anorexia and difficulty in swallowing. A few hours after this acute onset diarrhoea began. The stools became green and loose. This got worse in the course of the day, and during the whole day passed 7 stools of such a type as described. Swelling on both sides of the neck was also noticed towards evening.

Examination on 14.2.39.

A fine sturdy child, but obviously ill. Feverish-
Temperature 103.0° F. Face: flushed. Slight swelling of the tonsillar lymph glands, especially the left.


Other Systems. A trace of albumin was the only abnormality found on examination of these systems.

**Laboratory Investigations.**

A throat swab was taken. This gave a slight growth of haemolytic streptococci.

**Diagnosis.**

Parenteral Diarrhoea due to Follicular Tonsillitis and associated Adenitis.

**Progress Notes.**


Taking apple well, 2 tablespoonsful 4 hourly. Drinking well. Motions: very loose and green. Buttocks becoming/
becoming reddened.


Continues to take the apple well, and drinking very freely. Stools: inoffensive, but rather loose. Less green.


Child much brighter, taking feeds of milk and water 4 hourly very well. Stools: improved, less frequent, not green and not offensive. Apple being continued twice daily.

22.2.39. Flare up of all symptoms. Throat: increased redness and return of difficulty in swallowing. Adenitis: tender and increasing in size. No evidence of a retropharyngeal abscess. Ears and urine: clear. Temperature 103.8°F.

Diarrhoea returned, profuse and loose and undigested. Not particularly offensive. All other systems appeared to be clear.

Starvation regime instituted, after a preliminary dose of castor oil had been given. Glucose (5%) in boiled water taken freely. No vomiting.

24.2.39/

Stools less frequent and less loose, but still rather green. Having 2 ounces of milk and 2 ounces of water, but no carbohydrate, every 4 hours.


Stools almost normal. Slight traces of green on occasion. Having 7 ounces milk and 1 ounce water with 1 drachm of sugar, 4 hourly. Taking well. Free fluid administration inter cibos.


Stools normal. On a full diet.

Case 5.

James B. Aged 8 months. Second child. Legitimate.

History.

Child was breast fed until 6 months of age. Weaning was then begun. This was accomplished successfully in some 5 weeks. He was having at the time of this illness, whole milk, cereal (Farex), milk pudding, a small piece of fried bread at breakfast, vegetables and fruit.

17.8.38. Seized with acute diarrhoea, the stools rapidly/
rapidly losing their faecal characters, and becoming fluid and watery, at first green then yellowish brown. Very offensive. Associated with this diarrhoea, but following on its onset, vomiting occurred. This vomiting was at first very severe, but it soon settled. The child was feverish and fretful.

He was seen some 6 hours after the onset of the illness.

Examination.

An obviously ill child. Irritable. Nutrition: good, but signs of dehydration becoming manifest, and skin could be pinched up between finger and thumb. Anterior Fontanelle 1½ fingers breadths. Low tension. The face was pale, and the child was constantly moistening its lips with its tongue. Temperature: 102.4°F.

Alimentary System. Appetite: lost. Vomiting: severe for the first 2-3 hours of the illness, but had apparently settled by the time of examination. Thirst: very marked. Stools: very frequent. Had passed 5 loose, watery foul smelling motions since onset of illness some 6 hours previously.


Nose: clear. No discharge. Ears: appear to be normal.

Abdomen: rather swollen, but quite soft on palpation. Moved freely on respiration. No tenderness was apparent. Evidence of loss of subcutaneous tissue manifest, but no loss of elasticity of skin.

Other/
Other systems. Nothing to note which was pathological.

Laboratory Investigations.

Two specimens of stool were examined bacteriologically. A pathogenic organism was not isolated on either occasion.

Diagnosis.

Non-specific enteral infection. No evidence of a dietetic indiscretion or of a parenteral infection was ever forthcoming.

Progress Notes.

As the child's home was a good one, it was decided to treat him at home. The older child went to live with a grandmother. It was of school age.

Child was put on the starvation regime, after a preliminary dose of castor oil. Fluid in small quantities was given every 1 hour. 5% glucose in boiled water was the solution used. This period of rest lasted for 12 hours. The child took the fluid well, there was no vomiting. The stools improved slightly towards evening.

18.8.38. Child looking better. Less evidence of dehydration to-day. Fontanelle: less soft, pulsation present. Temperature: 99.8° F.

Stools: had 4 stools since seen on 17.8.38. These were still fluid, but less offensive.

As child's condition was much improved from the previous/
previous day, it was deemed that a start could be made with a dilute milk feed. Consequently, 1 ounce of skimmed boiled milk and 1 ounce of boiled water was started every 3 hours. No carbohydrate was added, but the glucose was continued in the water given between feeds.

19.8.38. Continued improvement. Stools much better, and less frequent. Only 4 were passed during the previous 24 hours. No evidence of any parenteral infection.

Feeds cautiously increased to 1½ ounces of skimmed milk and a similar quantity of boiled water. Glucose in water continued between feeds. Less of this solution seemed to be desired, as appetite improved.

22.8.38. Child brighter, now able to smile. Temperature: 98.8°F. Stools now almost normal. Milk increased to 2 ounces and water to a similar amount.


30.8.38. Progress continues favourably. On 6½ ounces milk and ½ ounce water. Instructed to add dextrimaltose/
dextrimaltose, \( \frac{1}{2} \) drachm per feed. Still taking sips of water between feeds.

2.9.38. Progress well maintained. Beginning to put on weight now more readily. Advised to introduce whole milk into feeds, by replacing 1 ounce of skimmed milk per feed with 1 ounce of whole milk, each day.


16.9.38. Return to a normal diet being made satisfactorily. No further upsets.


Case 6.

Stuart McD. Aged 17 months. 6th child.

History.

Child was breast fed for only 3 weeks. Thereafter he was brought up on cow's milk and water mixtures until he was 9 months old. At that time, weaning was begun. History was uneventful save for occasional 'colds'. Owing to the parents having been evicted from their house, the child was taken into a Day Nursery, on 22.4.39.

25.5.39. Child taken with a sudden bout of sickness, followed by the occurrence of diarrhoea. The sickness was quite acute, and nothing would lie on his stomach/
stomach, not even sips of water. About an hour after the onset of the vomiting, the boy passed a loose green foul smelling motion. This was followed shortly afterwards by another similar stool. Between the passage of these two motions, there had been abdominal pain, in the nature of colic of intestinal nature. The diarrhoea continued, the motions becoming fluid, brownish and less offensive.

**Examination.**

Child was seen some 8 hours or so after the onset of the illness.

Child was in bed - an unusual thing for this particular boy, who was normally so active. Listless and pale. Temperature: 100.6°F. Anterior fontanelle: almost closed. No depression or lack of tone. No obvious signs of great loss of fluid.

Alimentary System. Appetite: none, but very thirsty. Took water well without vomiting at time of examination.

Mouth: clean, moist. Teeth: \( \frac{102}{112} \frac{211}{211} \). Tongue: thickly coated with a brownish fur, especially over the posterior third. Breath: foul. Throat: clean. No enlarged tonsillar glands.

Abdomen. Moved freely on respiration. Slight tenderness over the whole abdomen. Greatly exaggerated intestinal movements on auscultation of abdomen. No evidence of appendicitis or similar infection.

Other Systems. Ears: appeared to be normal.

Urine/
Urine: normal to chemical and microscopical examinations. It was however, reduced in quantity, and had a heavy deposit of urates. Other systems: normal.

**Laboratory Investigations.**

Two specimens of stool were examined bacteriologically. No pathogenic organisms were isolated.

**Tuberculin Tests.** The Mantoux test was carried out. Reactions were negative to tuberculin dilutions of 1:10,000, and 1:1,000, but weakly positive to 1:000.

**Diagnosis.**

At first it was thought that the case was one of enteral infection, probably non-specific. A closer investigation of the diet, however revealed that the child's mother had visited the nursery earlier in the day of 14.5.39. At this time she had given the child a sugar coated cake of rather doubtful quality. This occurred without the knowledge of the staff. It was only ascertained at a later date. The subsequent sickness and diarrhoea developed in the early hours of the morning of 25.5.39. Consequently it was considered that the case was probably one of dietetic diarrhoea rather than an enteral infection. At no time during the illness was a parenteral infection ever apparent.

**Progress Notes.**

The child was put on the apple diet whenever seen (25.5.39). No purgative was administered as a preliminary to this treatment. In view of the sickness which/
which had taken place, it was thought that this diet might not prove very successful, but that a trial ought to be given to it at any rate. 2 tablespoonsful of raw apple pulp were administered every 3 hours, together with very weak and unsweetened tea, as much as the child would take without increasing the sickness. Whether by coincidence or as a result of the diet, there was no return of the vomiting. Child took the apple well, for the rest of the day.

26.5.39. Child brighter and taking freely of the weak tea. Demanding more apple which was accordingly increased to 3 tablespoonsful, or more if the child was able to take it.

Stools: 4 were passed since institution of treatment. The first three showed no great improvement over those previously passed, but the fourth was less fluid, and practically devoid of offence.

27.5.39. Apple had to be increased to almost 6 tablespoonsful by the evening of 26.5.39. Child also taking fluids well.

Stools: these showed a remarkable improvement from the previous day. They were now formed, of a reddish brown colour and quite odourless. Only 2 were passed in the previous 24 hours. Child wanting to get up, and jumping about in bed.

28.5.39. Though the child had only had the diet for 2½ days, it was deemed that he could now start a modified milk diet. Lactic acid skimmed milk, 5 ounces 4/
4 hourly was given. No carbohydrate was added. Instructions were given to increase the milk by 1 ounce per day if progress was maintained, since the child was not to be seen until 30.5.39.

30.5.39. Well. Sugar added to feeds in proportion of 1/3 drachm per feed. Simultaneously, whole milk was added to the feeds, viz. acidifying 6 ounces of whole milk, and giving one such feed for the first 24 hours, two in the following 24 hours, and so on in this manner. When the child was taking this full milk diet, acidification was reduced to every second feed, and ultimately stopped. Slow additions were then commenced, and a full toddler's diet achieved by 13.6.39, when child was considered cured.

Case 7.

Ian V. Aged 7 months. Only child. Legitimate.

History.

Child had been breast fed until 6 months of age, and was now in the final stages of weaning. Had always been healthy and mother was very conscientious, giving the child excellent attention.

2.11.38. Child feverish with a slight nasal discharge, and some impairment of appetite.

3.11.38. Symptoms of a 'cold in the head' more evident to-day. Stools rather loose, greenish and undigested/
undigested, but not offensive, though having a faecal odour. During the day he passed 4 such motions. Normally child passed one or at most two motions per diem.

Examination on 4.11.38.


Mouth: clean, moist. Teeth $\frac{0}{1} | \frac{0}{0}$. Tongue: quite clean. Throat: slightly reddened. No evidence of specking of tonsils. No enlarged glands.

Abdomen. Moved freely on respiration. No tenderness.

Ears: appeared to be quite normal. Urine: considerable difficulty in obtaining a specimen. This was not obtained until the following day. It was found to be clear, with no abnormalities.

Other/
Other Systems. Nothing abnormal to note.

Laboratory Investigations.
None were carried out.

Diagnosis.
Parenteral Diarrhoea due to simple Rhinitis.

Progress Notes.

4.11.38. Child was treated by the starvation method. An initial dose of castor oil was administered but not repeated. The child was starved for 12 hours, but during this period of rest, the child was encouraged to take as much boiled water as possible. This it took well. Local treatment to the nose was given, using Ung. Hydrarg. Ox. Flav. (B.P.) At 10 p.m. the same day, a feed of 1 ounce of skimmed milk and 1 ounce of boiled water was given. This the child seemed ready for, and took it well.

5.11.38. Nasal catarrh seemed to be better. Child brighter. Taking water well. Motions: had had 4 since being seen previous day. These were still rather loose and greenish. Child, however, was somewhat better generally. Ears: appeared to be normal. Chest: remained clear.

8.11.38. Nasal catarrh much improved. No more snuffling. Motions: practically normal now. Only 2 stools passed per day for last two days. These were very satisfactory both in consistence and colour. Taking less water, and more of a milk feed. Having 3 ounces of skimmed milk and 3 ounces of water 4 hourly. Mother advised to increase the milk by $\frac{1}{8}$ ounce per feed daily, while keeping the water constant at $2\frac{1}{2}$ ounces/
ounces per feed, until total volume of feed was 7 ounces. Then instructed to increase the skimmed milk \( \frac{1}{2} \) ounce per feed per diem, reducing the water correspondingly, until getting 7 ounces of skimmed milk.


15.11.38. Having 6\( \frac{1}{2} \) ounces of skimmed milk and \( \frac{1}{3} \) ounce of water. Sugar now being added, \( \frac{1}{3} \) drachm per feed. Instructed to gradually replace the skimmed milk with whole milk. Good progress continues.

25.11.38. Getting on to a transition diet with no upsets. Taking egg yolk, milk pudding, strained vegetable soups and broths. Considered to be cured.
For purposes of clarity, the remaining 243 cases have been arranged into groups. The number of cases in each group will be given, the age and sex incidence described, and a brief commentary on the history and treatment of each group will be discussed. The groups are as follows:

(1) Breast fed infants - Parenteral Infection.
(2) Breast fed infants - No Parenteral Infection.
(3) Enteral Infection in(a) Artificially fed infants.
       (b) Infants on a mixed diet.
(4) Dietetic Causes in(a) Artificially fed infants.
       (b) Infants on a mixed diet.
(5) Parenteral Infection in(a) Artificially fed infants.
       (b) Infants on a mixed diet.

Each group will be described in the order in which they are enumerated.

**Breast fed--Parenteral Infection.**

This group included not only those cases which were completely breast fed, but also those instances where complementary or supplementary feeding was employed. There were 27 cases in this group, and of these 20 were wholly breast fed, and 7 were receiving either a complementary or a supplementary feed in addition to the breast.

**Age Incidence.** Average age: 4 months. Age extremes: 1 month - 8 months.

**Sex/**
Sex Incidence. Males. 10. Females. 17.

Aetiological Factors. The commonest parenteral infection affecting the infants in this group, was the common cold. This manifested itself mainly as a simple rhinitis, with or without some degree of pharyngitis. This infection accounted for some 14 cases. There were never any signs of aural infection in these cases at any time during their illness. Definite otitis media accounted for 5 cases, and this infection was bilateral in 2 instances. Tonsillitis and bronchitis were judged to be the causes in 6 cases. Vaccination and Whooping Cough were the causes of 1 case each.

History. In most of the cases, the diarrhoea was not the first indication of an upset. Evidence of the parenteral infection was usually present at the time when complaint was made of diarrhoea. The diarrhoea was not severe in any of the cases in this group, save in one. This exception occurred in the case of the infant suffering from Whooping Cough. The diarrhoea in this instance was so severe at one time that the question of hospital treatment was seriously considered. In view of the fact that the child was breast fed, it was decided to continue treatment at home under constant supervision. Recovery, though slow, was ultimately complete.

Treatment. The starvation regime was carried out in all instances. None of this group was treated with the raw fruit diet. Local treatment to the focus of infection was carried out simultaneously. The results were/
were quite satisfactory. The duration of the period of 'starvation' was never more than 24 hours, usually 12 hours. During the period of 'starvation', free fluid administration was encouraged. The fluid given was boiled water in every case. After this preliminary period of rest, a slow return was made to breast feeding.

Breast fed—No Parenteral Infection.

There were no cases of Enteral infection in this group. The causes were dietetic in all instances.
There were 13 cases in all, of which 11 were completely breast fed, and 2 were receiving complementary feeds of milk and water mixtures.

**Age Incidence.** Average age: 3 months. Age extremes: 1 month - 12 months.


**Aetiological Factors.** The commonest cause of the diarrhoea in this small group was mismanagement of the feeding in some form or another. This was most usually the too frequent application of the infant to the breast, the child being put to the breast whenever it cried. This crying was not due to underfeeding in any case. Rather was it due to a degree of overfeeding, associated with abdominal colic. Consequently, the frequent application of the child to the breast aggra-
vated the tendency to overfeeding, and precipitated the attacks of diarrhoea. This diarrhoea was frequently combined with vomiting. The two cases where comple-
mentary/
complementary feeding was being employed, were being overfed, the complementary feed was too strong in each case.

**History.** The diarrhoea was usually gradual in onset, with the occurrence of loose, green, undigested and offensive smelling stools. Less commonly, the diarrhoea was sudden in onset. In this latter type of case, vomiting was often a prominent symptom, much more prominent than in the cases where the intestinal upset was of a more gradual onset. Toxaemia was not prominent in any of the instances in this group. The temperature was but slightly raised. There seemed to be an aggravation of the already existing abdominal colic, but there was no evidence of any abdominal tenderness.

**Treatment.** The 13 cases were all treated by the starvation regime. The results of this form of therapy were eminently satisfactory. The period of starvation, usually lasting 12 hours, resulted in mitigation of the symptoms, and lessening of the diarrhoea. A dose of castor oil (1 drachm) was given at the outset of treatment in each case, but it was not repeated. There was never any need for further doses. There seemed to be less tendency to relapse in these breast fed babies who suffered from dietetic diarrhoea, than obtained in similar cases occurring in the artificially fed infants. The institution of regular feeding was particularly stressed, both as a prophylactic and curative measure in this group.
Enteral Infection—in Artificially fed infants.

This group consisted of babies fed entirely on cow's milk and water mixtures. There were 9 cases in this very small group.

**Age Incidence.** Average age: 5 months. Age extremes: 3 months - 8 months.

**Sex Incidence.** Males: 8. Females: 1.

**Aetiological Factors.** 3 cases were due to infection with the *B. Dysenteriae Sonne*. These cases were removed to an isolation hospital. Twins accounted for 2 of these cases, and one of them died from the infection. The other twin recovered after a rather stormy convalescence. The third child also recovered.

The remaining 6 cases were judged to be due to a non-specific infection of the bowel, probably *B. Coli* in origin. Stool specimens were examined, at least twice, in 4 of these cases. On each occasion, no pathogenic organism was isolated. The other 2 cases did not have specimens of stool examination.

**History.** The illness in all of these 9 cases was of sudden onset. Vomiting usually ushered in the illness. This was rapidly followed by diarrhoea, which was profuse from the commencement of the disease. The stools rapidly lost their faecal characters, and became watery and foul smelling. In the dysenteric cases, though each was seen in the early stages of the disease, the typical small green stools, streaked with blood, and containing a fair amount of mucus, were already present. In the case/
case of the twins, the toxaemia was extreme.

Generally, the toxaemia was quite severe in the other cases. There was fever, with dry coated tongue, and some degree of dehydration was present without exception. Abdominal pain and tenderness were usually present. In one of these non-specific cases, the phenomenon already referred to, viz. the disappearance of an eczema during the acute bout of diarrhoea, was observed, only to return after recovery from the acute illness.

Treatment. 4 cases were treated by the starvation regime and the remaining 2 with the apple diet. In the former instances, the method of treatment adopted was as has been outlined in another section. Each case received an initial dose of castor oil. This dose was not repeated. Free fluid administration, short of producing or aggravating existing vomiting, was insisted upon, during this period of 'starvation'. The fluid was given frequently and in small amounts. Boiled water with added glucose was used. The return to modified cow's milk feeds was made very gradually. Recovery was complete in all 4 instances, though in 3 of them progress seemed to be rather slow.

2 cases were treated with the raw apple diet. This was given 3 hourly, together with as much water as the child would take. The improvement in both of these cases was indeed remarkable. The toxaemia rapidly abated,
abated, the stools became less frequent, and especially less offensive within some 12-24 hours of the institution of the diet. Modified cow's milk feeding was instituted on the 4th day of treatment, but the apple was continued for 2 feeds in the day for a further period of one week.

The results in these two cases were eminently satisfactory and compared most favourably with those treated by the starvation regime. The raw apple was well taken by the two infants, both aged 5 months.

**Enteral Infection—in infants on a mixed diet.**

There were 15 cases in this group. All had been weaned and were having a mixed diet.

**Age Incidence.** Average age: 17 months. Age extremes: 10 months - 23 months.

**Sex Incidence.** Males: 7. Females: 8.

**Aetiological Factors.** In 3 instances, a specific pathogenic organism was isolated. *B. Dysenteriae Sonne* accounted for 2 cases, and *B. Dysenteriae Flexner* for the third case. In a fourth instance, the child was treated in an isolation hospital for clinical dysentery. Specimens of stool were all negative to pathogenic organisms. The child was classed as a non-specific dysentery. Diarrhoea persisted after discharge from hospital, however, and treatment was required when the child returned home.

The/
The remaining 11 cases were judged to be instances of non-specific enteral infection. In 8 of these cases, one or more specimens of stool were examined and proved negative bacteriologically.

**History.** The clinical history was practically identical with that presented by the group of younger infants affected by an enteral infection. The infectivity of the non-specific cases was very low. In only one instance were the other members of the family affected by diarrhoea and/or vomiting.

**Treatment.** 9 cases were treated by the starvation method, and 3 were treated with the raw apple pulp. One of the latter cases was the non-specific dysentery case mentioned supra, which required treatment after discharge from hospital. In this and the other 2 cases, the apple was very successful in clearing up the diarrhoea and combating the toxaemia.

The cases which were treated by the starvation regime also responded satisfactorily. It was felt, however, that they did so less rapidly than those submitted to the apple pulp. In 3 instances there was a recurrence of the diarrhoea during convalescence. In these 3 cases, there was an associated return of the offensiveness of the stools.

Comparing the results of these two forms of treatment in this enteral group of cases, the impression was formed that the employment of the raw apple diet had certain/
certain advantages over the more orthodox method of therapy. It produced more rapid amelioration of the symptoms. However, the series is too small to be able to draw any really useful conclusions.

Dietetic Causes—in Artificially fed infants.

These were infants which were entirely fed from the bottle, usually on cow's milk and water mixtures. There were 28 cases in this group.

Age Incidence. Average age: 5½ months. Age extremes: 1 month - 10 months.

Sex Incidence. Males. 17. Females. 11.

Aetiological Factors. The commonest causes encountered were due to feeding on too strong mixtures of milk and water, and the addition of such cereals as oat flour to the diet of the very young infant ere it was capable of digesting and assimilating such additions. Oat flour was the commonest of the cereals used in these infants. Usually the children did well on it for a time, and the mother was encouraged to increase the quantity. This resulted in a breakdown of the digestive processes under the strain of the unsuitable addition to the simple milk and water mixtures. In other cases, when the mother took it upon herself to add a 'food' to the milk-water mixtures, the digestion of the infant promptly revolted, with resulting vomiting and diarrhoea, and the other signs and symptoms of an acute digestive upset.

The/
The fat content of the milk seemed to prove too much for the digestion of some of these infants in this group. This was particularly noticeable in the warm summer months, and attacks of dietetic diarrhoea were fairly frequent. Skimming the milk is required in these cases. This was done both as a curative measure and also as a prophylactic one.

The too enthusiastic use of malt extracts was occasionally judged to be the cause of the diarrhoea. Stoppage or reduction of the extract usually resulted in improvement in the child's condition.

**History.** The onset of the diarrhoea in this group of cases was usually somewhat gradual, but occasionally it was fairly sudden. The motions were always loose, green, undigested and extremely offensive, especially in the early stages. Toxaemia was manifest to a greater or lesser degree, and a low grade pyrexia was the rule. Weight was lost in most cases, rapidly in those with a sudden onset, more slowly in those with a gradual onset.

**Treatment.** 18 cases were treated by the orthodox starvation regime, and 10 with the raw apple pulp. The results in both instances were quite satisfactory, but there was little question that those treated with the raw fruit responded more rapidly. In 4 instances in which the raw apple was used, the intensive course of apple could be restricted to 2 days, and a return to modified milk feeding was possible on the third day instead of the fourth. The apple in all those cases so/
so treated, was continued twice daily for a further period of a week after the intensive course. A further point concerning the after-results of the apple treatment was the definite absence of a tendency to relapse—a feature of not unusual occurrence in those treated by the starvation method.

Dietetic Causes - in infants on a mixed diet.

There were 38 cases in this group. All were completely weaned and having a mixed diet.

Age Incidence. Average age: 22 months. Age extremes: 9 months - 23 months.

Sex Incidence. Males. 20. Females. 18.

Aetiological Factors. The common causes in this group were indiscretions of diet, such as, the giving of unsuitable and indigestible articles of diet to the child. Fried foods, prepared and recooked foods, unripe fruits, and vegetables which are notoriously upsetting to the young child, e.g. turnips, new potatoes, and cabbage, to mention only a few. Ice cream in excessive quantities, and consumed at the wrong times was a frequent cause in the summer months.

History. The onset of the diarrhoea was often sudden. Vomiting was often present in the early stages. The motions were loose, green and offensive. Undigested material was frequently present in these stools. The temperature was raised, often to 102 F. or more. Appetite was lost or greatly impaired, and the child looked ill.
The tongue was thickly coated with a dirty brown fur and the breath was heavy. Dehydration was occasionally quite marked, but never extreme.

The cases judged to be due to the rather liberal consumption of ice cream were particularly rapid and sudden in onset. This was a rather striking feature. The diarrhoea, associated almost without exception with vomiting, set in within a few hours of the time of consumption of the cream. There was always, of course, the possibility of an enteral infection in these latter cases. One or two had specimens of stool examined bacteriologically, but no pathogenic organisms were isolated.

**Treatment.** 25 cases were treated in the orthodox starvation method, and 13 with the apple diet. The results were encouraging in both instances. Generally the cases in which the starvation regime was employed, responded well. In a few instances, however, where the diarrhoea was of long standing and more subacute in type, the starvation method was of less pronounced value. The diarrhoea tended to recur even when the greatest care was taken in carrying out the prescribed regime.

On the contrary, the cases treated with the raw fruit diet did not show this tendency. The motions rapidly improved, the offensive odour disappeared quickly, almost as if by magic, and during the transition periods from apple diet to milk diet and on to light/
light diet, there was no apparent tendency to recurrence of symptoms. This was in direct contrast to what took place in some of the cases treated on more usual lines.

Taking this complete dietetic group as a whole, 43 cases were treated by the starvation method, and 23 were submitted to the apple diet. It may be said that, though most cases responded well to therapy, the better results were obtained in those instances in which the apple pulp was employed. The rapidity of the beneficial action and the absence of a tendency to recurrence were the main features in the cases which were submitted to this raw fruit diet. Out of the 23 cases which were treated with the apple, 4 received Aplona, in lieu of the raw fruit. There were no noteworthy benefits to be derived from this particular preparation. The unpleasant taste of the Aplona was a decided disadvantage mitigating against its free administration.

**Parenteral Infection - in Artificially fed infants.**

There were 43 cases belonging to this group. The babies were all bottle fed, on cow's milk in some modified form or another.

**Age Incidence.** Average age: 5 months. Age extremes: 1/2 month - 9 months.

**Sex Incidence.** Males: 23. Females: 20.

**Aetiological Factors.** The common infections accounting for these cases of diarrhoea were mainly respiratory in type. Simple rhinitis or rhino-pharyngitis/
rhino-pharyngitis accounted for the majority of the cases. Otitis media was judged to be the cause in 12 instances, and bronchitis accounted for 4 cases. The remainder comprised a heterogeneous group including tonsillitis, pyuria, 1 case, and septic spots of the body, 1 case.

**History.** In most, though by no means all, cases a history of close contact with an adult who had suffered or was suffering from, an upper respiratory infection, was admitted. The snuffling at the nose, coughing, apparent pain in the ear, as indicated by pulling at the ear or crying when it was touched, practically without exception, preceded the onset of the diarrhoea by as much as 24-48 hours. In 2 instances the diarrhoea was the first symptom of an abnormality. Both these infants on aural examination, had bilateral otitis media, though there was no indication of an aural infection until examination of the ears was carried out.

The case associated with pyuria had mild dysuria. Confirmation of a urinary infection was obtained on microscopical examination of the urine when pus cells and debris were found. The presence of septic spots on the body was the only ascertained cause of the diarrhoea in the single instance where such were present.

The diarrhoea was never severe in any of these cases in this group. The stools were rather loose, green and undigested. They were not so offensive as in/
in the instances of enteral infection or where the cause was dietetic. Vomiting was most noticeable in those cases associated with a frank aural infection. Dehydration was not a feature of the cases encountered in this group. In only one or two instances did dehydration much concern, and it responded to measures taken to combat it, viz. free fluid administration.

**Treatment.** The starvation regime was employed in all but 8 cases. In these 8 cases, the apple diet was used, the raw fruit pulp in 6 instances and Aplona in 2 instances.

All cases received treatment appropriate to the causal infection, in addition to dietetic measures.

In contradistinction to the other forms of diarrhoea in infants, the orthodox starvation method proved decidedly superior to the apple treatment. Only in the rapidity in abolishing the offensive odour of the stools was the apple diet more beneficial. The reduction in frequency of the stools in some instances was scarcely noticeable with the apple diet or Aplona. The starvation regime, however, was very successful in this respect, even though the local focus of infection was slow to respond to treatment.

The number treated with the apple diet was very small, of course, and a very guarded opinion can only be given concerning its failure. However, in comparison with the good results obtained in the other two types of/
of diarrhoea, it was decidedly disappointing in this group.

**Parenteral Infection - in infants on a Mixed diet.**

There were 70 children in this group. All were on a mixed diet.

**Age Incidence.** Average age: 14½ months. Age extremes: 7 months - 23 months.

**Sex Incidence.** Males. 36. Females. 34.

**Aetiological Factors.** Similar aetiological factors obtained in this group as were observed in the preceding group of parenteral infections. Rhinitis and rhino-pharyngitis again accounted for a large proportion of the cases. The other parenteral infections which were deemed to be the causal factors of the diarrhoea may be cited in order of frequency as follows: - bronchitis, tonsillitis, stomatitis, pneumonia, rickets and whooping cough.

**History.** In most instances in which an upper respiratory infection was the cause of the diarrhoea, there was a history of contact with an adult who was, or had been, affected by a similar infection.

The diarrhoea, as has been seen before, was not the primary symptom as a rule, in those cases. There was usually a history of nasal irritation or discharge for some days prior to the occurrence of the diarrhoea. Similarly, in the instances in which stomatitis was the primary cause of the diarrhoea, the sore mouth was present for some time ere the intestinal upset became manifest. The diarrhoea appeared in the course of the disease in the 2 cases of pneumonia.
In none of the cases in this group was the diarrhoea very severe. The stools were green, undigested, rather loose, but less so than in the similar group of younger children. There was slight dehydration in 9 instances, the worst degree being manifest in the two cases of rickets included in this group.

Treatment The starvation method of treatment was employed in 56 cases, and the raw apple pulp in 14 cases. The initial dose of grey powder or castor oil was given to those on the orthodox regime, but was not administered to the cases treated with the apple pulp. The starvation period lasted not longer than 24 hours in any of the cases. As much water as the child would take was given during the period of 'starvation'. In some instances, 5% glucose was added to the water. After this preliminary period of rest to the alimentary tract, a return was made to cow's milk feeding in a modified form, and as progress continued favourably, gradual additions to the milk, until the child was finally on a proper mixed diet suitable to its age. Local treatment to the focus of infection was carried out whenever and wherever possible. The two cases of rickets did not receive cod liver oil during the period of intestinal upset, but received instead short exposures to the mercury vapour lamp. This had a very beneficial effect, especially on the general health of the child. These exposures were continued for a period of/
of 3 months in each case, and after the intestinal tract had recovered from its upset, the benefit derived from the lamp was supplemented by the administration of cod liver oil. The results in the cases treated by the starvation method were very gratifying. There was a rapid abatement of the toxaemia and of the diarrhoea in the great majority of the cases treated by this regime. Those cases which were rather slow in recovering were due to rather troublesome upper respiratory infections, which responded but slowly to treatment directed towards their relief.

This satisfactory state of affairs could not be said to exist when the raw fruit diet was employed. There was a rapid reduction of the offensive odour of the stools with this treatment, but the diarrhoea itself was not specially influenced. It often continued unabated, in spite of vigorous treatment of the associated infections. The diet was given in the same way as was employed in the other cases.

Taking this group and the preceding one, there was little doubt that the starvation method was the treatment of choice as far as the alimentary tract was concerned. The cases submitted to the raw apple diet responded disappointingly in contrast to its good effect in the few cases of enteral and dietetic diarrhoeas in which it was used. Combining the parenteral infection cases in artificially fed infants and in those on a mixed/
mixed diet, 81 cases were submitted to the common starvation regime, and 22 to the raw fruit treatment. The latter cases constituted a very small group in comparison to the former, but the infections causing the diarrhoea were similar to those which caused that symptom in the children who were treated in the orthodox manner. Consequently, it is suggested that the starvation method of treatment is the better and more satisfactory of the two adopted in such types of diarrhoea in infants.

The incidence of the various parenteral infections as they occurred in this series of cases, is analysed in full in the discussion.
SECTION VI.

DISCUSSION.
SECTION VI.

DISCUSSION.

It has been seen when discussing the aetiology of infantile diarrhoea, that no one cause has been found, which will explain every case. The various theories on the causation of diarrhoea in infants, have been reviewed in detail in another section. The cases of the present series have been found to fall into three well defined groups.

Let us now examine these cases in more detail. For the sake of clarity, the following table shows the distribution into the three groups.

**Table 5.**

**Distribution of Cases.**

<table>
<thead>
<tr>
<th>Type of Case</th>
<th>Number (N)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infective cases</td>
<td>25</td>
<td>10.0%</td>
</tr>
<tr>
<td>Dietetic cases</td>
<td>82</td>
<td>33.0%</td>
</tr>
<tr>
<td>Parenteral cases</td>
<td>143</td>
<td>57.0%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>250</strong></td>
<td><strong>100.0%</strong></td>
</tr>
</tbody>
</table>

It will be seen that in 25 cases, the assigned cause of the diarrhoea was a bacterial infection in the bowel. In only 6 of these cases was a specific pathogenic organism isolated. In 5 instances, the B. Dysenteriae Sonne was isolated, and in the remaining 1 instance, B. Dysenteriae Flexner was the causative organism.
organism. In addition to these specific infections, 1 case was also treated in an isolation hospital, for a non-specific infection, considered by those concerned in the care of the child in hospital, to be a dysenteric infection of unidentified type. In most of the remaining 18 cases, one or more bacteriological examinations revealed no pathogenic organisms, but usually profuse growth of B.Coli was obtained. Unfortunately, the species of B.Coli was not specified in the reports.

If the intestinal flora in these cases were normal, could the symptoms have been due to any of the other recognised causes of diarrhoea? The infants were all examined thoroughly, when they were brought for consultation. A careful history of the feeding was taken. Particular enquiry was made as to the occurrence, recent or present, of an upper respiratory infection, in other members of the household. In none of these cases was such admitted. There was also no evidence of any infection in nose, throat, ears, chest or urine in any of these 18 children, at the first examination. All of the children were re-examined, two or at most, three days later. In not a few, daily examination was possible. At the second and subsequent examinations, no signs of any parenteral infections were ever present. Dietetic faults of such/
such degree as to merit their being regarded as the cause of the diarrhoea, could not be said to have existed.

The suddenness of the onset, with diarrhoea the urgent and presenting symptom, frequent loose and offensive motions, the refusal to feed and the frequent occurrence of vomiting along with the diarrhoea, were all suggestive of an enteral infection. Occasionally, traces of blood were present in the motions. Invariably, the temperature was raised considerably, and the child looked ill and toxic.

In view, then, of the negative findings in respect of a parenteral infection, and the absence, save in a very few, of gross dietetic errors the diagnosis of an enteral infection, non-specific in type, seemed justified. Naturally, since daily examination of the infants was, in most instances, impossible, an undiscovered focus of infection might have existed throughout the duration of the illness. It was observed, however, that some of the cases, treated by the apple diet, responded satisfactorily and rapidly, whereas in those cases of well-marked parenteral infections, similarly treated, the response was much less marked. Consequently, the 18 cases were classified as belonging to the Enteral group.

None of the cases were breast fed. All were fed on/
on cow's milk and water mixtures, or on condensed milk (e.g. Nestle), or on a light diet. The seasonal incidence was fairly evenly distributed throughout the year. There was no noticeable increase in the warmer summer months. The group is, however, so small, that no weight can be attached to this finding.

The cases which were due to dietetic causes were much easier of classification. The history given by the mother was often sufficient to suggest the diagnosis at once. Large, bulky feeds, usually also, too strong, the cause of unboiled milk, excessive zeal in the administration of malt extracts and cod liver oil, were the commonest causes of the intestinal upset. The use of excessive quantities of sugar was not, contrary to what might be expected, a feature of many of these artificial feeds. In fact, the mothers tended to use rather too small amounts of carbohydrate. In the warmer months the too frequent feeding of the breast fed children seemed to be a frequent cause of the diarrhoea occurring in these cases. Rich cream mixtures appeared to be the cause in many of the artificially fed children. This was particularly noticeable in the warmer months. In fact, by reducing the fat intake of the infants in the summer months by skimming the cow's milk as a routine, impressed me as having a beneficial effect on the infants/
infants. Ice cream was a not infrequent cause of diarrhoea in the older children. In this case it seemed to be not so much a case of excessive quantities of ice cream, but rather its indulgence at the wrong time.

In the cases due to the indiscreet consumption of ice cream, the onset was sudden, often occurring a few hours after taking the sweet. The children presented the symptoms of an acute diarrhoea, with loose offensive motions. Vomiting was often troublesome. Pyrexia and the early signs of dehydration, with loss of normal firmness of the fontanelle, and evident loss of subcutaneous fluid were usually present. Several of this form of dietetic diarrhoea showed also, evidence of upper respiratory infection.

Several of the dietetic cases had specimens of stool examined for any infection, but all those examined were negative.

As to the seasonal incidence, there was an appreciable increase in this type of case in the warmer months, though the cases occurred throughout the whole year, generally.

Some 11 cases of dietetic diarrhoea were breast fed, and 2 were breast fed with a complementary feed of cow's milk and water and carbohydrate.

The causes of the parenteral cases may be classified as/
as follows:

Table 6.

Causes of the Parenteral Cases

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colded, considered as a nasopharyngitis</td>
<td>68</td>
</tr>
<tr>
<td>Bronchitis and Tracheitis</td>
<td>23</td>
</tr>
<tr>
<td>Otitis Media, including otorrheas</td>
<td>22</td>
</tr>
<tr>
<td>Tonsillitis</td>
<td>15</td>
</tr>
<tr>
<td>Stomatitis and Gingivitis</td>
<td>5</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>2</td>
</tr>
<tr>
<td>Whooping Cough</td>
<td>2</td>
</tr>
<tr>
<td>Impetigo and Septic Spots</td>
<td>2</td>
</tr>
<tr>
<td>Urinary infection</td>
<td>1</td>
</tr>
<tr>
<td>Vaccination</td>
<td>2</td>
</tr>
<tr>
<td>Rickets</td>
<td></td>
</tr>
</tbody>
</table>

143 cases

It will be seen from the above table, that nasopharyngitis was the commonest cause of the diarrhoea. It accounted for 68 cases. The nasal irritation and discharge were frequently present before the onset of the diarrhoea. The latter was gradual in onset, and dehydration was not a feature of the cases.

The relatively low incidence of otitis media in this group of 143 cases, was an interesting finding. It was not in agreement with the findings of others (Smellie, 1939, Marriott, 1935, Baber, 1939 and others). Smellie found that otitis media was the cause in 125 cases out of 231 cases of parenteral diarrhoea, and Baber in her paper, found otitis media to be the cause of the diarrhoea in 111 cases out of 189 cases of diarrhoea/
diarrhoea due to parenteral causes. Ebbs (1937) quoted post mortem figures in his important paper, and found a high incidence of otitis media though there was little evidence of aural infection prior to death, in many of the cases he examined. The question, therefore, arises, was otitis media present more frequently than was diagnosed in this group under review? The answer is; I think, in the affirmative, and for the following reasons. The cases were not, as a rule, examined daily. As a result of this, several instances of otitis media might have occurred and resolution have begun ere the next examination, especially if the second examination took place at a rather prolonged interval after the first, as happened in a very few cases. The difficulties in diagnosing otitis media previously commented upon, were also present. Only the experienced otologist could be expected to detect slight changes in the ear drum. Again, an otitis might be present with a normal drum appearance. The same difficulties as beset others, also occurred in several instances in this Series, viz.—an apparently normal drum on one day, followed the next day by a profuse otorrhoea. This indicates an infective process going on behind an apparently normal drum—or apparently normal one to those not experienced in the practice of otology. The absence of aural symptoms/
symptoms is no indication, that the ear is healthy. However, it may be said here, that all cases, from whatever cause, were examined aurally at each visit. Lastly, exploratory paracentesis of the tympanic membrane in a doubtful case, as has been advocated, was not undertaken.

Bronchitis and Tracheitis accounted for some 23 cases of parenteral diarrhoea. In 2 cases Whooping Cough was associated with diarrhoea. Tonsillitis was the assigned cause in 15 cases, and Stomatitis and Gingivitis accounted for 5 cases. In only 1 case was a urinary infection present. This was a mild infection, and was treated at home on the starvation regime and administration of alkalis. The result of such therapy was very satisfactory.

In 20 cases of parenteral infection, associated with diarrhoea the child was entirely breast fed. Though the resistance of such children to infection is much greater than that of the artificially fed infant, the close contact of the former with the mother, suffering from a 'cold in the head' or sore throat, must be a potent factor in determining the occurrence of such an infection in the infant. Macgregor (1939) showed that this close contact of the mother and infant was a frequent source of the development of pneumonia in the latter.

Concerning/
Concerning the seasonal incidence of these parenteral cases, there was a decided increase in this type of case in the cold, wet months. It was also noted to increase in frequency when the weather was very dry, and especially when the dry weather was accompanied by warmth, unusual for the season of the year. In the series under review, the peak of incidence was found to be in the month of January for both years when the cases were investigated.

Taking all the cases, enteral, dietetic and parenteral, the following table illustrates the age incidence:

**Table 7.**

<table>
<thead>
<tr>
<th>Age group</th>
<th>Number</th>
<th>Per cent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 3 months of age</td>
<td>23</td>
<td>9.0</td>
</tr>
<tr>
<td>3 months and under 6 months</td>
<td>58</td>
<td>23.0</td>
</tr>
<tr>
<td>6 months and under 9 months</td>
<td>51</td>
<td>13.0</td>
</tr>
<tr>
<td>9 months and under 12 months</td>
<td>43</td>
<td>17.0</td>
</tr>
<tr>
<td>12 months and under 18 months</td>
<td>52</td>
<td>21.0</td>
</tr>
<tr>
<td>18 months and under 24 months</td>
<td>43</td>
<td>17.0</td>
</tr>
<tr>
<td></td>
<td>250</td>
<td>100.0</td>
</tr>
</tbody>
</table>

It will be seen that 32.0% of the cases occurred in infants under 6 months, and that 32.0% occurred in infants under 12 months. This finding is in keeping with most authors.
Out of these 250 cases of infantile diarrhoea, seen at the various clinics throughout Edinburgh, there was only 1 death, occurring in an infant suffering from a severe attack of Sonne dysentery, its twin brother recovering from a similar infection. This very low mortality may give a somewhat rosy complexion to mortality rates from diarrhoea in infants under 2 years of age, but it must be remembered that those cases were all seen, for the most part, in the early stages of the disease, and therefore, prompt treatment could be immediately instituted. Hospital statistics, would give a much higher mortality rate, for many reasons. Two only of these reasons need be given. The first, and probably the most important of these reasons, is that a hospital receives the most serious cases, which are associated with a high mortality. The second reason is that a hospital is entirely concerned with the ill child whereas the clinics deal with the healthy child, or the child who is showing the very earliest symptoms of ill health.

The explanation of the problem why one child with a parenteral infection develops diarrhoea, whereas another afflicted by a similar infection does not develop diarrhoea, and may even be constipated, is well nigh impossible. It may be that the reaction of the individual child to the same organism or toxaemia differs.
differs. Constitutional factors undoubtedly play a part. Whatever may be the explanation, the concrete fact remains, that a parenteral infection can and does produce diarrhoea in many infants.

From this group of 250 cases of infantile diarrhoea, 55 were selected as being suitable for treatment by the raw apple diet. The selection was made, in order that the value of this form of treatment could be more easily assessed. Only those cases were chosen whose mothers displayed such a degree of intelligence that we might be certain that the regime would be carried out as prescribed. There is the very real difficulty, when treating such cases at home, of convincing the mother that an article of diet, so often the cause of an intestinal upset, can, when administered in a different form, alleviate and cure the alimentary disturbance. Although the cases were selected on these grounds, they were irregularly distributed throughout the series. All three types of case were represented in this group of 55, save the dysenteric ones. Consequently, the group may be said to be fairly representative of the type of case met with in clinic practice.

The raw apple diet in the treatment of infantile diarrhoea, was only introduced into orthodox therapeutics a decade ago. Heisler (1929 & 1930) and Moro (1929)/
(1929) were the original pioneers in this form of treat-
ment. Moro originally established this raw fruit diet as a method of treating dysentery. His results were so encouraging, that he later used it in all the acute and chronic bowel infections of infancy. He did not, however, as has been pointed out before, advocate the free administration of fluid during the period of the diet. He only gave sufficient to relieve thirst.

Birnberg (1933) also commented favourably on this raw fruit diet in infantile diarrhoea. Earnshaw (1934) recorded his experience with this raw apple diet, in some 50 cases of dysentery in children, culled from his private and hospital practices. He concluded that the diet was one of the greatest advances made in the therapy of dysentery. All Earnshaw's cases showed a normal appearance of the stools within 48 hours of the institution of treatment. He advocated the free administration of fluid during the diet, and claimed better results from so doing, in comparison to when he restricted the fluid intake as advocated by Moro.

Earnshaw also experimented with ripe banana pulp, as a substitute for the apple, when the child tired of the latter. Again he claimed favourable results. More recently, Socola (1938) recorded similar results with banana pulp, and also banana powder, in a series of 56 cases of acute diarrhoeal disorder in infants.
my own experience, the incorporation of banana pulp in the apple pulp is a very useful adjunct in those cases where the infant is beginning to refuse the plain apple. In such instances, the addition of a little banana pulp results in an improved appetite.

Holst (1934) was favourably impressed with the improvement shown by the cases of diarrhoea in children, treated by the raw apple diet. Ellis (1933) commented that the apple diet was more adapted for use in the older child, viz. the child over 1 year of age. With this view, I am not in full agreement. I agree that the most rapid results are achieved by this diet in the older child, but the younger child does take the diet well and appears to benefit therefrom. Sheldon (1938) considered that the child of 6 months or more was the ideal subject for the diet. I found, however, that even the very young tolerate the diet well.

Mignot (1932), Freud (1934), Giblin and Lischner (1935), Barondes (1937), Stephen (1937), Grant (1937), Reuss (1938) and Sheldon and Hall (1939) have all commented favourably on the uses of the raw apple diet in diarrhoea in infants. Freud used a powdered apple preparation in the treatment of 9 acute cases, and 2 chronic cases of diarrhoea in infants and children. He claimed good results in this small series. Barondes described a typical 'apple stool'. This stool/
stool he describes as being reddish-brown, with little water content, and acid in reaction. This typical stool, he states, appears some 11-18 hours after institution of the diet. Sheldon and Hall consider the diet as being rather a useful adjunct in treatment, but not as a specific form of treatment. They state that the child with persistent diarrhoea is the best type of case for the application of the diet.

All observers, however, are not agreed as to the efficacy of the diet, and some (e.g. Smellie 1939, and Ward 1939) consider that the diet produces no more beneficial effects than the more usual methods of treatment.

The type of case which is submitted to the apple regime, is an important factor. Obviously, the severely toxic and dehydrated infant is an unsuitable subject. Those cases which display intractable or persistent vomiting, are also unsuitable for the diet. The child suffering from the parenteral form of diarrhoea, is likely to respond to the diet less successfully, than the child with an enteral or dietetic diarrhoea. The reasons for this will be discussed later.

In the 55 cases of the present series which were treated by the raw apple diet, 5 were of the enteral type, 26 were dietetic, and the remaining 24 belonged to the parenteral group. The enteral and dietetic cases/
cases generally showed a much more rapid response to treatment than did the parenteral ones. The motions in both these classes of case, were all odourless, more homogeneous and formed, as well as being less frequent, in 48 hours from the onset of treatment by the apple. The parenteral cases did not show, on the other hand, such a rapid improvement. This was particularly noticeable in the frequency of the motions. Indeed, in some of the parenteral cases, there seemed to be no great reduction in frequency. In all three types of case, however, there was a rapid loss of offensive odour, in some instances this was manifest within a few hours of the institution of the raw fruit diet. The probable explanation of the failure of the apple diet to reduce the frequency of the motions in the parenteral cases is that a simultaneous improvement must be brought about in the focus of infection. When such is produced, then the apple did seem to hasten the recovery from the diarrhoea. It is generally agreed that the action of the apple is a purely local one on the alimentary mucosa. The same argument of course applies to the cases treated by the starvation regime, and as one would expect, the same necessity of treating the focus of infection is present. The benefit to be derived from administration of the apple in all cases, as compared with the starvation regime, was that the diet/
diet could be more rapidly increased after cessation of
the apple. In the starvation regime, the increases in
the diet had to be made much more gradually.

The exact mode of action of the apple is obscure.
All are agreed, however, that the action is a purely
local one on the alimentary tract. Heisler and
Baumann believed that the fruit juices were the active
principles, which produced the beneficial effects. In
their opinion, these juices acted either by direct
action on pathogenic organisms, or, by promoting
changes in the Hydrogen-ion concentration of the bowel
contents, transformed the life conditions of the
disease-producing germs in the bowel to such an extent,
that their further development was impossible. Baumann
also believed that, associated with this change, there
was a change in the Calcium-Phosphate mechanism in the
alimentary canal. He suggested, that more calcium was
made available for local use, and that this increased
calcium exerted a soothing effect on the intestinal
mucosa.

Moro considered that the tannic acid contained in
the apple was responsible for the beneficial effects of
the diet. He believed that the acid, by virtue of its
astringent action, formed a thin protective coating
over the inflamed mucosa, thus protecting it from
further damage by bacterial or chemical action.
Barondes/
Barondes also suggested that the tannic acid present in the apple acted as a sedative. This supposed action of the tannic acid is therefore analogous to the tannic acid treatment of burns, the skin formed by the acid permitting of satisfactory healing beneath it. Against this theory that tannic acid is the active principle is that fact that when the acid is administered by mouth, it produces no benefit in acute diarrhoea. Consequently this theory is contradictory to what actually happens when the acid is given therapeutically itself.

The more modern theories are based on the researches of Malyoth (1934). This worker first described a substance called Pectin, and drew attention to its importance. Pectin is a cellulose derivative, is present in the structure of numerous plants, and is found in combination with calcium in pulpy fruits, especially apples. It is present in apples in varying amounts, depending on the type and degree of maturity of the fruit. Tompkins (1935), in a review of the opinions on the action of apple diets, concluded that pectin should be regarded as the active principle. He believed that pectin acted by virtue of its buffer action, its absorptive capacities and its ability to fix amines.

Manville, Bradway and McMinis (1936) spoke of pectin/
pectin as a hydrophilic colloid. The basic part of the molecule is formed, at least in part by the pentose, arabinose, together with an acid, galacturonic acid. These workers classed pectin as a carbohydrate with but little calorific value, but contributing to the bulk of the intestinal contents, by virtue of its power to imbibe water. Freud (1937) agreed with this theory.

This action of pectin explains the observation that when it is given to normal children, it actually increases the frequency of the bowels.

Pure pectin is moderately soluble in water, but its solubility is increased when it is combined with certain metals. In the bowel, the pectin molecule is believed to be disintegrated, with the liberation of the galacturonic acid. This acid is thought to act as a detoxifying substance through its ability to form conjugation products with toxic materials. The remainder of the molecule imbibes water, thereby increasing the intestinal contents, and so mechanically assisting in the removal of toxic material.

Winters and Tompkins (1936) tried pure pectin as a substitute for apple pulp. They had been unable to satisfy themselves that all apples contained sufficient quantity of pectin, to produce therapeutic effects. A further difficulty that these investigators had encountered/
encountered was that of obtaining ripe apples always. They combined pectin with agar to provide sufficient cellulose, and added a carbohydrate to provide caloric value. This preparation was given to a group of infants with diarrhoea, and the results compared with a similar group receiving raw apple pulp. A more satisfactory result was obtained in the former group. Washburn (1938) also recorded favourable results with a similar pectin-agar-carbohydrate mixture in 70 cases. Since then, it has been shown that pectin alone, without the addition of bulky substances such as agar, and without any added carbohydrate, gave a satisfactory response in cases of infantile diarrhoea.

Very recently, a rather disquieting report on the effects of pure pectin has been published by Block, Tarnowski and Green (1939). These investigators failed to obtain favourable results in a series of cases of dysentery in a state institution, when they used a pure pectin solution along with a smooth, high calorific value diet, and parenterally administered fluids. In 11 acutely ill cases, they found pure pectin was incapable of altering the course of the disease. The toxic and diarrhoeal symptoms continued unabated.

Is pectin, then, a satisfactory anti-infective and anti/
anti-diarrhoeal substance? Haynes, Tompkins, Washburn and Winters (1937) carried out experiments to determine the bactericidal powers of pectin. The organism employed in these experiments was B. Coli. They found that the pectin they used had a very definite bactericidal action, producing effects within 48 hours. These workers pointed out that the most marked bactericidal effect was present when the pH of the pectin-broth mixture was between 5.0 and 5.5. The same workers (1938) pointed out in a later paper, that the pectin they had used in their previous experiments had been contaminated with nickel. Later research, after this discovery, were carried out with pectins derived from several sources. These experiments revealed the interesting disclosure that only those pectins which were combined with nickel had any bactericidal power. Numerous organisms were used in these latter experiments, Staphylococcus Aureus, Beta Haemolytic Streptococci, B. Dysenteriae Flexner, etc. Nickel pectinate had decided bactericidal effects on all of these organisms. Pure pectin had but little effect, in the control experiments. Arnold (1939) has actually found that pectin may promote bacterial growth.

Block, Tarnowski and Green (1939) tried this nickel pectinate in those cases which did not respond to pure pectin/
pectin, as described supra. Almost without exception, the cases immediately responded favourably. The temperature fell, the diarrhoea lessened, and the toxaemia abated under this treatment. The nickel pectinate was well borne, and there were no toxic effects. Arnold (1939) influenced by the possibilities for therapeutic use of nickel pectinate, investigated its toxicity and fate in the rat. He found that the rats fed on this substance, continued to put on weight, in spite of relatively large doses. Autopsy on those animals revealed no gross pathology. Drinker et al (1924) had already published a review of the literature on the biological behaviour of nickel. They had produced evidence that 25 mg. of nickelous oxide per kilogramme of body weight is the lethal dose for rats. Arnold in his experiments had actually administered as much as 1256.0 mg. per kilogramme body weight, of nickel pectinate during a period of 8 weeks. Experiments have been carried out recently by other workers in this field of investigation, and it has been found that relatively large doses of nickel-pectinate can be borne without ill effect. Excretion of nickel compounds takes place through both the urinary and intestinal tracts. If an inorganic salt of nickel be administered to rats, e.g. nickel sulphate, some 23% is excreted by the kidneys and 65% by the alimentary tract. /
tract. If nickel pectinate be administered, only some 14% is excreted by the kidneys, but 73% is excreted by the alimentary tract. This tends to show that nickel pectinate is not readily absorbed, and is further evidence in favour of a local intestinal action of the compound. Toxic effects are more readily produced if inorganic nickel compounds are used. These toxic effects are mainly on growth, together with an action on the appetite, both being depressed.

The therapeutic value of pure pectin is still obscure. Reports on its effects are, as we have seen, conflicting. Certainly nickel-pectinate seems to have a very decided anti-bacterial and anti-diarrhoeal effect. The value of the pectin in the raw apple pulp is, however, open to question. Until we know the exact chemical constitution of pectin, whether or not it exists in an active and an inactive form, and whether all species of apple contain the same type of pectin, and in similar quantities, it will be impossible to state its mode of action with any degree of certainty. A further point which needs clarifying, is the question of the pectin content of the same species of apple at varying stages of maturity. If such is not the case, then it may be the reason why some authors claim good results from the diet, while others remain unimpressed (Wolff, 1932). It/
It would seem that pectin is most effective when in a more soluble form, e.g. nickel pectinate, than when it occurs in its natural form. If such is the case, it would be interesting to know how another metal, e.g. sodium or potassium, combined with pectin would act. No work has been done in this connection as yet. It seems reasonable to assume that such a combination actually takes place, then it is not so effective as the nickel pectinate. It may be that the beneficial effects of the raw fruit are due entirely to its mechanical properties, the cellulose cleansing the gut and adsorbing the organisms and/or their toxins.

An important reason for the inconsistency of the published results on the apple diet lies in the age of the patient submitted to the treatment. Undoubtedly the older child reacts better to the apple diet than does the younger one, i.e. the curative results are better in proportion to increasing age (Frank, 1937). I was able to confirm this to a certain extent in this series.

The type of case submitted to the apple regime is also important. As we have seen, the action of pectin and probably of the raw apple pulp also, is a purely local one. We would therefore expect that local intestinal conditions such as infection or dietetic upsets/
upsets would respond more satisfactorily to the diet than would a parenteral infection. In the latter instance, the diarrhoea is merely another manifestation of the clinical picture appropriate to the focus of infection. This small series of cases seems to bear this suggestion out. The best results were seen in the cases due to non-specific infection, and to dietetic causes. The results obtained in the parenteral cases were less satisfactory. The rapid reduction in the frequency of the stools, so noticeable a feature of the first two classes of case, was much less marked in the parenteral cases. In fact, in not a few of the latter group, there was little or no reduction in the number of the stools. In all three types of case, however, the motions quickly lost their offensive odour.

Six cases were treated with a powdered apple preparation, Aplona. The results were no better than those which were obtained when the raw fruit pulp was used. The only benefit seemingly to be derived from the use of this preparation, was its reputedly high calorific value. It had been stated that loss of weight occurred in most of the cases treated, both by the raw fruit pulp and by the Aplona. It was never sufficient to cause any concern, however.

The impression was formed that the continuation of the/
the apple pulp in two feeds per diem, for the week following the intensive course of three days apple alone, enabled the feeds to be increased more rapidly in strength, than obtained in those cases treated by the starvation regime. The rapid return of the diet in several of the enteral and dietetic cases enabled one to begin the milk feeds earlier than the fourth day of treatment, as is the more usual procedure.

The treatment of the dysenteric group by the apple diet was not undertaken. The cases were all treated in an isolation hospital. It was observed in a previous assay in this form of treatment in dysenteric cases that the tenesmus, so often present in such cases, was frequently not relieved by the apple alone. The addition of a saline, such as sod. sulph. or mag. sulph, given at one hourly intervals for three doses every morning, along with the apple diet, brought about marked relief of this distressing symptom. It seemed, also, that the children thus treated were able to resume a normal diet more rapidly than when a modified milk diet was given from the commencement of treatment.

The remaining 189 cases were treated by the more orthodox starvation method, with local treatment to the parenteral focus when such existed. The results of such therapy were also very favourable, and particularly so in the parenteral cases. In the enteral and dietetic/
dietetic instances, the results compared unfavourably with similar cases treated by the apple diet. Progress was often much slower, the diarrhoea tended to persist longer, and recurrences of the diarrhoea occasionally occurred. A return of the offensive odour in the motions was also noted, appearing shortly after the modified diet was resumed. The conclusion was reached that the starvation regime had its greatest value in the treatment of the parenteral type of diarrhoea, and that the apple diet was the treatment, par excellence, for the enteral and dietetic types. According to Sheldon and Hall (1939) the best results of the apple diet are to be obtained in the child who has suffered from a persistent diarrhoea associated with the passage of frequent loose, offensive motions. The apple diet is highly thought of in dysenteric cases by Moro, Earnshaw, Kissling and others.
SECTION VII.

SUMMARY AND CONCLUSIONS.
Summary and Conclusions.

A brief historical survey of Infantile Diarrhoea has been given. The high mortality from this disease in the part has been shown to have fallen considerably in recent times. Reasons for this progressive fall have been discussed. A table, also reproduced graphically, has been shown to demonstrate the fall in mortality from Infantile Diarrhoea, in children under two years of age, for Scotland and Edinburgh 1871-1937 inclusive.

Prophylaxis of the disease has been discussed. Better education of the doctors, nurses, mothers and mothers-to-be, has been urged.

The aetiology of Infantile Diarrhoea has been considered in detail. No single factor has been found to account for every case. All cases fall into three groups:—

(1) Enteral. Due to infection in the bowel.
(2) Dietetic. Due to dietetic errors.
(3) Parenteral. Due to infections outwith the bowel.

All the cases under consideration in this series have been found to fall into one or other of these three groups. The parenteral group has been found to be the largest. The commonest parenteral infections causing diarrhoea have been found to be upper respiratory infections, with or without otitis media.
bottle-fed infant is more liable to develop diarrhoea from any cause than is the breast-fed child. The commonest cause of diarrhoea in the breast-fed infant is upper respiratory infection, usually contracted from the mother. The Enteral group has been found to be the smallest, both in the breast-fed and artificially-fed infant. Cholera Infantum is now practically unknown.

The pathology and morbid anatomy of Infantile Diarrhoea have been discussed.

The symptoms and signs of all three groups of case have been described. The onset may be sudden and acute, or gradual and mild. The difficulties in the diagnosis of aural conditions have been stressed.

The treatment undertaken in the series under review, has been described in detail. The Raw Apple Diet was used in some 55 cases. It was not attempted in the breast-fed infants. Other methods of treatment have been outlined.

7 cases have been described, and 243 other cases have been described in classified form.

The results of the clinical investigation, and of the treatment of these 250 cases have been discussed. Theories on the mode of action of the Apple Diet have been reviewed.
CONCLUSIONS.

(1) Mild forms of Infantile Diarrhoea are still common.

(2) Breast feeding is the best prophylactic measure against diarrhoea.

(3) The parenteral form of diarrhoea is the commonest in children attending Infant Welfare Centres in Edinburgh.

(4) The treatment of diarrhoea by the Raw Apple Diet is an excellent line of therapy. The best results are obtained in the enteral and dietetic cases. All infants can be submitted to the regime.

(5) Parenteral cases respond less rapidly to the Apple regime, but increases in the diet of milk can be made more rapidly than when the infant is treated by the commoner starvation method.

(6) The treatment of the parenteral focus is essential.

I would like to express my sincere thanks to Dr T.Y. Finlay, Medical Officer, Child Welfare Department, City of Edinburgh, for allowing me all facilities for carrying out this investigation.
SECTION VIII.

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