# INFANTILE MARASMUS.

THESIS

SUBMITTED FOR THE M.D. EDINBURGH

BY

W. A. ROBERTSON, M.C.,

M.B., Ch.B. 1907. M.D. 1926

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# 1. INTRODUCTION.

Until recent years it was thought that, provided the human body, be it during the period of infancy or in the adult state, was supplied with the main constituents of food, namely proteins, fats, carbohydrates and inorganic salts in their correct proportions and in sufficient quantity, life would be maintained in a healthy state and growth would proceed normally, other factors being equal. During the last decade, however, our knowledge of the dietary requirements of the human organism has advanced rapidly and many beliefs previously held have been abandoned in the light of recent research. It is now generally accepted that the above mentioned foods are not in themselves sufficient to satisfy the needs of the animal for growth and maintenance of health and that there exist a number of other indispensable dietary components. These components, which have at different times and by different authors received a variety of names such as "food hormones", "exogenous hormones","growth determinants", "advitants" or more commonly "Vitamines" (Casimir Funk) but even better/

better termed "Accessory Factors of Diet", have recently been the subject of considerable attention, and the importance of these accessory factors in the question of the proper feeding of infants will be realised. The subject of Infantile Dystrophy generally known as Marasmus, Simple Atrophy or Wasting, has occupied the minds of the medical profession and the public for some considerable time, and, despite the great advances made in recent years in the training of young expectant mothers by means of Anti-natal Clinics and Infant Welfare Centres for the poorer classes in the larger towns, and the greater variety of scientifically prepared foods both for the mother and the child - for the former when she is able to feed the child on the breast and for the latter when breast-feeding is not possible and when for any reason cow's milk is not available/or advisable suitable, still the fact remains that there is an uncomfortably large per-centage of cases of Marasmus occurring which would appear to be preventible. Though a large number of so-called Marasmus cases are traceable to Organic disease such as Tuberculosis, Syphilis, Congenital Heart Disease or even Chronic Constipation, still a large proportion of cases is due to imperfect or improper nutrition. This latter may be divided into two main/

main categories:-

1. Faults on the part of the mother, such as, giving feeds too frequently, giving them too strong, or in too large quantities.
2. Faults on the part of the infant - one finds a certain group of cases where, in spite of the best feeding - even breast-feeding - the child goes rapidly down-hill. This condition often occurs in several members of the same family and may be ascribed to the infant showing an idiosyncrasy against one type of food or in certain severe cases even against two types which would soon prove fatal.

In the present thesis the writer proposes to give an account of the present day views of Infantile Marasmus considered as a deficiency disorder, to discuss the recent views with regard to the nature of Vitamines, and generally to endeavour to make some small contribution to our knowledge of Infantile Marasmus.

## 2. HISTORICAL OUTLINE.

Before commencing a brief account of the History of this Disease it would be well to state that the term Marasmus or Simple Atrophy (Athrepsia and Infantile Malnutrition are other names which have been used) is strictly applied to a condition of Wasting in Infants where there is no complication such as Tuberculosis or Syphilis and is usually ascribed to imperfect or improper nutrition as has already been stated.

Talbot has shown that there is no appreciable change in metabolism of Infants until there is a loss of 20% of the body weight; on this principle Parsons makes it a working rule that an infant is regarded as suffering from Simple Atrophy if the body weight is less than 80% of the expected body weight for age and if there is no other evidence of disease.

The term Marasmus was first introduced by Soranio, a physician of Aragon who, writing in 1600, states that the expression "Infantile Atrophy" was used in Greek Medicine.

In 1583 Hieronymus Mercurialis - an Italian Physician teaching in Padua and Bologna published "De Morbis Puerorum" in/

in which (according to Marfan) Infantile Atrophy is described under the term "Macies" and which the Greeks called "ἰσχνότης λεπτότης". The Earliest Reference by an English Author was in 1653 "A Treatise of the Diseases of Children with their Causes, Signs, Prognosticks and Cures for the benefit of such as do not understand the Latine tongue and very useful for all such as Householders and have Children" by Robert Pernell, Practitioner in Physick at Cranebrooke in Kent.

In 1659 "De Morbis Puerorum" by James Primrose contained a description of Infantile Atrophy under the heading "Macies" and from some statements comtained therein it would appear that the author was familiar with Pernell's book.

In another book on "Diseases of Children" - habited in English at Oundle in Northampton by J. S. and dedicated to Sir Robert Boyle, Marasmus is described, "In children it is caused for the most part by the defect of nourishment being either too little or too vitious and unuseful proceeding from the fault of the milk."

In 1689 Walter Harris'(who was Lumleian lecturer from 1710 to 1732) book "De Morbis Acutis Infantum" was the Standard work for a long time - the last edition being a part of Martin's "A Full View of all the Diseases of Children"/

Children" published in 1742. In this latter book Marasmus is not actually mentioned, but he describes a condition called Atrophia Vernimosa.

Dr. John Astruc, in his "Treatise on all the Diseases incident to Children" published in England in 1746, gives a good description of Infantile Atrophy.

About 50 years later Michael Underwood published his "Treatise on Diseases of Children 1784 - 1827". Like many others he confused Tubercular Peritonitis with Marasmus. When writing of "a confirmed hectic fever and marasmus, or a wasting of the whole body called by some writers Atrophia lactentium" he describes enlargement and suppuration of the mesenteric glands but he clearly recognises the condition thus: "There is indeed an atrophy or universal decay in infants for want of the breast, or from unsuitableness of it (which is the true Atrophia Lactentium) or of whatever else may be the child's ordinary food, but this is not usually attended with fever... Harris recounts some remarkable recoveries in what he calls the Atrophia Vernimosa (or worm hectic) but I have seen none so marvellous as in Atrophic Dententium or tooth Atrophy". In another passage he states that the "Macies" of ancient writers is"a true Atrophy or Marasmus".

In 1830 Dr. John Darwell, Physician to the General Hospital/

Hospital, Birmingham, wrote a small book "Plain Instructions for the Management of Infants with Practical Observations on the Disorders Incident to Children". In an article on Marasmus he says, "This term, which in its original significance, meant nothing more than emaciation, we now understand as a general expression for Infantile Fever. It is almost always the consequence of improper diet or regimen or is a sequela of the exanthematous diseases". He then proceeds to give P.M. findings which are those of Peritonitis and General Tuberculosis. This may be taken as representing the general opinion in this country until Dr. Charles West's lectures on Diseases of Infancy and Childhood in 1848. He clearly differentiates "Infant Atrophy as a Nutritional Disorder", although stating that it may occur weakened by previous disease or the subject of General Tuberculosis. He shows how insufficient and unsuitable food produces the condition. "Death takes place from starvation; its approach, indeed, having been slower but the suffering which preceeded it not therefore less than if all the food had been withheld".

Eustace Smith in his book "Wasting Diseases of Children" 1868, under the heading "Simple Atrophy" well describes the clinical picture, and obviously regards the condition as "a vice of nutrition". The same view was taken/

taken by Parrot in 1875.

The frequent occurrence of diarrhoea and vomiting in cases of wasting led to a great deal of attention to the alimentary tract, and attempts were made to classify them on a morbid anatomy basis. In 1879 Bonchut criticised Parrot for introducing "Athrepsia" which he said was Entero-colitis. Atrophy or Marasmus were little mentioned about this time. This morbid anatomy theory failed through the want of any constant lesions, and a number of the appearances found were due to post-mortem changes.

From 1885 onwards, when Escherlich of Vienna established the fact that the type of bacteria found in the Intestine depended on the kind of food given, most investigators held an intermediate view between Charles West's and Eustace Smith's i.e. that Atrophy was due to starvation and that there were morbid changes in the alimentary tract which were the cause of Atrophy. Some held both views. Eustace Smith maintained that it was not only due to insufficient food but also unsuitable food, and that the difficulty in digesting the proteid was the cause of atrophy in children fed on cow's milk. This doctrine of the harmfulness of protein in cow's milk originated by Bredert in 1869 was very popular and until recently the presence of curds in the stools was thought to be due to undigested proteids.

Czerny and Finkelstein have done much work since the beginning/

beginning of the present century. Czerny states that Atrophy is not a disease in itself, but a disturbance in growth and in general nutrition which is the result of nutritional disturbances or infective processes or both. As a result of these conditions infants do not invariably become atrophic - the reason why some become atrophic and others do not is a constitutional anomaly. Those who do become atrophic are either suffering from an exudative diathesis or are descended from neuropathic or psychopathic parents, and show an extreme nervous irritability of the intestinal tract, or are children with the hydriopic constitution, i.e., show a difference in Salt + Water retention from the normal child. Czerny's classification might well be included at this stage.

I. Disturbances from Food.
   a. Milchnahrschaden.
   b. Mehlnahrschaden.
   c. Infantile Scurvy.

II. Disturbances from Infective Processes.
   a. Acute Disturbances.
   b. Enteral Infections.
   c. Parenteral Infections.

III. Disturbances from Constitutional Anomalies.
   a. Exudative Diathesis.
   b. Rickets.
   c. Anaemia.
   d. Neuropathic Constitution.

IV. Disturbances produced by Congenital Malformation.

The two most important divisions to be considered are (1) Milchnahrschaden and (2) Mehlnahrschaden.

(1) The former is due to overfeeding with milk and the particular factor causing the injury is fat in the milk; it might therefore be called Fettnahrschaden. The clinical features of this condition are overfeeding with fat and failure to gain weight, constipation with pale, dry, crumbly soap stools, pallor and flabbiness. Czerny did not believe in cream in infant feeding.

(2) Mehlnahrschaden is due to feeding on a diet containing too much carbohydrate - frequently starch - such a diet being not infrequently deficient in fat and protein, a good example of such a diet being a weak sweetened condensed milk mixture. The chief symptoms of this condition are marked wasting, stools often frequent, usually acid, green and watery, with a variable amount of mucuous and small whitish curd masses. Occasionally the wasting is masked by oedema (this is the hypertrophic type.) According to Czerny, Protein never causes any upset. Curds nearly always consist of fat and very rarely contain proteid, and the factor in the production of atrophy is the difficulty of dealing with fat. He assumes that these children have a low assimilative power for fat and, as a result of fermentation and digestion, fatty acids are found in excess in the intestinal/

intestinal tract. To neutralise these fatty acids, alkalis are taken from the tissues and excreted into the intestine and lost in the stool. In constipation with soap stools, calcium, and in diarrhoea, potassium and sodium are thus excreted. Demineralisation of the body is the essential cause of atrophy. "On account of this mineral loss, it is assumed that the cell structure and tissue composition is altered, and that if sufficient loss takes place the normal cell function will be so disturbed that the building up of the new tissue is impossible even if the processes in the Intestinal Canal are restored to normal."

Czerny's work though greatly criticised was of the greatest importance for, for the first time, he emphasised the importance of the consideration of the infant as a whole, and not simply that of his food and his alimentary canal. He further demonstrated the harmlessness of protein.

Finkelstein published, in his book in 1912, a classification of nutritional disorders adopted to some extent by John Thomson and Dingwall Fordyce. He agreed with Czerny that fat was the main factor but he said that sugars and salts are also to blame. He based his views largely on Meyer's whey exchange experiments, etc. When the curd of cows' milk was mixed with whey of human milk, the mixture was well tolerated, whereas when the curd/

curd of human milk was mixed with whey of cows milk this mixture was badly tolerated. He explained this by virtue of the fact that the whey salts may injure the intestinal mucosa and interfere with its activity by decreasing the normal secretion, depressing the anti-bacterial functions of the cells and thus allowing the growth of bacteria which produces sugar fermentation. The injured mucous membrane allows the passage of harmful substances - lactose and salts - into the circulation and is itself liable to bacterial infection.

Finkelstein in pursuance of his view that the whey salts and sugar fermentation are factors in nutritional disturbances devised a special milk modification to combat this fermentation. This modification is the well known protein milk which has a high protein but a low sugar content.

It is, in his view, the milder form of sugar fermentation that is the cause of the commencement of diminished tolerance for carbohydrates. The children are fretful and there may be slight attacks of diarrhoea. Less fermentable carbohydrates, such as dextri-maltose, may be tolerated but in the worst cases Finklestein advised using his protein milk. If this condition does not improve then the infant passes into one of the two following categories, which may be influenced by the food given.

1. Alimentary Intoxication-if the food contains much sugar and little fat.
2. Alimentary Decomposition-if the food contains little sugar and more or less plentiful fat.

In the latter the weight chart shows a steady fall, the condition being that of Marasmus or Simple Atrophy; in the former the fall of the weight is rapid and is accompanied by considerable diarrhoea, an irregular temperature, respiratory and circulatory disturbances. In its worst form this condition becomes that of cholerine or cholera infantum and is due, not to a bacterial infection, but to the passage of whey salts through the unhealthy mucous membrane into the general circulation as previously explained.

Recent work has been carried out in America and Britain. Of the Americans L.E. Holt and his co-workers Marriott and Ultheim have contributed largely to the subject.

Marriott states that atrophy is essentially the result of partial starvation continued for long periods of time. There is a negative mineral balance and in fully developed cases of atrophy there may also be a negative Nitrogen balance pointing to a destruction of body tissue. "There is diminished functional capacity of the body to oxidise organic material, and the basal energy metabolism of athreptic infants is often high as compared with those that are normal. The destruction of body substance is not confined to the solid portions of the organism but

affects the blood as well. There is a diminution of blood protein, a destruction of blood corpuscles and a decrease in the total blood volume, resulting in a diminished blood flow which brings about a lowering of the functional capacity of all parts of the body. When the blood flow is increased there is a return of normal function." The well known clinical fact that atrophic infants may have a stationary weight for a long period, despite a fairly large intake of fluid is probably due to this being"a period of repair" during which the functional capacity of the body for utilising food is returning. It has been shown that during this period the blood volume is being restored.

L.E. Holt believes that Marasmus is due to failure of assimilation owing to imperfect digestion, improper food, unhygienic conditions, or feeble constitution. The theory that atrophy of the intestinal tubules explains Marasmus has little support. Marasmus is a common sequel of Vomiting and Diarrhoea. Some works describe a cirrhotic change in the deeper layers of the intestinal mucosa which would suggest deficient absorption as a cause, but these findings have not been confirmed and this appearance when found in an autopsy may be due to natural post-mortem changes.

It seems to be generally held that in Marasmus there is a deficient absorption of fat, though some maintain that the condition results from an abstraction of the salts from the tissues as a result of excessive soap formation. From Glasgow important contributions have been added by Fleming, Hutchison, Guy and Telfer. H.S. Hutchison concludes that:-

1. There is no evidence to show that in Infantile Marasmus a defective absorption of fat is a factor in its causation either through imperfect digestion, passage of unduly large motions or through a defect in the absorptive capacity of the bowel wall.
2. Saponification of fats does not lead to a loss of fat through defective absorption. A high percentage of insoluble soaps in the faeces fats simply upsets the soap fatty acid balance and since the insoluble soaps possess feeble hydrophélic properties constipation results.
3. The Iodine value of the faeces fat is lower than in health and this suggests that in Marasmus there may be a qualitative error in absorption rather than a quantitative one.

4. The Alkaline reserve of the blood in Marasmus is lower than in health, but the diminution is small and does not suggest an acidosis of any consequence. There is no evidence that there is a lower alkaline reserve on a whole milk diet than on a weak fat milk so that apparently in Marasmus there is not a incomplete metabolism of fat.

L.G. Parsons, prominent amongst English writers, makes the important statement that "There can be no doubt that Carbohydrate and Fat Intolerance or Indigestion do occur, and that such groups can be separated at times; but they are not a sufficient explanation of all the simple atrophies of infants", and he further states that a case whilst still atrophic may show Carbohydrate indigestion at one time and fat indigestion at another time or the same case may show a combination of both at one and the same time.

Parsons' Classification is as follows:- (He maintains that there are no degress of Atrophy, but retains the 20% rule)

1. Simple Atrophy.

2. Simple Atrophy with Dyspepsia
   (a) Diarrhoea and Vomiting
   (b) Fat Indigestion
   (c) Carbohydrate Indigestion
   (d) Protein Indigestion
   (e) Pylorospasm

3. Simple Atrophy from deprivation - Avitamosis
   (1) Absence of fat (dystrophia adipo-genetica) frequently also associated with excess of Carbohydrate (dystrophia amylogenetica) and often insufficient protein.
   (2) Absence of anti-scorbutic and anti-neuritic vitamines.

4. Atrophy associated with other diseases and infections. Pyloric stenosis, and other deformities, Pyelitis, Otitis Media, Syphilis, Tuberculosis, Broncho-pneumonia, Acute epidemic Diarrhoea, etc.

It would be well to give at this stage a short account of our present knowledge of Vitamines in order to fully understand Section 3 of Parsons' Classification.

Accessory Factors in Diet or Vitamines.

As briefly pointed out in my introductory remarks, it has now been firmly established that, besides the ordinary and necessary constituents of food sc. proteins, fats, carbohydrates and inorganic salts, certain unidentified principles known as Accessory Food Factors or Vitamines must also be present in the Diet in order to stimulate growth, maintain health and prevent the occurrence of deficiency diseases. These substances have not so far been isolated nor are their chemical or physical properties known, but their presence has been fully demonstrated by experiments with animals, and their absence in the diet of infants, which is of more importance as far as this work is concerned, is soon shown by the rapid appearance of Atrophy.

The Accessory food factors at present recognised are three in number

1. Water - soluble B. or Anti-neuritic or Anti-beri-beri factor.
2. Fat - soluble A. or Anti-rachitic factor.
3. Anti-scorbutic factor.

As far as is known the animal organism cannot manufacture the accessory food factors but is dependent on their supply directly or indirectly from plants, therefore a brief account of their distribution and properties is of interest.

1. Water soluble B. or Anti-Neuritic or Anti-beri-factor is necessary to promote natural growth in young animals. It is found in almost all natural foodstuffs to a more or less extent, but chiefly occurs in the seeds of plants and eggs of animals where it is deposited for the nutrition of the offspring. Highly cellular organs such as brain and liver are richly endowed with this Vitamine but flesh is poorly supplied. It occurs excessively in yeast cells also in yeast extracts e.g. "Marmite". In the pulses, it is contained throughout the seed, but in cereals, it is only found in the germ and in the peripheral layer which in the process of milling is peeled off ~~with~~ with the pericarp and forms the bran. In certain countries where cereals are the staple diet and where the seeds are

are milled such as polished rice, beri-beri- is of common occurrence. In this country though the bread (white wheat) is made of milled flour this disease is comparatively unknown because other foodstuffs are taken in sufficient quantities to supply this accessory factor.

2. Fat soluble A. or Anti-rachitic factor is also necessary to promote growth and at the same time prevents Rickets in young animals.

The main sources of this Vitamine are

1. Certain fats of animal origin especially occurring in cream, butter, beef fat, fish, oils such as cod liver oil and whale oil, and egg yolk. On the other hand it is almost entirely absent in vegetable oils such as linseed, olive, cotton seed, cocoanut and palm oil.

2. Green leaf vegetables contain this factor in large amounts but it is deficient in root vegetables.

3. Anti-scorbutic factor.

This Vitamine is not so much essentially connected with the growth of the animal as that it is a preventitive against Scurvy. It is found in fresh vegetables and to a lesser extent in fresh animal tissues. Its richest sources are such vegetables as cabbages, lettuces, turnips, watercress, and fruits such as lemons, oranges and tomatoes; it occurs in lesser amounts in potatoes, carrots beetroots, etc. Though lemons are particularly valuable as an anti-scorbutic food it is worthy of note that lime

juice is much less so.

The resistance to heat varies considerably in the three different Vitamines. Fat Soluble A. accessory substance is gradually destroyed at 100° C. and it has been shown that four hours' exposure to that temperature is sufficient to render butter fat of little greater nutritive value from the standpoint of Vitamine supply than an equivalent quantity of lard.

Water Soluble B. withstands desiccation for long periods of time as may be appreciated from the fact that its principal sources are found in dry foodstuffs. Its resistance to heat is considerable. A series of experiments carried out by Chick and Hume showed that destruction of this Vitamine takes place very slowly at 100° C. but much more rapidly at 120° C. This fact is important in as much as in the baking of bread and biscuits during which the interior of the substance does not rise above 100° C. no serious alteration in the composition of the factor takes place, whereas in the process of tinning foods temperatures much above 100° C. are sometimes required therefore the Vitamine element is entirely destroyed.

The Anti-scorbutic Vitamine is very sensitive to heat and easily destroyed, therefore all dry foodstuffs

such as cereals, pulses, dried vegetables and dried milk are almost totally deficient in this accessory substance .

## 3. ETIOLOGY.

In discussing the Etiology of the various conditions causing Infantile Dystrophy one has to consider in the first place what influence pre-natal factors may have in the production of this Condition. In certain cases there may be a history of ill health of the mother during the gestation period, e.g. anaemia, debility, bronchitis, Tuberculosis, Syphilis, even mental conditions; the father may also suffer from any of those conditions. Further there may be a history of other cases of Marasmus occurring in the same family, which points to the likelihood of some hereditary history underlying.

In those cases of Atrophy which are not directly due to Constitutional or Organic disease, such as Tuberculosis, Syphilis, Cardiac disease, etc., a large proportion is due to imperfect or improper nutrition, and, as briefly stated already, they may be grouped into two main divisions.

1. Faults on the part of the Mother - such as giving the feeds too frequently or irregularly, giving them too strong - either as a whole or one or more constituents being proportionately in excess of the others as the case may be,

even giving the feeds in too great a dilution or in too large a quantity, and

2. Faults on the part of the Infant.

In the first group, i.e. Faults on the part of the Mother - all those causes mentioned refer to Artificial feeding and will be referred to later, but there are cases wherein, as Robert Hutchison states, poverty of the mother's milk may cause wasting as a result of habitual constipation; but this is not the cause of wasting in every case, because when the bowels are regulated by an aperient the child begins to put on weight even if the form of feeding be not altered. Therefore there must be other causes for this wasting other than mere deficiency of the milk constituents.

In some cases it may be due to deficient secretion of the Intestinal juices leading to imperfect digestion of the food or, perhaps, imperfect absorption of it, and when by giving suitable aperients one stimulates the secretion of the intestinal glands, the digestion and absorption are restored to normal and the child proceeds to put on weight. Further, the process of wasting may be intensified by restlessness of the child owing to colic and loss of sleep, thereby expending much muscular energy.

Then there are the mechanical consequences of habitual constipation, such as straining of the abdominal muscles, sometimes causing a hernia usually of the umbilical type.

There may also be a partial prolapse of the Rectum which may - in rare cases - lead to Dilatation of the Colon - the so-called Idiopathic Dilatation of the Colon or Hirschsprung's disease. This causation is disputed, however, the contention being that Hirschsprung's disease is a congenital defect with chronic constipation its most prominent symptom.

In the second group, i.e. Faults on the part of the Infact, these may be divided into two further classes.

1. A class of cases wherein, despite the best feeding, owing to some error of metabolism or for some unknown reason the infant is unable to assimilate either one or more constituents of his food in the ordinary way, and instead of putting on weight as a normal child would do, his weight chart shows a gradual downward curve which in severe cases proves fatal. This is the True Marasmus, or Simple Atrophy.
2. A class wherein all the other factors causing Infantile Atrophy may be included.

Robert Hutchison gives a list of conditions which may impede the infant's capacity for sucking and which would certainly predispose towards wasting if not relieved. They are as follows:-

1. Local conditions in the mouth such as Stomatitis.
2. Inability to breathe through the nasal air passages being blocked.
3. Other local conditions such as Congenital Cleft Palate or hare lip.
4. Unsuitable teat.

As regards faulty feeding - giving the feeds at irregular intervals - varying the times from day to day - and very often in too large quantities rather than in too strong a mixture - is the commonest form of error on the part of the mother in the writer's experience.

In breast-fed children this tendency is easily understood in an ignorant mother of the working class - for the temptation to give the baby the breast every time he cries must be great if the doing of this is to keep him quiet in order to allow the parent to get on with her housework.

When overfeeding is indulged in in bottle-fed children it is very often due to the well-meant but ill timed advice of the kindly neighbour or maternal grandparent who remarks "The baby is hungry, give him a bottle". In one case of typical atrophy on record when the writer was called in the mother bewailed the fact that she could'nt understand why the child

(aet, 3 months) was not thriving as she thought she was giving him enough nourishment. On inquiry the facts were elicited that the infact had been given 7 bottles of varying quantities in the course of 12 hours! This overfeeding had been going on for several weeks and the child was emaciated.

On the question of giving the correct quantities or otherwise of the various constituents of milk mixture, Still has shown that an infant may tolerate 4% of fat in human milk, but the cream from cows' milk is less easily tolerated, so that the percentage of fat must not exceed 3.5 in artificial feeding; on the other hand a mixture containing less than 2% of fat, in a child over three months, is liable to cause rickets. In the case of sugar a percentage above 8 is likely to cause digestive disturbances with flatulence and pain, while any proportion below 5% will in time show an impoverished nutrition.

A frequent cause of Marasmus is that following an acute gastro-intestinal disturbance - diarrhoea with or without vomiting. A case of this type E.K. 2¾/12 came under observation on 11.8.25. Family History - mother and father alive and well - during pregnancy mother suffered from general weakness with varicose veins. Breast-fed for four weeks, vomited occasionally and stools became ~~so~~ so green, put on cows' milk.

On examination - very thin, loose dry skin. No rash, buttocks healthy, head normal. Chest, abdomen, and limbs show normal appearance. Circulatory and Respiratory systems normal. Alimentary system - good appetite. Mucous membrane of mouth red and tongue furred. Stools three or four per diem, green and undigested. Weight fluctuated between 5 lbs. 12 ozs. and 7 lbs. 4 ozs. between date of first examination and middle of January. i.e. a total gain of 24 ozs only, in 22 weeks. On 22nd January the feeds which had been

| | | |
|---|---|---|
| Skim milk | 200 | |
| Sugar | 75 | Caloric equivalent |
| Orange | 8 | |
| | 283 | |

were strengthened to equal parts whole milk and water 5½ ozs. per feed with one glucose feed making in 24 hours

| | | |
|---|---|---|
| Milk | 300 | |
| Sugar | 87 | |
| Orange | 8 | |
| | 395 | Calories |

This led to a steady gain of an average of 5 ozs. per week till on 12.3.26 he weighed 10 lbs. 10 ozs. ~~i.e. a total gain of 24 ozs. only in 22 weeks.~~

In cases of this type some resist all treatment and it has been suggested that there is a profound alteration in the lining membrane of the intestine which interferes with absorption, but on the other hand an intestinal dyspepsia may be the explanation. There is a distinctive group of cases wherein the Infant shows great difficulty in digesting one

particular type of food. In one case the idiosyncrasy might be shown towards fat and it may be impossible for the child to assimilate more than 1.5 to 2% without digestive trouble whilst he can keep up the normal proportion of protein and sugar (Still). In another case protein may be the "stumbling block" as shown in the following example.

S.P. 3/12. 10.8.25. Wt 8 lbs. 11 ozs.

Father and mother alive and well. Family of 9. Three died (under two years) two of gastro-intestinal disorder and one of bronchitis respectively. Two premature (lived, 6 weeks and 5 minutes), one still-born, one miscarriage. Two girls at 13 and 9 alive and well.

Normal Pregnancy and birth. — On breast three weeks, but was underfed. Robinson's patent food, then Pasteurised milk were tried, but infant lost weight. Horlick's malted milk next tried - temporary gain. Was next put on cows' milk and water, then vomiting and diarrhoea set in 14 days before observation.

There has been occasional vomiting since he was put on the bottle. Stools have been green and undigested. Rash on skin and thrush. Slight cough and discharge from eyes and nose.

On Examination patient was emaciated. Skin dry, loose and shiny. Limbs cold. Head slightly bossed, sutures thickened Fontanelle 1½" x 2". Chest and

abdomen normal in appearance. All systems normal. Urine normal.

Alimentary system - slow feeder, mouth clean. Stools greenish-yellow. In this case the infant was fed on 3½ ozs. of Skim milk and water in the proportion of three parts to two. About 25.9.25 an endeavour to get on to ordinary cows' milk in same dilution was of no avail. Stools continued greenish and loose until 10.11.25 and weight remained stationary till 24.11.25. On this date Nestle's sweetened condensed milk was commenced. ℥iss to ℥iii of water (along with ℥ss of orange juice per day). On the 29th of the same month the feed was increased to ℥iiss to ℥iii water - there was no appreciable change either in the stools or the weight until 5.12.25 when the stools began to improve and the weight has steadily increased to 12 lbs. 6 ozs. on 6.3.26.

So much then for those cases of Simple Atrophy mainly attributable to inability to assimilate either one or more of the constituents of normal milk.

The next section that comes under observation is that of Simple Atrophy from deprivation or "Avitamosis". This section has reference to the important question of Vitamines which have already been described, and the Etiological factors are governed by whichever of the three Vitamines may be lacking in the diet either singly or in combination.

As the Etiology of the Vitamines is closely connected with the Pathogenesis it is proposed to leave over its further discussion to a subsequent chapter.

There still remains a large group of Atrophies which, unlike the foregoing, are associated with other diseases and infections. Included in this group are all cases of Organic disease such as Pyloric-Stenosis, Pyelitis, Otitis Media, Syphilis, Tuberculosis, Broncho-pneumonia and infective conditions such as Acute Epidemic Diarrhoea, etc. which may result in a Marasmic Condition; and these different complications will be dealt with briefly.

Pyloric-stenosis - the chief features of this condition are vomiting and wasting, but there has been very little dietetic error to account for these symptoms - in most cases the infant has been breast-fed, or/carefully bottle-fed. Very occasionally does the vomiting begin from birth usually commencing about the 3rd or 4th week and the vomiting is typical. One mother described it in her infant as "throwing like a whale". As the wasting continues the abdomen gets pear shaped and exaggerated gastric peristalsis is visible. In most cases the thickened pylorus can be felt.

In connection with Pyelitis and Broncho-pneumonia, a case that came under the notice of the writer recently

is of interest.

Peter D. aet 4/12 on 28.9.25.

History that 2 days after birth a condition of imperforate anus was discovered and the motions were being passed per penem. A colostomy was performed and the child appeared to thrive, weighing 7 lbs. 5 ozs. at 4 months. His weight increased slowly but without remission until 14.11.25 when it had risen to 9 lbs. The temperature uptill now had remained normal but about this date 17.11.25, the infant showed signs of chest complications which later developed into pneumonia with swinging temperature. The weight remained stationary with slight variations until 5.12.25. when it started to fall until on the day of the child's death 17.12.25 the weight was 7 lbs. 14. ozs.

The Urine was normal uptill the beginning of December but on examination on 12.12.25. it was found to be Acid in reaction, opalescent. Some albumen, no blood and on microscopical examination a large number of pus cells was demonstrated with some granular casts and epithelial debris. Post Mortem examination showed - condition of pneumonia at base of both lungs, liver and spleen enlarged and both kidneys were lobulated.

Syphilis as a cause of Marasmus, though of great frequency, is sometimes difficult to detect and therefore very often "missed". It is well known that

the Wassermann test is not reliable in infants under 6 months, and then again it has been shown that the exhibition of Hydrarg c Cret has produced excellent results in cases which have been thought to be non-specific in origin. Usually the atrophic condition dates from birth; though the infant may have been born apparently healthy, he rapidly wastes and in a large proportion of the cases the outlook is gloomy.

Tuberculosis practically never occurs in infants under 3 months and rarely under 6 months as a cause of Marasmus.

A condition causing Marasmus which can be easily "missed" is Congenital Heart Disease, in those cases, where, unlike the typical Blue boy appearance associated with a patent Foramen Ovale, etc., there are no visible signs of heart disease and only when attention is called to the poor nutrition of the child and a routine examination is carried out, is the diagnosis of congenital heart disease decided on as the cause of the malnutrition.

C.A. Botwood, H.S. Raper and Edith Wilcock in a recent article endeavour to show that in some cases dyspepsia appears to have been the original cause of Marasmus. In cases that have failed to respond to dietetic endocrine or vitamine treatment, they suggest that Anoxemia may be the cause. They state that when Post Mortem examinations have failed to demonstrate any

pathological changes, the condition might be due to a fundamental physiological factor such as anoxemia which may be either, general due to some deficiency in the proper oxygenation of the lungs or, local due possibly to a capillary Stasis in the intestines as the result of a more or less severe initial digestive disturbance. They are not certain whether capillary stasis can occur in a single organ thus causing local anoxemia, but they point out that the condition simulating surgical shock (which has been generally accepted to be associated with widespread capillary stasis) has been produced experimentally in animals under anaesthesia by the injection of histamine which is a product capable of production by tissue disintegration. They suggest, therefore, that under certain conditions dysfunction of a tissue may be brought about locally by some toxic process as a result of bacterial action, thus causing local anoxemia due to capillary stasis. They carried out a series of experiments by which they increased the supply of oxygen (by placing the infant in an air-tight box into which oxygen was introduced) until the atmosphere contained 45% of oxygen. The child was exposed to this atmosphere for 2 hours on the first day, 4 hours on the second, gradually increasing the length of

exposure up to 8 hours. This treatment was carried out for periods varying from 2 to 3 weeks, and they showed that out of 16 cases 13 improved markedly - the improvement commencing within the first week.

## 4. PATHOGENESIS OF DEFICIENCY DISEASES.

Under this section it is proposed to give a short account of the recent views as to the causation of the different diseases which are grouped under the term Deficiency diseases in order that they may be differentiated from the true Marasmus.

### 1. Infantile Muscular Dystrophy.

Although this condition is not included in the list of the deficiency disorders, a short outline of its pathogenesis is given in order to bring out the differences clinically between this and early simple Marasmus.

Under the term Muscular Dystrophy, or Myopathic Muscular Atrophy one finds a group of diseases in which muscular weakness and atrophy result from primary changes in the muscles. In some forms an initial increase in size occurs in certain muscles, but this condition is usually found later on in childhood and does not come within the scope of this thesis.

No predisposing factors are known as to their cause other than hereditary and familial, and the onset may commence almost immediately after birth. The muscle fibres are atrophied, nuclei increase in number, and in those cases where hypertrophy is present - muscle fibres are increased in size but not in number; there is

an increase of connective tissue and an excess of fat. This is only a temporary condition and later the enlarged muscles atrophy. The nervous system is normal though in severe cases there may be slight secondary changes in the anterior horns of the cord.

There is an atypical modification of this condition occurring rarely, in which the fingers, wrists, toes and ankles, and occasionally the face are affected only.

Amyotomia congenita (Oppenheim first described this condition as Myatomia) is a congenital affection showing a general flaccidity of the muscles and absence of deep reflexes, usually appearing at birth or shortly afterwards; in this case there is no hereditary or familial history, and it is probably a primary disease of the muscles. On examination the muscles are very flaccid, and small but not atrophied, joints are abnormally moveable. Weakness of muscles is very marked but they have voluntary control and there is no paralysis. Faradic reaction is diminished. Both sets of limbs are affected especially the lower limbs, and the face usually escapes.

Progressive muscular atrophy of childhood (Werding-Hoffmann Type) is a very rare disease with symptoms ressembling progressive Muscular Atrophy commencing in infancy with pathological changes ressembling amyotrophic lateral Sclerosis. There is an extensive degeneration

of the anterior horns and pyramidal tracts. This disease occurs in families and can be transmitted by either sex. Its progress is slow. It commences with symmetrical paresis and atrophy of muscles affecting the proximal segments of limbs, thigh, trunk, and pelvis, later upper limbs and neck. Reflexes absent, muscles flaccid and there may be fibrillary tremors. Electrical reactions are diminished or there may be reaction of degeneration, but no ~~too~~ sensory alterations.

2. Infantile Scurvy.

A deficiency disease occurring in infancy due to the absence of the Anti-Scorbutic accessory food factor from the diet, and characterised chiefly by sub-periosteal haemmorhage and anaemia. In earlier descriptions, this condition was confused with rickets, for, though both scurvy and rickets are deficiency diseases and are due to absence of insufficient supply of vitamine in the food, it has since been demonstrated that they each are caused by the absence of a distinctive vitamine, though both conditions may occur simultaneously owing to a lack of the necessary anti-scorbutic and anti-rachitic accessory food factors. As has already been described it occurs in children who have been nourished on proprietary or preserved foods, wherein the vitamines have been destroyed in the process of preparation or on cows' milk which has been boiled and where no fresh juices have been

given as a preventitive.

The haemorrhages occur between the periosteum and the bone and fractures or separation of epiphyses are common. There is usually a swelling at the lower end of femur of an indefinite nature, never into the joint however, and there is rarefaction of bone and even haemorrhages into marrow. There is swelling of the gums around the teeth, but if there are no teeth the gums are normal.

3. Rickets.

A disease of metabolism occuring in late infancy and due to deficiency in the Anti-rachitic or Fat Soluble A. accessory food factor. This disease very rarely occurs in breast-fed babies and rarely under 6 months.

As has already been described this Fat Soluble A. vitamine occurs mainly in fats so that deficiency of fats in the diet is the chief factor in producing rickets. There is no question of Heredity though Syphilis aggravates the condition. Experiments by Mellanby and others on puppies show that puppies develop Rickets on a diet deficient in a vitamine similar to Fat Soluble A. Further that the absence of this vitamine is not the sole cause of rickets, because the absence of Fat Soluble A. inhibits growth, whereas rickets develops

most rapidly in growing puppies and in apparently well nourished babies. Therefore it would seem, that more than one factor is involved in the production of rickets and experiments tend to show that any excessive supply of carbohydrates is the probable explanation. Mellanby concludes that "a diet containing an excess of carbohydrates (and possibly otherwise ill-selected) especially when associated with rapid growth and with a deposition of fat requires a certain amount of anti-rachitic vitamine, and rickets result in the absence of that amount", this means that there is not necessarily an absolute deficiency of this vitamine - there may be a relative deficiency.

It has been shown that Light has an influence on Rickets and cures have been effected by means of sunlight natural and artificial. The lack of light on the other hand is a strong predisposing factor to Rickets as shown by the prevalence of this disease in the poorer classes living in slums.

Deficiency of calcium has been suggested as a cause of Rickets, but it is generally accepted that this is not the case unless the diet is otherwise deficient; at the same time, however, lack of calcium will result in the softening of the long bones which will aggravate rickets.

4. Ber-beri.

A condition due to the absence from the diet of

Water Soluble B. accessory food factor, and is characterised by multiple peripheral neuritis, along with oedema, effusions and cardiac weakness. This is the first and probably the most fully investigated of the deficiency diseases and is closely associated with rice dietary, and it occurs when the aleurone layer (containing phosphates and fats) and especially the germ (which has been proved by Chick and Hume to contain for the most part the anti-beri-beri vitamine) are simultaneously removed and discarded by the polishing or steam-milling process.

Apart from polished rice diets outbreaks of beri-beri will occur anywhere if the diet is

1. Deficient in anti-beri-beri vitamines.
2. Sterilised or autoclaved.

These outbreaks have been known to occur in ships, jails, and other institutions.

Beri-beri at various times has caused enormous mortality amongst infants and post-mortem changes have been shown by McLaughlin and Andrews, 1909, to be similar to those found in adults.

Two definite types of this disease in infants have been noted.

1. Acute type - the more common, in which there is sudden paroxysmal pain and tachycardia. Attack follows attack with increasing violence and death occurs within a few hours.

2. Chronic type - less common, in which one finds vomiting, constipation, tachycardia and oedema. There is no paralysis. Death usually supervenes.

The age of occurrence in infants is usually over three months and under one year and is due to deficient vitamines in the milk of a beri-beri mother.

5. Acidosis.

Acidosis - or Acetonaemia - whilst of special significance in adults as indicating an effect of carbohydrate starvation due to abstinence from, or deprivation of, food, or by elimination of carbohydrates from the diet, is often induced in children by very trifling causes, such as apparently insignificant changes in diet. R.S. Frew examined the urine of 600 children admitted to Hospital and found acetone in 61.6 % during the first three or four days following their admission. The maximum reaction occured about 36 hours after the child was admitted, and after that time the acetonaemia tended to disappear. On analysing the cases it was shown that the presence of acetonaemia bore no obvious relation to the disease for which the patient was admitted, that it tended to occur more frequently in the younger children but it was less frequent in children under one year unless they were changed from breast to artificial feeding. Frew attributes this temporary acetonaemia to change of diet and to a temporary loss of ability

to assimilate carbohydrates due to such a change. Among the morbid conditions causing the more severe forms of acetonaemia are cases where vomiting is a prominent symptom, e.g. cyclic vomiting or delayed chloroform poisoning. The cause in this case is not easy to define because the presence of acetonaemia is not constant in all cases of persistent vomiting - it sometimes appears before the vomiting commences - so that its presence cannot be ascribed to the effects of starvation as a result of the rejection of all food.

Acetonaemia has also been found in pneumonia and broncho-pneumonia.

6. Cyclic Vomiting.

This condition first described by Gee consists of attacks of severe vomiting accompanied by prostration recurring at more or less periodic intervals. The attack usually lasts for two or three days, although it has been known to last as long as 14 days.

On post-mortem examination the only constant change found has been marked fatty degeneration of the liver. Langmead noted the enormous size of the fat globules in the liver cells,-only in acute yellow atrophy and phosphorus poisoning are they found as large. Slight degenerative and necrotic changes have also been found in the mucous membrane of the stomach and intestines.

Mellanby found, in a case which he investigated chiefly from the chemical point of view, that the patient constantly excreted a certain amount of creatin in the urine, although a normal child does not excrete creatin and that the creatin excretion reached a maximum during the attack. He is inclined to attribute this to abnormal intestinal decomposition. On the other hand, the acidosis which is often such a prominent feature of cyclic vomiting and to which many have attributed the production of the symptoms, is now believed to be merely a by-product of the pathological chemical process at work and to be the result of the acute starvation which the vomiting induces, although as has already been stated it may be responsible, when once developed, for the actual occurrence of the attack.

There may also be a permanent "hepatic inadequacy" in those patients, which is accentuated during an attack and may be shown by a failure of the liver to efficiently deal with fat. The genesis of this disease may therefore be ascribed to two factors, namely that

1. The diet which contains an excess of fat forming food may throw too great a strain on the liver, and

2. Constipation may lead to the formation and accumulation of excessive toxins in the intestine.

7. <u>Oedemas found in wasting conditions</u>.

A form of oedema is found in wasting children which may be due either to prolonged failure of digestion or

to chronic diarrhoea. It first appears on the dorsum of the hands and feet and thence extends up the legs and forearm. The swollen limbs are usually cold and there may be a blue mottling.

The urine is usually albumen-free, though there is occasionally a slight trace. The causation of this oedema in wasting conditions in not clear. It has been suggested, as in other oedemas, that retention of chlorides in the system may be a cause and it has been shown that Citrate of Soda given excessively has produced oedema in a wasting infant. As it occurs, however, almost always in a child suffering from wasting and exhaustion it would appear that this condition may be due to some altered state of the blood, or to some alteration in the structure of the capillaries along with feebleness of the circulation.

## 5. CLINICAL FEATURES OF SIMPLE ATROPHY.

In most cases of Infantile Marasmus from whatever cause, the incipient symptoms follow more or less along the same lines. The usual history is that the baby was delicate from birth or perhaps that he appeared to be born healthy and remained healthy until the mother's milk began to fail and he was put on artificial feeding. Various foods were tried to no purpose and the child continued to lose flesh, vomiting and diarrhoea may have occurred frequently; along with loss in weight the length of body remained stationary and development is generally retarded. Teething may be delayed, though in certain types dentition may progress normally without discomfort; in one case known to the writer the child was born with three teeth. The infant is more or less anaemic, sharp-featured, and wasted with dark rings round his eyes, deficient elasticity of the skin, poor circulation and low temperature. He is fretful, irritable and sleeps badly. The appetite is poor and capricious, indigestion is common and attacks of diarrhoea are frequent. The fontanelle is depressed, the muscles are flabby, subcutaneous tissues have wasted and the skin appears "to fit loosely".

As the condition gets worse, there is extreme emaciation which gives the old man appearance with a skeleton-like body. The abdomen is usually distended, often tense with a semi-transparent appearance and large blue veins showing. The complexion pale at first, later assumes a pasty gray colour while the mucous membrane of the exaggeratedly huge mouth has a dark red hue. The urine is usually negative as regards albumen and sugar. The stools vary - usually inclined to be dry and constipated though there is often diarrhoea. Occasionally at intervals the stools may be almost normal and formed.

In Fat indigestion there is constipation as a rule with pale dry crumbly soapy stools, whereas where Carbodydrate indigestion is the cause diarrhoea is a prominent feature with acid stools, green and watery, with a variable amount of mucous and small whitish curd masses.

In the former, calcium and in the latter, potassium and sodiumsalt are excreted in the faeces, and this demineralisation of the body is generally accepted as being the sole cause of Atrophy.

During the fretful irritable stage the child may devour his food ravenously, later he becomes dull and lethargic. Pulse usually becomes slow and irregular. Respiration becomes slower and occasionally Cheynes -

Stokes in type. Oedema and cyanosis are often present in severe cases, the former showing first on the dorsum of the feet, later in the eyelids and rest of the body. There is no effusion into the serous cavities. Petechial haemorrhages often appear on the lower part of the abdomen, later spreading over the trunk, upper part of the thighs and elsewhere.

Infectious diarrhoea is particularly fatal though it has little effect on the temperature, pulse and respiration.

Terminal pneumonia is a common result and it may show few respiratory symptoms.

As the symptoms in these cases are so negative in character and the wasting so gradual, occasionally the young mother fails to realise the severity of the case, and certain cases of sudden death are attributable to this type, death being due to sudden failure of the heart or respiration, from a general nervous collapse or from the rapid onset of acute symptoms of intoxication following an unsuitable meal.

An interesting feature of this condition is the high sensitivity shown by the little patient to nutritive, infective and other outside influences. For instance a slight alteration in the quality and quantity of the nourishment may result in an immediate exacerbation - a minor bacterial infection, a coryza

or a bronchitis may lead to a serious decline. A little overheating may produce high fever and collapse. This sensitiveness is very often of considerable importance diagnostically when the child is undergoing a remission, or is beginning to recover and even when the symptoms of the body loss are not apparent. This reduced immunity predisposes to such infective complications as furunculosis, pyelitis, bronchitis, pneumonia, etc. A case of pyelitis and pneumonia with death supervening has been already quoted under Etiology.

The term "Reversion" has been applied to a clinical feature which sometimes occurs and is of especial significance in diagnosing Marasmus wherein the patient appears to be doing exceeding well for several days, weight is being put on steadily when suddenly through some slight error in diet or some external injury the patient loses all the weight gained and is left even weaker than before. The increase in weight has in fact been due to a retention of fluid in the system- possibly liver engorgement-rather than due to an increase of healthy tissues.

The following case is of interest in this respect.

John B. first seen 23.11.25. aet 2½/12.

Mother complained that he was not thriving and was suffering from constipation. F. and M. alive and well

aet 28 and 24.

1 Boy premature 7 months, died 1 week.

1 Boy aet 3 a. and w. suffered from atrophic dyspepsia in infancy. Now a. and w.

Patient. He was breast-fed for 1 month then weaned owing to constipation (in both mother and child) He was then fed on Pasteurised milk and milk 1 - 2 parts every three hours with a little sugar added. This mixture was continued for 5 weeks, then increased gradually in strength and Magnesia was given every other day. The mother thinks he was thriving on this mixture and the bowels had become regular and normal when he was started on Allenbury's No. 1 (two feeds in the day). The stools promptly showed white lumps of undigested material.

On examination the child had a fairly healthy appearance, the general nutrition was fair. Complexion pale, skin warm dry and fairly elastic. Anterior fontanelle 2" x 2", not depressed, no bossing. Weight 6 lbs. 14 ozs.

The mouth and tongue were clean, mucous membrane healthy, appetite healthy. Abdomen moderately distended - no resistance or tenderness. Liver and spleen normal. Circulatory system normal. Respiratory system - an occasional rhonchus was heard on inspiration Urine nothing to note.

Infant was put on three hourly feeds of

equal parts milk and water 3 ozs. in all with 3ss sugar (and 3i orange juice once daily). He lost weight 6 ozs. during the first week. The feeds were increased to 3½ ozs. equal parts and he very slowly put on weight without loss. On 25.12.25 he weighed 7 lbs. Stools were improving greenish yellow. On 3rd January he was put on a 4 oz. feed, two parts milk to one of water and 3iv of sugar. By the 29th January he was getting 4½ ozs. (2 parts milk to one water) sugar 3v . Stools were normal and weight 8 lbs. On 22.2.26 feeds had been increased to 5 ozs. (milk 3½, water 1½) and sugar 3v ; stools occasionally greenish yellow varying from one to three motions per day and weight had reached 8 lbs. 13 ozs. on 12.3.26. when there occurred a sudden fall within two days to 8 lbs. 2 ozs. i.e. 11 ozs. in 48 hours. 20 C.C. normal saline was injected subcutaneously and feeds consisting of equal parts Whey and Glucose 3i in 3iv water were given. 24 hours later a further 20 C.C. normal saline was given. By 17.3.26. the weight had increased by 14 ozs. to 9 lbs. and skim milk feeds 3v 3 hourly with 3i sugar are being given. It is interesting to note that along with the injection of saline the temperature rose to 100.7° but returned to normal in 24 hours.

As a correct interpretation of the different conditions found as the result of errors in feeding is essential to their successful treatment, a brief account of the clinical features occurring owing to either excess or deficiency of the three main constituents of milk is necessary.

1. Protein in excess - an excessive amount or the wrong type is one of the commonest causes of serious gastric and intestinal disturbances in the young infant. One finds frequent vomiting, colic, flatulence with green offensive motions. Constipation may be present at first but when the protein has been given in great excess the increased irritation of the bowel produces diarrhoea. The stomach and intestines are in a pathological condition owing to chemical changes occurring in the masses of undigested albuminoid material, and this requires appropriate treatment before the defect in the food can be dealt with.

2. Deficiency in Protein - a deficiency in protein affects the whole of the tissues. For a time the infant may maintain a surprising amount of vigour and increase in weight on a diet rich in carbohydrates but lacking in sufficient protein. A good example of this type of feeding is sweetened condensed milk. The baby is deceptive in appearance being chubby but flabby, and they are undoubtedly in the first stage of rickets with an

atonic condition of the muscles and soft parts. If the condition is allowed to progress a severe anaemia results. There is severe headache and pains in the limbs accompanying the anaemia. A haemic murmur may be got on auscultation and even oedema has been known to occur.

3. Excess of Fat. Effects due to excess of fat show vomiting shortly after feeding along with diarrhoea and green motions. A high fat and a high protein content tend to be antagonistic, therefore in these cases symptoms may disappear on a reduction in the amount of protein. A healthy infant requires not less than 3% of fat and not more than 4 %. Death may occur rapidly if this is exceeded from acute fat intoxication. Severe constipation is the first symptom. The combination of the excessive amount of fat with the precipitated ~~xikkxtka~~ casein gives rise to the formation of a pale dry crumbly stool which cannot be digested and its greasy tendency inhibits peristalsis. There is therefore a partial or complete intestinal obstruction with the vomiting of a watery fluid. Vomiting occurs about 2 hours after food and is due to reflex irritation. The appearance of the child is characteristic. The skin is white and wax-like and the body is oedematous. Appetite is lost and there is extreme drowsiness or partial unconsciousness.

Usually, marked strabismus and convulsions are frequent.

4. Deficiency of Fat. When there is deficiency of fat in the diet it follows that the Fat Soluble A. accessory food factor is also in insufficient quantity, therefore a condition of rickets will occur. There is constipation associated with a chronic indigestion in the form of abdominal pain between feeds and if the dieting is not corrected rickets with all its sequelae will result.

5. Sugar. The chemical properties of milk sugar are important in as much as they take part in the production of lactic acid, and without the lactic acid, bacilli cannot carry out the normal intestinal digestion.

When Lactose is deficient, there are symptoms of intestinal irritation in the shape of diarrhoea and skin eruptions.

Jones conducted an autopsy on three cases of Marasmus and found the organs almost all normal. All the tissues were pale and anaemic. The stomach and intestines were distended and the walls were thin. The liver in one case showed fatty degeneration. The mesenteric glands were slightly enlarged in all cases, the lungs showed small areas of atelectasis, so that deficient lung expansion would account for the leaden hue often seen in such cases. One case showed during life opisthotonos and contraction of both legs and arms but nothing more was discovered post-mortem.

The Diagnosis of simple Atrophy is easy and depends on the history of the case to a great extent. The patient is usually in the first few weeks of life and he has been ailing continuously. Dyspepsia, Diarrhoea, and infectous conditions predispose. It may follow a period of starvation for diarrhoea. Moreover, it is important that a correct diagnosis be made between cases of simple dyspepsia and marasmic conditions before subjecting what is thought to be the former to a line of treatment suitable for such cases (namely a diet rich in carbohydrates and a period of starvation) lest serious injury be caused to the child suffering from the more severe condition.

The viscera should be examined carefully in every case, for the diagnosis must be determined by the absence of symptoms pointing to a local disease. For example, wasting may be the only apparent sign in insidious cases of empyema or broncho-pneumonia in infants. Congenital heart disease - not of the obvious blue-boy and club-fingered type - has also been mentioned. Marasmus of syphilitic origin too is very common and is especially indicative when breast-fed cases show signs of wasting. Though tuberculosis is extremely rare in infants under 6 months the symptomatic emaciation concomitant with this disease and other diseases leading to cachexia must not be forgotten.

The Clinical Features and Symptomatology of those cases of Atrophy due to deprivation of vitamins will now be considered.

1. Infantile Scurvy occurs about the end of the 6th month and is of slow origin - the child having probably been fed on a scorbutic diet for several months before the characteristic symptoms commence - marked by a slowly developing state of cachexia. There is extreme listlessness and debility with a rapid pulse and disinclination for movement of any kind owing to extreme tenderness. The child screams when touched and both legs are often everted.. There is anaemia and the face has a sallow earthy tint in severe cases.

If the condition is allowed to progress a pseudo-paralysis of one of the lower limbs develops more or less suddenly. This pseudo-paralysis is caused by a sub-periosteal haemorrhage which usually occurs at the lower end of the femur though sometimes at upper end of tibia. There is a sinking-in of the costo-chondral articulation. Effusion of blood and oedema may also occur in the muscles and intra-muscular spaces. The joints are not usually affected. In severe cases soft crepitus may be elicited from separation of the epiphyses and even a fracture of the shaft of the bone may occur. There may be harmorrhages into the orbit with proptosis and in

rare cases haematuria. The gums, pale at first, later show small sub-mucous haemorrhages - and if several teeth have appeared they become red, swollen, spongy and ulcerated. The heart is sometimes dilated or hypertrophied and sometimes tachycardia occurs as an attack. Scurvy differs from simple malnutrition in that it causes a definite retardation in the growth in length.

Boils, nasal catarrhs, and septic conditions of the urinary system are indicative of lowered resistance. Diagnosis is easy in well marked cases but is sometimes missed in mild cases when complicated by the presence of some other disease. In all cases of scurvy there is present a variable degree of nervousness, cachexia or debility.

A fracture of femur, an acute periostitis of the same bone, and angio-neurotic oedema of the thigh might be mistaken for scurvy.

2. Rickets does not occur before the fourth month and often much later. In most cases excessive perspiration on the neck and head and less commonly over rest of body is the first symptom - most severe during sleep. There may be restlessness in bed and throwing off the bed-clothes - though healthy children often do this. A disinclination to be moved is the next stage owing to a varying amount of tenderness of

the limbs (if this tenderness be well marked there is probably an attendant scurvy).

There is often recurrent diarrhoea alternating with constipation, in the former the stools are slimy and offensive, and bronchitic attacks are frequent. Convulsions are frequent, the child is often fat but flabby; in severe forms there is wasting and he is irritable and has a poor appetite.

The changes in the bones are typical - beading of the ribs or "Rickety-Rosary", and enlarged epiphyses at the costo-chondral articulations are the most constant and often the earliest symptoms of rickets. Pigeon-breast- the sternum projects especially at its lower half and the costo-chondral junction is sunk - a section through the thorax gives a triangular appearance. Harrison's Sulcus - a groove from ensiform cartilage outwards with costal margin turned upwards. Epiphyses are enlarged especially those of lower end of radius, and less commonly of tibia and femur.

The Head is enlarged, square and flattened on vertex (caput quadratum) less commonly lengthened antero-posteriorly. The anterior fontanelle remains patent until 2nd or 3rd year. Bossing of frontal eminences. Craniotabes is often found - sometimes associated with syphilis. Curvature of long bones owing to weight of body and muscular traction. The

commonest deformities occur in the tibia with

1. at its lower third with concavity on its external surface or a sharp forward bend.
2. at its upper third with concavity on internal surface - "bowlegs".

Deformity of femur is less common . When it does occur there is a general antero-posterior curve. Rarely coxa-vara and genu valgum. In the spine, kyphosis is got if the child sits up, whilst curvatures are uncommon in the upper extremities unless the child crawls on his arms. The pelvis is flattened antero-posteriorly, may be small and contracted. Greenstick fractures are common owing to friability of the bony structure. Permanent dwarfing may result from the deformities and disease of the epiphyses.

The ligaments are extremely lax and there is great weakness of the muscles simulating paralysis. The diagnosis is difficult in mild types but simple in severe cases. The rapidity of the onset and loss of reflexes would preclude infantile paralysis. Spinal Caries might present difficulties, but in this condition there is a sharp local curve and manifestations such as long bone deformities are absent.

3. Ber-beri occurs in infants between the age of 3 months and one year and is due to deficient vitamines in the milk of a mother suffering from beri-beri.

There are two distinct types.

1. The Acute type which is much the commonest. The infant gets repeated attacks of sudden paroxysmal pain and tachycardia increasing in severity and death supervenes in a few hours.

2. The Chronic type is less common. The chief symptoms are vomiting, constipation, tachycardia and oedema. There is no paralysis. There is more chance of recovery from this type but in severe cases death takes place in the acute form.

## 6. TREATMENT.

Correct feeding forms the sum and substance of the successful treatment of Marasmus.

The recent progress (mainly by von Perquet and Schick of Vienna and Finkelstein in Berlin and Marriott and Ultheim of St. Louis) has lain chiefly in the recognition that stools are not the immediate determining guide to the feeding of the infant; that many infants thrive only when they receive concentrated milk mixtures, that many infants require 150 - 200 calories per kilogram of body weight and finally that milk fermented with the lactic acid producing group of bacilli is more easily digested by many infants than is sweet milk.

It has recently been shown that in artificially fed infants a frequency of stools may not be of serious moment - not that this state of affairs is normal, but that it is due to partial starvation, and the condition is ameliorated by increasing the quantity of food usually with a greater proportion of carbohydrate. Finkelstein was the first to show that diarrhoea in early infancy does not necessarily denote indigestion and, that diarrhoea is more frequently the result of an insufficiency

of food in a qualitative or quantitative sense, or of some idiosyncrasy in the constitution of the infant, than the result of damage to digestive functions. In many cases infants will do better when fed on concentrated foods than when on diluted foods. The premature infant will not thrive on a dilute mixture either because he is not strong enough or cannot retain a sufficient amount to suit his requirements. Vomiting is more often the result of mechanical conditions than of indigestion, and for that reason vomiting in the great majority of instances can be cured by giving small quantities of concentrated food at frequent intervals. Even in pyloric stenosis this method of procedure will sometimes prove effective.

The question of the necessity of diluting cows' milk is disputed. It is generally agreed that milk soured by lactic acid producing organisms does not require diluting, but few infants are able to digest ordinary milk undiluted - not so much because it is too concentrated, as because it is improperly balanced. The addition of citrate of soda has the same effect in softening the curd as has the lactic acid preparation. Arthus and Pages showed that clotting of milk could be entirely prevented by precipitating the Calcium Salts with Oxalates and Fluorides. Sir Almroth Wright substituted Citrate of Soda. There is more Calcium

in cows' milk than there is in human milk.

Dr. T. Wood Clarke at the Rockefeller Institute attributes the value of Citrate of Soda to the fact that it acts on the HCl of the stomach thus converting it into NaCl and thus markedly reducing the available acid.

Langmead showed that undiluted milk was better with Citrate of Soda for the reason that

1. Addition of water increases the bulk of the feed.

2. If Citrated milk be diluted the fat and sugar are reduced to proportions below that in human milk, which means adding cream and sugar.

3. There is difficulty with the varying degrees of dilution.

In the breast the transition from colostrum to coagulable milk is relatively slow probably 14 days (Pritchard). During this period the stomach is functionally developing; it is acquiring tolerance for coagulated casein and learning to peptonise or to liquify the clot soon after its formation; acting on this theory Dr. Dicks (London Infirmary) dealing with infants belonging to the poorest classes, finds it safest to give them whey during the first fortnight in life.

Citrate of Soda has been known to cause oedema, so should be employed with caution.

The following is the system of bottle feeding in atrophic infants in the University of California Hospital

as explained by Spalding. Certified milk with germ count of less than 10,000 per c.c.m. averages 3.6 % butter fat. Various top milks are obtained from the quart bottles of milk with a Chapin dipper which average :-

top 12 ozs. 8.5 %
top 14 ozs. 7.5 %
top 16 ozs. 6.5 %
top 20 ozs. 5.5 %,

lower 20 0.5 %

Whey from whole milk averaging 1% fat and 1% albumen, and whey from skim milk are also used. The different milks average about 5% sugar and 3.5 % proteid except in the case of the whey. For carbohydrates pure milk sugar and Robinson's barley are used.

Salts of Sodium and Magnesium (namely NaCl) are used to aid the gastric digestion of casein and to promote absorption of the different proteids. Occasional resort is made to buttermilk, malt sugars, and dextrinised gruels, but as a routine such feeding is not equal to carefully prepared and modified fresh cows' milk.

The immature babies are started on Sugar Solution 5% or NaCl Solution (0.5 %), and the atrophic babies with whey, in order to ascertain the amount of liquid which the infant will retain. Gradually the sugar solution is replaced with whey and the whey replaced with one of the top milk, whole milk or fat free milk mixtures according to the behaviour of the child.

Barley water is not added unlessthe food contains from 20 to 30 % of milk until the sugars fail to agree. The quantity of food is increased to meet the desires of the child and its ability to retain the feed. The relationship between the weight chart, the clinical chart and the feeding chart is closely studied and at intervals the percentage formula of the food: the calories with the energy quotient and the proteid ratio are estimated to avoid continued overfeeding or underfeeding. The strength of the gruel is important. A rounded tablespoonful of Robinson's barley to 12 ozs. of water cooked for half an hour and the water loss replaced makes a 5% solution of barley.

When an immature or atrophic infant can digest 10% whey then milk (usually top milk 16) is added and this is increased gradually but constantly until the baby can digest from 30 to 40% of milk. With this 5% of sugar and 0.5% of salt is also given, and 10% of the 5% barley gruel is added and gradually increased as the whey is eliminated. According to Spalding these infants do better when the ratio of fat to proteid in the food is not more than 2 to 1 and while the number of calories needed for the normal infant may not be more than 100 per kilo. of body weight, and can gradually be reduced as the child grows older, he has found it necessary to feed the immature and atrophic

infants on a diet containing as much as 250 calories per kilo. of body weight for several weeks at a time, and with food of this high caloric value he has seldom found signs of overfeeding except when the fat calories ran high. However, when a baby digests fat successfully, he is fed on fat but he is watched carefully and the fat is reduced with the first signs of indigestion. "The weight chart urges always for more food and the clinical chart acts to hold this in check. Intelligent interpretation of these two charts indicates the sensible way in which to feed such cases of malnutrition." (Spalding).

Christopher Rolleston quotes an article by P. Nobécourt in Archives de Medecine des Enfants.

Nobecourt controls the vomiting of infants by the addition of 10% ordinary sugar lactose or saccharose. This has a sedative action and evokes a considerable liquid secretion in the stomach which may diminish activity and consequently decrease any erosive action. Cachectic infants lose flesh rapidly and the loss of heat becomes greater. The consumption of albumen rises and urea accumulates in the fluids of the body. An easily digested and assimilated food is therefore necessary. Fat and albumen are difficult to digest, while sugar is easily digested and furnishes 4 calories to each gram ingested.

W. Langford Symes recommends alcholic dextrines "in cases of Marasmus which defy all our efforts and

most approved methods of feeding, where cows' milk has failed and has been abandoned ~~easily~~ even though skilfully modified, peptonised or condensed, where breast-feeding has likewise failed, also acid milk, where patent foods have possibly aggravated the condition and where special Marasmus food, such as bread jelly, wine whey, eggs, meat juice and brandy have disappointed - ~~XXX~~ give Dublin Stout with, after a time, the addition of proteids and fat."

When first an atrophic infant comes under notice the digestive functions are almost at a standstill because he has probably been fed on sterile or cooked food for a long period. A wet nurse is the ideal means of attempting to effect a cure; where this is not possible and where asses' milk is not obtainable as an alternative, the treatment must be undertaken with great caution and the natural food must be given in small proportions. Each case must be treated on its individual merits, or, one should say, according to the pathological conditions one has to deal with. As regards fats - the fat should rarely exceed 1% to start with, slowly increasing the quantity as tolerance is established.

Lactose may be given in normal amounts or may want reducing since the normal amounts of lactic acid may prove too strong for the infant and diarrhoea result. In such cases dextri-maltose combination may be better sustained.

Proteins should be given cautiously and it is usually advisable to commence by giving them in the form of whey only. Casein is poorly tolerated at first, but casein digestion is not so severely interfered with as a rule as is fat digestion. The rules followed in the treatment of fat indigestion are regulated according to whether diarrhoea is present or not.

1. If diarrhoea is present - as the result of peristaltic hurry there is rapid passage through the intestine and fat splitting may be poor. In this case the treatment consists in putting the infant on a low fat dietary and giving carbohydrate in the form of one of the less fermentable sugars, e.g. dextri-maltose. Skim milk or perhaps a mixture of skim and whole along with dextri-maltose supplies the necessary amount of fat and is the mixture which is usually employed. A condensed sweetened milk diluted to lower proportion of fat content may be used. Protein milk has also been tried with success. Then as the diarrhoea improves the fat content should be increased until the child is on a normal milk mixture.

2. The other type of fat indigestion occurs when high fat content dried milks are given and in these cases fat splitting is always good, the stool is large and contains much soap and free fatty acid. The treatment in this case consists in giving a lower fat content

dried milk or substituting an ordinary cows' milk mixture for the dried milk.

In certain cases where a period of starvation at the commencement is inadvisable owing to the condition of the infant glucose feeds ℥i - ℥ii in ℥ii - ℥iii of water in 3 to 4 hourly feeds have proved beneficial.

Peptonised milk is often of value where other forms of milk are not tolerated. This is diluted at first with equal parts of water and gradually increased in strength. Where ordinary cows' milk is indicated and cannot be digested completely by the child Citrate of Soda at the rate of gr x to ʒi has been employed successfully as a means of softening the curd. No cases of oedema have occurred in the writer's experience though used in several cases. This treatment of the milk is much preferred to that treated by lactic acid.

As regards Protein-milk - first recommended by Finkelstein, it is especially of value where there is a tendency to excessive acid fermentation. It acts by reducing the milk sugar content of the milk by a dilution of whey, which, in itself, improves the tolerance of the intestine for sugar and by the subsequent addition of large quantities of protein which tend to counteract the acid fermentation by the development of an alkaline reaction.

In cases of rapid dehydration as evinced by a sudden drop in weight, subcutaneous injections of normal saline 5 c.c. even up to 20 c.c. have been highly successful (case already quoted).

In cases of haemorrhage especially in early life 5 c.c. injection of blood from parent is highly successful. In the early month of life the infant's blood is not classified, therefore any type of blood may be employed .

As regards drugs, an initial dose of castor oil is often efficacious. In other cases small doses of Hydrarg ℂ Cret gr. 1/12 - 1/3 t.i.d. are very useful as an intestinal antiseptic.

Cod liver oil is heavy for many infants and generally speaking malt mixed with the feeds is more digestible.

Small doses of Syrup of Hypophosphite either with or without malt have responded remarkably well in certain cases.

Thyroid Extract gr ⅓ twice or thrice daily has given good results in chosen cases.

A final word regarding those deficiency diseases due to the absence of vitamines in the diet - chief of which are Scurvy, Rickets and Beri-beri. These conditions yield rapidly to dietetic treatment by supplying food containing the required vitamine in each case.

1. Scurvy - give no proprietary or preserved foods, give unboiled milk and give some fresh juice

either orange, grape or even swede juice up to ℥iv daily or raw meat juice.

Drugs are valueless. In severe cases wrap limbs in cotton wool and put cage to keep weight of bed clothes.

2. Rickets - a sufficiency of fat is essential. To each bottle of milk add a ℥i of cream. Cod liver oil ℥ss to ℥i t.i.d with or without extract of malt. In older children, yolk of egg is useful. Avoid too much starch.

3. Beri-beri - as this disease in infants is due to deficient vitamines in the milk of the beri-beri mother, the mother requires treatment as well as the child. Chamberlain and Vedder recommend 20 drops of Extract of rice polishings every two hours and later rice polishings to infant and mother.

## 7. SUMMARY AND CONCLUSIONS.

1. Accessory food factors or vitamines are essential to life, and therefore, the absence of any one or more from the food will give rise to a series of symptoms according to which constituent is deficient or absent.

2. An infant is said to be suffering from True Marasmus if and when he shows a loss of 20% of body weight (average).

3. The term Marasmus or Simple Atrophy is applied only to those cases wherein there is no predisposing or concomitant condition such as Tuberculosis, Syphilis, and is almost invariably due to improper or imperfect nutrition.

4. Anomalies of fat and carbohydrate assimilation are the commonest causes of Simple Atrophy though errors in protein metabolism are also responsible for this condition.

5. Dehydration and demineralisation occur as a result of 4. and are therefore an immediate cause.

6. It is <u>not</u> those foods with the highest fat content that are most economical from the fat absorption point of view, for foods of this type such as full cream dried milk may throw too great a strain on the infant's fat metabolism which may lead to "fat intolerance".

7. It is important thatmilk foods should not be subjected to too much heat in order to avoid destroying the vitamins and further with bottle-fed infants it is advisable to give fresh fruit juice, preferably orange juice once daily to ensure a sufficient supply of vitamins.

8. In the causation of any deficiency disease, for example rickets, there need not be an absolute deficiency of the essential vitamine. There may be a relative deficiency.

9. Acetonaemia is not necessarily a pathological condition in infants. This condition is often found when the infant is changing over from the breast to artificial food and is due to temporary inability on the part of the infant to assimilate the carbohydrates consequent on this change.

10. Atrophy of Syphilitic origin is the most likely diagnosis when one gets loss of weight in a breast-feeding infant.

11. Wassermann test is not reliable in infants under 6 months.

12. Correct feeding forms the sum and substance of successful treatment of Marasmus, but there are certain cases wherein despite the most careful attention to those details of dietary the infant will not respond to treatment and with the gradual loss of weight and other symptoms death supervenes.

Further

13. /in artificially fed infants a frequency of stools does not necessarily mean indigestion. It may be due to a partial starvation and, if so, it can be relieved by increasing the quantity of food with a greater proportion of carbyhydrate.

14. In a premature or marasmic infant vomiting may be the result of a mechanical condition produced by overloading the stomach, and such cases do well on small concentrated feeds at frequent intervals.

15. For the treatment of Marasmus or Simple Atrophy a wet nurse should be obtained if possible, failing which asses' milk is a good substitute. When the latter is unobtainable and when the infant is unable to assimilate diluted cows' milk, milk treated with Citrate of Soda and undiluted ispreferable to that treated with Lactic Acid.

16. Sudden fall in the weight chart of an atrophic infant is due, not so much to the rapid loss of healthy tissue, as to the sudden dehydration of unhealthy oedematous tissue.

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