"THESIS for the DEGREE of DOCTOR of MEDICINE"

presented by THOMAS F. JOHNSTON, M.B., C.M.Ed.

"A CONTRIBUTION ON RICKETS"

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"Subject"

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In choosing a subject for my thesis I have experienced considerable difficulty, firstly in getting an appropriate subject and secondly in doing that subject sufficient justice. When a man has access to a laboratory he has ample opportunity of doing some original research work. To a man daily engaged in busy practice such opportunity is very limited and what original work he may do can only be in a clinical form, that is anything original, either symptomatic or Therapeutic, in connexion with the numerous forms of disease with which he daily comes in contact. It is under these circumstances that I have chosen Rickets as a subject for my thesis. How often has one not heard the Phrase, "Only a case of Rickets" as if to imply that such a case were trivial, of no consequence and beneath the notice of the average practitioner, but when one comes to realise the important and far-reaching changes that are apt to occur in the development and the growth of a rickety child, and how such a child, especially the female, is handicapped in life, we see that it is a very important disease indeed and quite worthy of the

greatest care and attention of every practitioner whose lot it is to treat such cases. To those who are constantly called to treat cases of rickets I need offer little excuse for my choice of this subject, but I feel assured that it will appeal to the Physician, the Surgeon and the Gynæcologist. How often is the physician handicapped in his treatment of pulmonary cases by rickety changes in the chest! how often is the surgeon called upon to operate upon rickety deformities, amongst the most common of which I may mention Knock Knee, and how often is the obstetrician confronted with Rickety Pelvis! The hospital physician, however willing he may be to enter into the study of Rickets, is seriously handicapped, inasmuch as he does not see much of the onset of the disease, which is its most interesting stage, but it is usually in its later stage that it comes under his observation. In fact he sees the cases that have been a failure in home treatment and have been sent to hospital as a last resource. While cases in the later stage pour in upon him he is unable to fill his beds with cases of chronic Rickets as he has to admit more urgent cases. Therefore his opportunities for careful clinical study are limited. In large towns where the great majority of cases occur amongst the poorer classes, the acute stage is frequently not observed, or, if it is, it is most likely treated by the mother, or by the chemist who may recommend a few simple

powders. Even if the case is recognised, and advised for hospital treatment, the mother is usually and naturally unwilling to leave her last-born in hospital, yet I am convinced that this is the time when active treatment may be undertaken with the greatest hope of success. In a large working class and colliery practice such as mine the state of things is different. The doctor knows all the families under his care and he keeps an eye on each member of the community from the time he brings him or her into the world. As the practice is a contract one and his visits are not charged, he is more frequently called in to treat the slighter ailments of childhood, than would be the case if his visits were paid for, and, as part of his contract is to supply medicine, the chemist has fewer opportunities of prescribing. During his daily visits to the houses of his patients, he has constantly before him the agencies affecting the health of the children and therefore has not to rely so much on the mother for a history of the surroundings and a description of the dietary of her children. Now, whilst the colliery doctor has these advantages in the clinical study of Rickets, it must be noted that in his busy life, some distance from the large towns, he is quite unable to follow up his observations by research work in a laboratory. It is nevertheless his duty to contribute what little he can, trusting that it may be of use in the solution

of one of our most difficult medical problems. What I am about to write has been learned in the course of my attendance, during the last six years, on some seven hundred families, in the North of England, where Rickets is fairly common. In my district 27% of the children born suffer from Rickets but this appears to be a much lower proportion than what occurs in many other places.

("Schwarz has observed rickety changes in 80% of cases at the second Vienna obstetric clinic.")

In writing I base my facts on 200 consecutive cases. I shall deal very lightly with the history and the geographical distribution of the disease as I am unable to add to what is already known in these respects and, for a reason previously given, I shall omit that part of the study which falls to be done in the laboratory or the Post Mortem room.

History. During the early part of the seventeenth century a peculiar, and at that time unknown, disease appeared in the counties of Dorset and Somerset and then gradually spread over the whole of England. The attention of the Royal College of Physicians was directed to the disease and a commission was formed, a member of which named Glisson first described the disease under the term Rachitis. His description was the first known publication of it and the history does not go back beyond this time, but, when we consider the origin and the

In 1895. Dr. Laing of Blyth read a paper, before the Society of Antiquaries of Newcastle-on-Tyne - on a Celtic Skeleton found in a stone Kist. on the banks of the Tarrelt burn near Bellingham in Northumberland.
The skeleton was supposed to be 2000 years old & was that of a female. It shewed well marked Rickety deformities in the Pelvis and lower limbs. The skeleton is now in the "Blackgate" Museum Newcastle-on-Tyne

cause of the affection, we may refer it to a more remote period of history. Famine and pestilence were fairly common in the early and middle ages and no doubt as a consequence Rickets in the younger members of the community would be anything but rare. No doubt feeding bottles and artificial feeding would be unknown and the doctrine of "Survival of the fittest" would play an important part in the weeding out of weakly children, yet the conditions of life such as famine, lack of Hygiene, etc. would play an important part in the causation of the disease. Rickets must have been peculiar to England before being introduced to the Continent of Europe, as the Germans first described it as "Morbus Anglicus" or the English disease. After Glisson had described the symptoms of the new disease "Mayow" of Oxford in 1660 called attention to an important characteristic of the disease, viz. the softening of the bones. More than a century later, in 1751, "Duxerney" described the pathology and the anatomy. Again in the nineteenth century Frousseau and Virchow described the modern disease.

Rickets is most commonly met with in temperate zones. The disease is practically unknown in the extreme North, viz. Greenland, Iceland, Norway and Denmark, and it is also rare in southern climates, but natives of southern lands who have settled in northern climates are apt to have rickety children. Holt, in his text book on diseases of children, has

pointed out that the most extreme cases of rickets met with in New York were of Italian or Negro parents. In England the disease is met with everywhere and more especially in towns and the crowded manufacturing districts, and is most common in Glasgow and the big Towns of Lancashire. The reason for this is perhaps mostly due to the fact that women play such an important part in the labour of these districts, and that a great percentage of the children are reared on artificial diet. A woman from her labour in the mills and factories etc. cannot afford to nurse her child. It is consequently left at home to the mercy of the feeding bottle and unskilled nursing. In the agricultural districts of England the disease is rare.

Definition. Rickets as we see it in the North of England is a disease almost invariably starting in Infancy and early childhood and usually ceasing about the fourth or fifth year. Beginning as an acute illness it passes into a chronic Inflammatory state, affecting the long bones, causing softening of these bones and marked changes in the neighbourhood of their Epiphyses. These changes take the form of enlargement of the ends of the long bones in the region where the bone and the cartilage are in contact. Consequent on the softening striking and lasting results are produced by the bending of their shafts. There is also evidence of changes in the Viscera.

Three varieties of Rickets are generally recognised. These are:-

I. Fœtal Rickets. This term is applied to those rare cases, where a child is born in such a condition as to suggest the existence of Rickets during its fœtal life. They usually have all the deformities of ordinary rachitic cases, such as enlargement of the Epiphyses, curving of the long bones, beading of the ribs, and the usual chest deformity. Very often the child is born with numerous fractures of the long bones, which are no doubt traumatic and caused by pressure exerted on it in its passage through the maternal passages. Such children are very often premature and are either still-born or are in so debilitated a condition that they die very soon after delivery. These may be instances of true rickets or they may be instances where there has been an arrest of development of the osseous and cartilaginous tissues of the child during its fœtal existence. They must be remarkably rare, as, in over 1000 cases of confinement which I have attended, I have never noticed a case which I could describe as fœtal rickets. On one or two occasions I have had fractures during delivery but these happened in labours where the head did not present.

II. Late Rickets. By this we mean Rickets that appear after the age of four or five. Sometimes there is no evidence of previous rickets in

infancy and these cases may be attributed to any debilitating illness. Of late Rickets I have had three cases, one beginning at the 8th year, the second at the 11th year, and the third at the 15th year. At this period of life the bones of the head and the epiphyses of the long bones have undergone more complete ossification and we do not get the deformities that are consequent upon the rickety condition in infancy and early childhood. We do get enlargement of the epiphyses of the femur causing knock knee, and this condition happened in the eight year old case. No doubt also lateral curvature of the spine and flat foot are a result of late rickets.

III. The ordinary rickets that occurs in Infancy and early childhood and which is now under consideration.

Etiology. All children appear to be liable to rickets but there are certain predisposing causes to which special attention has been paid. Chief of these are any unhealthy condition of the mother during the period of pregnancy and syphilis and tubercule. As for exciting causes we may mention:-

I. Artificial feeding.

II. Nursing by a debilitated mother. Under this heading we may include those cases that arise from prolonged lactation. There is no doubt that cases arising between 12 and 18 months are

mostly due to this cause as the breast milk has probably been impoverished and deteriorated in proteid fat and also phosphates. I hope to demonstrate this in the course of the paper.

III. Unfavourable Hygienic surroundings, such as general neglect, cold and damp.

IV. Lack of fresh air and sunshine. It may not be so much a lack of these as an excess of vitiated air in the atmosphere in which the child lives.

From what I have observed in the cases I have attended, I have been led to believe that improper dieting is by far the most important of these. The disease, beginning as it does with a febrile attack and affecting as it probably does the whole body, is likely to be of a toxic origin and in several respects it bears a resemblance to chronic Gout and Rheumatism, which have long been held to be of this nature. If so, we appear at present quite unable to determine if the toxin is organismal or where it is produced.

Age.

As I have previously stated, I have never observed a case of fœtal rickets. The youngest child I have seen affected was 9 weeks old but was also syphilitic. Of the cases in which I have been able to determine the date of onset, I have found it as follows:-

21 % beginning during 1st six months.
26 % " " 2nd six months.
31 % " " 3rd six months.
12.5% " " 4th six months.
4.5% " " 5th six months.
2 % " " 6th six months.
2 % " " 3rd and 5th years.
1 % " " 5th year.

I have seen so many cases of delayed dentition that my impression is that of the 21% of cases beginning during the first six months of life I have made too low an estimate. In collecting my statistics, however, I did not accept delayed dentition alone as a positive evidence of rickets beginning during the first six months. Had I done so, the percentage would have been over 30, and perhaps this would have been nearer the truth, as the onset of the disease which I enquired for might easily have been overlooked in so young a child. These age periods will be further considered under the influence of diet.

Sex. Of my 200 cases, 113 were females and 87 males. This gives a decided tendency towards females, but, looking back over all the cases I have seen, I think the sexes are affected in about equal proportion. Sex does not appear to have any influence on the age at which the disease begins or on its severity.

Heredity. I do not believe that the disease is hereditary. I have seen healthy children grow up when both parents were affected with unmistakable rickety

deformities. Curiously in the three cases of late rickets I have observed, there was evidence of paternal rickets in each, whilst the mother was healthy and appeared to have escaped.

ealth of parents. I have been unable to trace any predisposition to rickets in the child, brought about by the state of the father's health, but the heads of nearly all our families here are in good health for if they are unable to pursue the arduous and fatiguing colliery work they have to go elsewhere for lighter employment. If the mother is in poor health during gestation and lactation the child is very likely to be rickety, but of all diseases that predispose to Rickets, Tubercule and Syphilis appear to exert the most marked influence. I have watched four children of mothers suffering from malignant disease grow up, and three of them escaped rickets. The mothers in this district never have to go out to work as the mothers in the mill towns of Lancashire and other places have to do. Consequently the mother has all her time to devote to the rearing of her infants and a greater percentage are kept on the breast. This fact may account for Rickets being less prevalent in the North of England than in the large manufacturing towns of Lancashire. The children of mothers under 20 years of age suffer to a greater extent than the children of mothers between 20 and 35. This, I think, is due to the fact that women under 20 are practically children themselves and

have absolutely no idea of the rearing of an infant. In these cases the breasts are often not fully developed and the milk is poor and scanty, which necessitates the child being brought up by bottle. In other cases the child never gets the chance of the breast, as the mother does not feel fit for the responsibility of nursing and realises that nursing will interfere seriously with her own pleasure. After the age of 35 there is again an increase in the proportion of children affected. Probably at this age a woman is in the decline of her reproductive life. The children I have noticed are apt to be born small and the mother from deficient milk is often unable to nurse and consequently the children are again artificially fed.

The following table is made from 200 consecutive births, after excluding children dying under 2 years of age without exhibiting signs of Rickets:-

Age of mother	Number of children born	Number affected with Rickets	%
15 to 20 years	18	7	38·8
20 to 25 years	41	6	14·6
25 to 30 years	46	11	23·8
30 to 35 years	43	12	27·9
35 to 40 years	38	14	38·8
40 to 45 years	11	5	45·5
Over 45 years	3	1	not reliable

We therefore see from this table that a greater % of children born between the age of 15 and 20 years and over the age of 40 years are liable to rickets than children born at other periods of life.

Fresh Air, Light, Heat, etc. Lack of these has been given as a cause of Rickets. After careful consideration of my experience of the cases coming under my observation, I cannot look upon them as an important factor in the causation of the disease. All of the pitmen's houses here are built in rows running North and South and facing the Sea. They have doors at the back and front of the house, and these doors usually lead directly into the living room of the house. One of these doors is open for the greater part of the day and there is good through ventilation. Further the fires, which are very large and in spacious chimneys, are kept burning all night and these make the chimneys sufficient extraction shafts day and night. The houses are free from recesses or box beds in which impure air might lodge. I am frequently called to visit their bedrooms at all hours of the night and can testify that the air in these rooms compares favourably in purity etc. with the air in the larger and more hygienic bedrooms of the better classes. The children are out in the open air during the greater part of the day, for, as soon as the child is able to walk, he finds his way outside through the open door. The rooms have ample windows which ensure plenty of sunlight. Two rows of houses

from their position are much damper than the other rows, but I have never noticed that children brought up in these rows suffer from rickets in greater proportion to children in the others, but I have noticed that Pulmonary complaints and Rheumatism are of more common occurrence in these houses. The Sanitary system is good, for, though the pail system is in use, the closets are well removed from the houses and are partly open to the air, and the scavenging is thorough and frequent. I have not been able to trace any relation between the time of the year the child is born and the tendency to rickets. I am not trying to argue that the absence of fresh air, sunlight, etc., may not be very important in the causation of this disease. Indeed, I feel sure that the absence of these must have an unfavourable influence, for my experience is that any factor which lowers the vitality of the child will predispose to Rickets. I merely wish to point out that in my district these causes are conspicuous by their absence, and so, while they may be looked upon as predisposing, they are certainly not the active agents. As regards the influence of the number of children in a family, I have studied this point with considerable care and tried to tabulate the results, but, on looking carefully into the question, I found the results so much affected by other causes, that they appeared quite unreliable. Thus in one family of seven, I found all free from rickets, whereas in a neighbouring house with a

family of four children, all more or less suffered. The feeding of the children, the health of the mother and other causes, were important elements which could not be eliminated. It was easily observed, however, that where the children were born in rapid succession they suffered largely from Rickets, becoming more severe in the younger members of the family.

ocial Position. On this point I cannot write very much as my practice is mostly composed of the mining population, which lives outside the Town. I have observed, however, that Rickets is by no means uncommon amongst children of the higher orders in the district, but these cases are not attended with such marked deformities. This must be attributed to the care taken by the better classes when their attention is drawn to the means by which commencing deformities can be remedied. The children of the farmers and the farm labourers appear to suffer very little.

Food and Feeding. My experience has shown me that this is by far the most important factor to consider in dealing with cases of Rickets. In practically all my cases I could detect some gross error in the feeding of the child. The chief sources of danger lie in the improper use of proprietary foods, the improper treatment of milk when the child is on the bottle, and in keeping the child on the breast too long. The worst cases of Rickets I have attended come under this heading and were children who had been kept on the breast for 16 or 18 months and in

addition were being fed on potatoes from the mother's plate at dinner. Here we have the child being fed mainly on impoverished milk - milk probably deficient in proteid and fat - and further, from its potato diet, having an excess of starch, practically a proteid fat starvation with excess of carbohydrate. Amongst breast fed children, provided the mother is healthy, the disease is almost unknown during the first year. The cases occurring during the first year are those amongst children taken off the breast at an early date and fed on an unsuitable substitute. At the beginning of the second year cases appear amongst children who were healthy until removed from the breast, about the 9th or 10th month, the natural time at which an infant should be weaned. These cases are seldom severe and are very amenable to suitable treatment. During the latter part of the second year cases due to too prolonged lactation are added to these. These latter are usually very severe. The disease seldom begins after the second year; when it does, the causes are not due to too prolonged lactation alone, as these have appeared before the end of the second year, although in one bad case the child had actually been suckled for nearly 2½ years, but they are cases of mixed feeding or are caused by prolonged use of patent starchy foods for delicate children. I shall now consider this point in detail in connection with the 200 cases already noted, in which I was able to

determine the date of the onset of the disease.

Of the 21% of the cases beginning before the end of the sixth month, none had been breast fed during the whole period. Five (i.e. of the 42) were twins. Various reasons were given for not suckling the children, such as ill-health, fissured and painful nipples, and abscess of the breast, which condition was always secondary to primary condition in the nipple. Others had been told that the milk was not suitable for the child. In a few the secretion was defective, as in premature confinement and in elderly women. These were cases, no doubt, of slow development of the breast. In many cases where the mother has been unable to suckle the child, I have had the opportunity of enforcing the necessity of feeding the child with fresh cow's milk, treated to make its composition resemble that of human milk. In very few of these cases in which I felt sure my instructions were being carried out, has the child become rickety, and where reasonable care had been taken the cases were of a mild type. Rules laid down at the end of the puerperium for the feeding of the child rest lightly on the mother. The grand-parents, the wet-nurse, or even the neighbours have much advice to give on the rearing of the infant, and often the young mother, seeing that everything is new and strange to her, is apt to take the advice, or may be forced to obey, even against her own better judgment. On the question of Infant

feeding in this locality I shall describe the usual routine. A large proportion are brought up on the breast and these are by far the healthiest. No regular system of feeding is adopted, unless one calls the irregularity as to time of suckling a system, the time for giving the breast in most cases being whenever the child cries. The breast becomes a universal soother. The majority of children are not weaned until they are from 14 to 18 months old and for the following reasons. Cow's milk is scarce and the mother, provided she has secretion, finds it cheaper and more convenient to keep the child on the breast. Again, she has a natural desire to limit her family and she usually has the idea that she cannot conceive as long as she is suckling. How erroneous this idea is may be shewn by the fact that she does conceive and her first warning of this is quickening and only then is the child weaned. I frequently find examples of this and cases where the mother has never menstrucated from one confinement to the next are common. Another reason is that in these cases the child is long in getting his teeth and the mother has the impression that it is necessary to keep the child on the breast until he gets his first teeth, being unaware, in her ignorance, that the prolonged lactation is the cause of the delayed dentition. The want of system in suckling infants is bad, but the slipshod and careless manner in which bottle-fed children are brought up is

infinitely worse and is a disgrace to the mother. The usual method is briefly thus. A long tubed bottle is filled or nearly so with a mixture of milk (which may be either cow's or condensed) and water, or of infant's food and water in varying proportion. This bottle is put in the crib or cradle alongside the infant, the teat of the tube stuck in its mouth, and the child left to suck, much as it wishes. No attention is paid to the quantity or composition of the fluid in the bottle, little to the temperature of the mixture, still less to the cleanliness of the bottle, and least of all to the unfortunate infant. Time of feeding is of no account, nor is the quantity and the composition of the fluid in regard to the age of the child ever considered. The crying of the infant is the sole guide as to whether it wants food or not. This method, or want of method, is erroneously supposed to save time and trouble. When one considers the careless habits of the majority of the females, one can imagine that a kind of tubing nearly impossible to keep clean, even with the utmost care and attention, is likely to become soon contaminated with dirt. When one considers that the careless habits of the mother with regard to the feeding usually spread to the keeping clean of the infant, and when one knows that the exhalations from the child's skin and lungs and from what passes through its bowels and bladder are sources of contamination to the food, that very often lead to serious gastro

intestinal derangement, and when one considers that these exhalations are usually confined within the narrow limits of a cradle, one cannot wonder how rickets can but occur and how a healthy race can be reared. Quite lately I went into a house and found a boy of eight, who had been left at home to nurse the baby in the mother's absence, busily engaged filling the feeding bottle with milk of his own composition, probably from directions left by the mother. If the infant survives this feeding for a few months he is usually put on to all sorts of food, chief of which are Boiley - which is scalded bread with milk - baked wheat flour, Infant's food, and potatoes. It is only the other day that I saw the result of the inquest on a child in a neighbouring colliery village, which child, aged 6 months, had died suddenly of convulsions produced by the mother giving it potato chips. This mode of infant feeding described cannot but lay the foundations of rickets and usually results in a high infant mortality. Year after year the annual reports of the various Medical Officers in the district deplore this high infantile mortality, and from what I have said the cause is not far to seek and is due entirely to want of knowledge on the part of the mothers. Knowledge in these matters is, however, fortunately spreading, thanks chiefly to the energetic crusade of the profession in the district, and we are gradually getting those long tubed bottles relegated to

the dust heap and getting a knowledge, however slight, instilled into the mother of the proper method of feeding her children, and I am pleased to say with good results. These children under 6 months suffer severely. They are very liable to convulsions, about 10% being affected. Of the 42 children under 6 months suffering from rickets, 11, i.e. 26%, died before the end of the first year, the causes of death being as follows:-

(a) Wasting associated with Gastro Intestinal trouble, 3.

(b) Bronchitis, 2.

(c) Convulsions, 2.

(d) Infantile Diarrhœa, 2.

(e) Whooping Cough, 1.

(f) Scurvy, 1.

All these troubles were greatly aggravated by rickets.

In this district the children brought up on the bottle are fed on those articles commonly advertised as Infant's food, and those most advertised are the favourites. The condensed milks save much trouble, as they only require the addition of water and are therefore largely used. These are all unsuited for children, especially the sweetened brands, as are also foods containing unconverted starch. When these cause excessive sickness and diarrhœa, barley water is usually resorted to, and as the sickness ceases, sometimes the child is fed on nothing but the barley water for a few days.

Another patent food is given a trial, or perhaps something still more harmful to the child is concocted. I remember one case in which a young mother brought her five months old child to me in a most wretched condition and with marked rickets. She assured me she had done everything possible for the child and that she was now giving it daily the nourishment of ½ lb. of meat, and yet it would not thrive. The actual history of the case was, that, when the child was a month old, she had been persuaded to take it off the breast, for no obvious reason. One patent food after another was tried, each without success. Then the poor mother, driven to despair and evidently taking her idea from the feeding of adult invalids with beef tea, boiled ½ lb. of meat daily and fed the child with the extract of this alone for a week. I was much surprised that the child had survived this well-meant but unusual method of nourishing.

Of the 26% of cases beginning during the second half of the first year, the majority consisted of children mostly brought up on proprietary foods but supplemented by breast feeding or the use of fresh cow's milk. A number of cases occurred amongst children brought up on cow's milk, and a smaller number amongst children brought up on the breast for a reasonable length of time. The following figures are from the cases I collected:-

I. Children brought up mostly on patent foods = 32.

II. Children brought up mostly on cow's milk = 13.

III. Children brought up mostly on Breast = 7.

In many of the cases in classes II and III there was some marked dietetic error associated with Gastric or Intestinal trouble. Too frequent feeding was common and in many of the cases in class II the cow's milk had been improperly treated. In only two cases, and these were in class III, did I fail to find any error in dieting and for the onset of these two cases I am unable to ascribe any cause. In one case I had a sample of the breast milk analysed and this is the result of the analysis:-

Total Solids... ...	13·09	per cent.
Fat	4·03	" "
Milk Sugar	5·82	" "
Proteids	2·36	" "
Salts	·18	" "

In this milk the percentage of fat is much the same as in ordinary healthy milk and the percentage of proteid is above the average, but there is a marked diminution in the amount of milk sugar - over 1 per cent.

The greatest proportion of cases begin during the first half of the second year - 31% in my table. These are cases brought up on the breast till about the 9th month and afterwards fed on one of the

patent starch foods or are brought up mainly on condensed milk. I notice too that children about this age are beginning to get a portion of whatever is going in the way of food. I find that these cases are not so severe as those in the first year, except in some instances where I found that the child was getting an excess of potato or had some faulty digestion. Potatoes form one of the staple articles of diet of the pitmen in the North of England, and from their softness and the ease with which they are masticated make a very convenient food for children. Towards the end of this period, viz. the first half of the second year, we begin to come across cases due to too prolonged lactation. They are seldom met, however, before the 16th month and they represent about 16% of the total cases during this period. Of the 12·5% of cases beginning during the latter half of the second year, I put down 18 of the 25 cases, which is 72%, to too prolonged suckling. In four of the cases the child had had up to this period, according to the mother's statement, absolutely no nourishment but the breast. These cases are very severe and lead to the most marked bony changes. The rest of the cases were due to the same cause as the majority during the earlier six months of the second year. In two of the four cases I have mentioned above, when the child had had nothing but breast milk, I had the breast milk examined. Both children had marked

rickets, one at the age of 20 months and the other 21 months, and the mothers were both debilitated, probably due to the prolonged lactation, but there did not seem to be any diminution in the amount of the breast secretion. Their reasons for the prolonged suckling were practically the same. They did not attribute the wasting and rickety condition of their children to the fact of keeping them on the breast too long, but thought that the children would die if put on to proper feeding, and as one child had only his lower central excisors and the other none at all, they had the idea that it would be safer to keep suckling the child until dentition was over. They both had large families and thought that the suckling would prevent pregnancy, which was a mistake in one case as I found that she must have been 3 months pregnant while the child was still on the breast. The analysis of the 20 months case was as follows:-

Total Solids...	...	11·52 per cent.
Proteids ...	...	1·63 " "
Sugar	...	6·45 " "
Fat	...	3·3 " "
Salts	...	·14 " "

The analysis of the milk from the 21 months case was as under. The woman in this case was more debilitated than in No. 1.

Total Solids...	...	10·11 per cent.
Proteids ...	...	1·17 " "
Sugar	...	6· " "
Fats	...	2·78 " "
Salts	...	·16 " "

In both cases there is considerable diminution of the solid constituents of the milk and markedly so in the second. If we suppose that the milk began to deteriorate from the 10th month, at which time both children were healthy, we see that for a period of 10 months they had been fed on milk deficient in Proteid, and specially so in the fat and salts, and at a time when, on account of rapid growth etc., the child required a more highly nitrogenous diet than obtained from the mother's milk. Both mothers said the child had had nothing else, but very possibly they meant nothing else in the way of supplementary milk. Probably the children would be getting potatoes in addition, which would certainly have the effect of aggravating the rickety condition. Both of these children died, one from Broncho Pneumonia, and the other from Convulsions associated with dentition.

Symptoms. These may be noted during three stages of the disease:-

I. The stage where the child is ill with the symptoms of an acute Fever.

II. The stage in which the child is anæmic and during which changes take place in the Epiphyses of the bones.

III. The stage in which bony deformities are developing.

These stages run into each other and vary in severity.

The first stage is most marked amongst cases beginning in the first year of life. In almost all cases it is preceded by Gastro Intestinal trouble. This stage usually lasts about two weeks. The child is first feverish at night time, but the temperature tends to rise higher and to remain high for a greater part of the day until the 8th or 9th day. It then gradually falls, and at the end of 2nd week it has probably returned to normal, but for some time afterwards it frequently shows an upward inclination on account of alimentary trouble. At the end of the first week the temperature is usually about 100-101° F. during the day and about a degree higher at night time. The accompanying temperature chart is a fairly good example (see Appendix I). Sweating is a very constant symptom during this stage. At first it is a general perspiration, often accompanied by crops of Sudamina, and is most marked in the evening. In addition to this, about the end of the 1st week, there is a very profuse perspiration from the forehead, very often so profuse as to soak the pillow on which the child's head is lying. This may continue for several weeks. It may occur during any part of the day, but usually comes on a short time after the child has gone to

sleep. This is the time when the child burrows its head in the pillows, causing baldness of the occipital region. Of more importance is an eczema behind the ears which may be observed in a fair proportion of the cases. I make it a rule to enquire for the symptoms of the onset of rickets when a child is brought to me with this condition and if there is no ear-discharge and the case is not one of Impetigo, my enquiries are often correct. During this stage the child is fretful and does not take his usual exercise of screaming and kicking. His face indicates pain when his limbs are moved and nursing in his mother's arms is not grateful or soothing to him, as it is to almost all other children. The fretfulness leads me to believe that the pain is not very severe, as I have found that when young children are suffering very acute pain, they are generally mute, though you may read suffering in their expression. The pain in the limbs appears to be in the muscles and not in the bones or joints. The child will cry out when you gently squeeze or pinch his muscles, while you may tap the skull or tibia or move the joints without causing pain. The muscles around the spine are often particularly tender. This tenderness usually passes off as the temperature falls and then the child becomes very restless at night and peevish during the day. This night restlessness has been noted by many observers to be accompanied by kicking

off of the bed clothes. This symptom may last three or four weeks and is very troublesome. On no account will the child have itself covered up at night and even in the coldest weather will kick off the clothes and lie completely nude. On this account he is apt to get Bronchitis, and, in the words of the mother, gets cold by not keeping himself warm. This kicking off of the bedclothes is not peculiar to rickets, for I have noticed it as a symptom in children suffering from early Broncho Pneumonia and Thread Worms. At the onset of the attack the child is usually constipated and then frequent, small, foul-smelling and clay-coloured or greenish motions are the rule. The tongue at first has a light fur and later it becomes brownish on the surface and very dry. There is usually slight abdominal distension which can be accounted for by the state of the bowels. There is some enlargement of the spleen and sometimes it can be palpated at the end of this stage. Not infrequently there is tenderness over it. The pulse rate is usually quickened, in proportion to the temperature, and varies with the age of the child. At the end of the first stage the child is considerably prostrate and anæmic.

2nd stage. During this stage the child suffers from the effects of the original febrile condition and the bony changes become apparent. About the 3rd week of the disease tenderness can

usually be made out along the Epiphyscal lines, especially of the wrists and knees. The enlargement of the extremities of the long bones shows itself about the fourth week and may be first noticed at the wrists and in the beading of the ribs, and later in the knee and ankle joints. The elbow and shoulder are seldom much affected and the small joints escape. The child suffers from thirst and frequently from diarrhoæ and he is very fretful. The sweating from the head and the restlessness already mentioned continue. During this stage the spleen continues to enlarge and may be palpated in most cases. It may extend almost to the umbilicus. When it cannot be palpated it can seldom be made out by percussion as the chest is very resonant and the abdomen is usually distended. This distension is very common in Rickets and along with the sweating of the head is one of the first signs to come under the notice of the mother. In healthy children the abdomen is large relatively to the size of the chest and this is caused by the flatness of the Diaphragm making the liver more of an abdominal organ in children, and by the small size and shallowness of the Pelvis. In Rickets these causes are exaggerated, thus there is a smaller capacity of the chest with greater flattening of the diaphragm, the liver and spleen are very often enlarged and the capacity of the Pelvis is diminished. Besides there is weakening of the abdominal muscles with lessened

resistance which affords facilities for the accumulation of flatus arising from the usual accompanying Gastro Intestinal trouble. The child is flabby and anæmic. His pale skin has a peculiar dull appearance. He does not resemble the syphilitic child with his muddy skin hanging so loosely and with snuffles and rash. He is equally unlike the tubercular child, with his clear delicate skin, regular features, bright eyes, and small joints.

3rd stage. Usually after the end of the sixth week the child seems to have improved considerably in health. The restlessness, sweating and anæmia pass off to a certain extent and the child begins to take more notice of his surroundings. If old enough, he begins to assume his active habits and attempts to crawl about or get on his feet again. The thickening at the epiphyscal lines of the long bones persists and is usually well marked until the 4th or 5th year. This thickening is most evident at the wrist joint, so evident as to be noticeable at the merest glance. At the first look this enlargement of the wrists seems more apparent than real, for the swollen wrists stand out in great contrast to the shrunk and wasted forearms. It is very difficult to measure a rickety child, or any part of it, with a child of the same age in perfect health, because of the great disparity in the general size, but in several cases of enlarged wrists that I did measure I found enlargement, and

this enlargement varied from $\frac{1}{4}$ to $\frac{5}{8}$ of an inch, taking rickety children with healthy children of the same age, same height, and same length of fore-arm. Seeing that the child is now beginning to move and crawl about and to exercise his muscles, it is in this stage that we get deformities and I will now consider these in the following order.

Head.

On looking at the head of a rickety child, we are struck by its size and by its shape. The head looks larger and more square. The forehead is especially large and prominent and the frontal eminences well marked and there seems to be an increase in the antero-posterior diameter. On examining the head we find the anterior fontanelle abnormally large. Towards the end of the second year, when it ought to be closing, it may be as large as at birth. I have noticed it incompletely ossified at the end of the fifth year. There is usually a groove in the position of the Fronto parietal fissure. The sutures are late in uniting and a well marked ridge of bone can be felt running along either side of the sagittal suture. The ossification may be irregular in the region of the posterior fontanelle. Formed bone seems to be absorbed in places, leaving the skull thin and membraneous in parts which can be easily depressed with the finger and emit a crackling noise on pressure. The bones of the head may be so loose

that at the 12th month it may be possible to slightly depress the edges of the occipitals under the parietals. The hair on these rickety heads is thin and scanty. The face is small, especially the lower jaw, and pinched and stands out in great contrast to the size of the head and seems to make the head appear larger than it really is. These children, especially when the body is wasted, look top heavy.

Spine.

The spinal deformity is in the nature of curvature and this can be easily understood when we consider the softening that takes place in the vertebræ and how easily the vertebræ can be influenced by the movements or posture of the child. Not until the child is able to sit up, or, failing that, is able to be propped up by cushions etc., do these deformities appear. At first, from the size of the head and the weakness of the cervical muscles, the head tends to droop forwards and as the muscles become weaker the head pulls forward the body with it. Naturally this is an uncomfortable position, in which he cannot sleep etc. He therefore allows his head to fall backwards as affording greater support to it and usually lies in that position. This backward position of the head causes an increase in the forward curvature of the spine in the cervical region. In the dorsal region the curvature varies as to whether or not the child has been able to walk. If the child has never

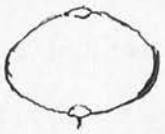
been on his feet we get a posterior curve usually extending from the first dorsal to the last lumbar vertebra. The absence of the normal lumbar anterior curve which is so distinguishing a mark between man and the higher apes is a striking deformity. This posterior curve may be so exaggerated as to look like angular curvature of the spine, but this can be demonstrated not to be so, by the ease with which moderate traction on the spine reduces the curve. If the child has walked this posterior curve is usually limited to the dorsal region and we get the normal anterior lumbar curve and of course we get exaggeration of this in proportion to the softening of the vertebræ and the muscular weakness. A lateral spinal curve is common. This curve is usually to the right and this is usually explained by the position of the child in the nurse's arms and to the frequency with which the child is nursed on the right arm. Vice versa if the child is nursed on the left arm the lateral curve will be to the left. This explanation may be correct but it was well marked in two of my cases of late rickets and these cases were too old to admit of this theory. The curvature was probably due merely to weakness of the muscles of the back and it disappeared when the weight of the legs was allowed to extend the spine.

<u>The Thorax</u>.

The deformities of the bony Thorax are striking

and constant, and this deformity is of the greatest interest to the physician. We get flattening of the back. The ribs as they pass from the vertebræ are bent at an acute angle at the junction of the dorsal and the lateral regions of the chest. It is in this part that we get the greatest lateral diameter. From here the ribs pass forward at an acute angle to their junction with their cartilages and form the smaller arc of a circle. At their point of junction with their cartilages the lateral diameter is the least. The sternum is thrown forwards and the cartilages are bent outwards before they join the ends of the ribs. Through this forward projection of the sternum we get an abnormal antero-posterior diameter. On section the rickety chest has a triangular appearance, thus:

as contrasted with the more oval shape of the normal



It seems to me that the explanation of this deformity is the weight of the heavy viscera in the child's thorax exerting their pressure backwards, when the child is on its back, flattens out the back and the pressure of the diaphragm acting on the softened ribs causes them to curve towards the sternum in a more acute angle. The enlargement of the sternal end of the ribs is well known as beading of the ribs. As the cartilages are curved outwards and the ribs inwards we get a groove running down each side of the chest as far as the 9th or

10th ribs. This groove is less marked on the left side than on the right and especially so in the region of the heart. The rest of the chest wall deformity is, I think, due mainly to the action of the diaphragm. By its continual contraction its tendency is to pull the chest wall inwards and as the chest wall is softened, it is able to make an impression it cannot produce in health. Those portions of ribs at the back are well supported by muscle and are little affected. The sides of the thorax are yielding, however, and are pulled inwards except where they are affected and supported by the abdominal viscera, such as the liver, stomach and spleen. Above this line of support a transverse groove is formed. It has been argued that this transverse groove is not due to the action of the diaphragm, as it does not follow its line of attachment, but rather conforms to a line of demarcation between the yielding and the solid organs of the chest. This, however, is in no way necessary as the diaphragm, with its broad base of attachment, has a strong pull on the whole chest wall and makes a permanent depression except where the ribs receive considerable support. It may be that the recession of the chest walls might be due to lessened pulmonary resistance. If it were so, I think the shape of the chest would be different and, instead of the deformity under consideration, we would have general recession of the chest with diminution of both antero-posterior

and lateral diameters. The liver, stomach and spleen, resisting the action of the diaphragm, maintain a broad base to an otherwise contracted chest and thus gives it a cone shaped appearance. The liver affords a better support on the right side than the stomach and spleen on the left. When an oval yielding body, such as the chest, is on transverse section and is subjected to a constricting force acting equally on all sides, such as the diaphragm, it will elongate in its longest diameter as the greatest amount of force is acting at right angles to this diameter. This is seen in cases of adenoids, where the diaphragm is abnormally developed and exerts an unusual constricting power on the chest wall, diminishing its antero-posterior diameter. This would happen in rickets but for the fact that the rib cartilages do not appear to be affected by rachitic changes and so are able to resist the action of the diaphragm, while the softened ribs are drawn inwards. This explains the lateral longitudinal groove. The lower end of the sternum which is but poorly ossified in early life is often dragged inwards, producing a deep depression.

Pelvis.

The deformities of the Pelvis consequent upon Rickets are very varied, a knowledge of which is of the utmost importance to the obstetrician. These deformities in their severity depend in great part

upon the severity of the disease and the time of life at which the child is affected. If the child has had rickets before getting on his feet the changes in the pelvis may be slight, but if a child has walked while the bones forming the pelvis are in a softened condition the changes produced in the shape of the pelvis are very striking.

The simplest form of pelvic deformity conforms to an Infantile type or what is known as Generally contracted Pelvis. In this Pelvis we get the bones small and poorly developed, the Pubic arch narrow, and all the diameters decreased. This pelvis occurs in cases where the rickets has been more protracted in time than severe in character and where the rickets has existed long enough to interfere with the development of the bones but where the softening has been so slight as to practically cause no deformity. In this type we may get little or no evidence of rachitic changes in any other bones of the body. In some cases of this nature there is evidence of rickets in the Pelvis, shown by the shape of the iliac bones and a relative alteration in the distances between the two superior iliac crests and the two superior iliac spines, and shortening of the antero-posterior diameter.

The most common form of Rickety Pelvis is that usually described as "The Flat Rickety Pelvis" and this is the most typical form. There is usually general contraction as well as flattening and in

the higher degrees of contraction this flattening is very prominent. The bones are usually small and thin but may be hypertrophied in parts. The thickness of the sacrum may be increased and the difference between the true conjugate and the external conjugate greater than usual. The brim of the Pelvis has usually a reniform or kidney shape, owing to the softening and the bulging of the sacrum into the brim of the Pelvis, and this causes marked shortening of the true conjugate. The Pubic arch is more widened than in the normal Pelvis. The sacrum sinks more deeply into the Pelvis, both in the direction towards the brim and towards the coccyx. The promontory of the sacrum is more rotated forwards so that its anterior surface looks more downwards and the curvature is usually increased on its antero-posterior section. The curvature on its transverse section is diminished and is generally converted into a curvature whose convexity looks towards the brim of the Pelvis. The Pelvis as a whole is relatively wide at its outlet. In this form of Pelvis the shape of the iliac fossæ is characteristic. The fossæ are flatter than usual and look more forwards, so that there is marked diminution in the difference between the diameters of the iliac crests and the iliac spines. In very marked cases the diameter of the superior iliac spines exceeds the diameter of the iliac crests. This shape of the iliac bones is probably due to

arrested development and also to the action of the gluteal muscles. The shape of this pelvis is explained by the fact that the deformities are caused before the child is able to walk or stand, and therefore the pressure is entirely through the promontory of the sacrum and the tuberosities of the ischium. The child being in a sitting position the weight of the body tends to force the promontory of the sacrum further in to the cavity of the Pelvis, causing diminution in the true conjugate diameter and the widening is due to the leverage of the innominate bones in the sitting position. The bending inwards of the lower end of the sacrum, which increases the curve on its antero-posterior section, is no doubt due to the pressure exerted on the lower end of the bone while the child is in the sitting position, but is probably also due to muscular action and to the resistance of the Sacro Sciatic ligament to the forward rotation of the promontory.

The most uncommon form of Pelvic deformity is that known as the "Pseudo Malacosteon Pelvis." This form of Pelvis is only found in exceptional cases of Rickets and for its production it is necessary that the bone softening should be exaggerated and that the disease should occur beyond the period of infancy, that is while the child is walking while suffering from it. It resembles a true malacosteon pelvis so much that the diagnosis can only be made by the evidence of Rickets in

other parts of the body and by the relative difference between the diameters of the Iliac crests and the Iliac spines and by the small size of the Pelvis. Again, the bones are hard and not pliant as they are in a Pelvis of mollities ossium. This deformity is easily explained. A child is walking about with a softened Pelvis, and the pressure downwards from the back through the promontory of the Sacrum causes that bone to project into the Pelvis and the pressure upwards from the femora through the acetabulis causes a projection on each side into the Pelvis at that part, giving rise to a Beak shape of the Brim, which simulates very much the true Malacosteon Pelvis.

The Extremities.

In dealing with the lower limbs the deformities all appear to be due to the weight of the body. The upper limbs of older children are seldom affected. Amongst younger children that are crawling about the floor or are able to sit and help to support the weight of the body by means of the arms, curvatures of the upper limbs are common but seldom so severe as those of the lower limbs. The forearm is most affected, the bones being bent and the convexity being on the extensor surface and inner side. There may be a similar curve of the humerus giving the limb somewhat the appearance of a frog's leg. Both of the normal curves of the clavicle are exaggerated. The most marked deformities are in the lower limbs. An antero-posterior curve about the

middle of the femur is common in young children, being due to the weight of the legs acting at right angles to the shaft of the softened femur, when the child is in a sitting position with the legs hanging free. Amongst older children the curvature of the femur is outwards, which is simply an exaggeration of its normal curve, and is due to the weight of the body when the child is in the upright position. From the same cause we may get the less common condition of Coxa Vara in which the neck of the femur is depressed and its angle with the shaft altered. In six years I have come across 2 cases of this, ~~and am able to give a very good sciagraph of one case~~. In the region of the knee the bones are frequently bent, producing knock knee, or bow leg. I have noticed that when the disease comes on before the child is able to walk the resulting condition is usually bow leg. The child sits usually with his legs folded in front, which gives an outward inclination to the bones round the knee, and when the child walks this curve becomes greater. Knock knee is more common among children able to walk at the onset of the disease. Most of these cases also suffer from flat-foot and I believe that the giving way of the arch of the foot in the debilitated child happens first, the weight of the body being then transmitted to the inner side of the limbs causes the knees to incline inwards and consequently the feet outwards. Any abnormal

growth of the internal condyle of the femur, which may take place, is, I think, not primary but compensatory. Rarely do we find knock knee on one side and bow leg on the other. This appears to be caused by the child sitting with both legs bent to the same side. Then the tendency would be for both to be curved either to the right or to the left. Any curvature thus started would be increased when the child begins to walk. An antero-posterior curve in the lower half of the leg due to the weight of the foot is common. The short bones escape deformity but they are small and poorly developed. The small hand of the rickety child exaggerates the enlarged wrist, which in many cases is made still more obvious by the wasting of the forearm. Bony spines at the attachment of muscles are common, especially in the lower limbs. The ligaments round the joints are lax and where bony changes do not affect the movements of the joints mobility may be excessive. This is well exemplified in one of the photographs in the appendix where we see the thighs completely flexed on the abdomen. The possible amount of dorsi flexion of the foot, and the amount of movement at the metacarpo-phalangeal joints are often very great. This excessive mobility and the largeness of the joints frequently lead parents to believe that their children are double-jointed.

Dental Conditions.

Rickets is the great cause of delayed dentition, but it is a mistake to believe that all cases of delayed dentition are due to rickets. I have seen cases where the first teeth did not appear until nearly the end of the first year, and where no certain signs of rickets could be detected. Many delicate children have no teeth until the end of the ninth month. After the lower incisors have erupted sometimes an attack of rickets delays the cutting of the upper. There is thus frequently a long interval after the eruption of the lower central incisors. In one case I noticed an interval of over five months. I have seen many rickety children 1½ years with only two teeth. When dentition is delayed beyond the end of the first year, the symptoms are usually severe and convulsions are not uncommon. It is well-known that these teeth decay easily.

Abdomen.

In all cases of well marked rickets the abdomen is large. I have already mentioned the causes of this but I will here recall them. These are:-

I. The constricted thorax pushing down the diaphragm and the organs lying under it.

II. The liver which is more of an abdominal organ and is said to be enlarged, the spleen is undoubtedly enlarged probably in all cases.

III. The smallness and narrowness of the Pelvis.

IV. In a few cases of rickets the child is fat and there is an unusual deposit in the abdomen.

V. Flatulent distension of the bowel arising from dyspepsia and intestinal fermentation.

VI. Loss of muscular tone of the abdominal wall.

Besides being late in cutting his teeth the child is backward in many ways. He is late in walking, a valuable provision of nature, and also in talking. They are usually dull mentally. They have an old-fashioned look and are very often considered clever and old-fashioned by their parents, but this is no evidence of increased intellect and I think all children who suffer from chronic disease tend to become old fashioned, no doubt from the close association with the adults around them. Rickety subjects grow slowly. Their bones are short and thick and some of them are almost dwarfs.

Complications.

The chief complications of rickets I have found are Bronchitis and Diarrhœa. The Bronchitis is easily explained when we consider the chest deformity. Associated with it we get Emphysema.

The cause of the Diarrhœa is also obvious. Sometimes the Diarrhœa associated with the first stage persists. In other cases, especially in warm weather, the artificial feeding, more so the feeding by unboiled cow's milk, is apt to cause it, and again rickety children, on account of their low vitality, are very subject to become victims of Epidemic Diarrhœa. I can well remember an Epidemic we had

in the hot summer of 1898 killing practically 90% of the bottle fed children in the district.

Diagnosis. This must be considered before and after the deformities have taken place. I wish to lay stress on the early diagnosis of rickets, as this is a point to which I have paid special attention and to which I attach the greatest importance. For some time I failed in practice to recognise the onset of the disease and so could not take advantage of the most favourable time for treatment. This is a point not sufficiently emphasized in books, as the writers of text books have few opportunities for seeing cases in this stage. In the early stage one is called to treat a child with a febrile condition. Now, while febrile states are common amongst children, those causes which keep the temperature up for over a week are limited, and, as we are able at the end of this time to exclude the acute infectious fevers, excepting typhoid, our field for diagnosis is becoming smaller. Typhoid may give trouble for, although it is not common amongst infants, it is likely to occur amongst milk fed children or children not on the breast, just as rickets does. Further, we have the tumid abdomen and dry tongue becoming brown in each. In forming a diagnosis we have to consider the prevalence of typhoid. The temperature of a child with typhoid is not nearly such a useful guide as it is in adults. The variations are much more marked and the course of the disease is less regular.

Typhoid as it affects a young child is a much graver disease than rickets and the child is much more quickly prostrated. In the very early stages of typhoid the limbs are not painful or tender and the child does not resent being nursed in his mother's arms. In those cases which develop a rash, the spots are of the greatest importance, but many cases give Widal's reaction without showing any spots. I should also lay stress on Epistaxis, a symptom not uncommon amongst children with typhoid but not occurring in rickets. The lips and tongue are much drier in typhoid and the fur thicker, and I have never seen the ridges and furrows on the tongue of a child suffering from Rickets. The spleen is more likely to be found enlarged in a child with typhoid than in early rickets. The sweating of the head in rickets is important and after the tenderness of the limbs has passed off the rickety child becomes more restless, while the child with typhoid is becoming more prostrate, more like a log, and sinking deeper in the bed. In doubtful cases the reaction of the blood must be tested. Pulmonary conditions must be differentiated and while the chest must be examined in every case, the friends will always notice the subjective symptoms. Empyema, however, must be carefully guarded against. Abdominal tubercule is more insidious in its onset than rickets, the temperature lasts much longer and shows greater variations and the

sweats are not confined to the head. The abdomen if distended has enlarged glands which can be palpated or contains fluid and the veins lying on the abdominal wall are very suggestive. The acute alimentary troubles of children, vomiting and diarrhœa, overshadow the other symptoms, and the temperature, while it may be high, is not continuous. They quickly respond to a mild purge and careful dieting. The possibility of worms must be kept in mind. Little need be said about the later stages of rickets. To anyone who has seen a few cases, the child with the deformed limbs, square head, pot belly and history of onset, forms a picture so complete that he could only with the greatest difficulty confound it with any other disease. It might be confounded with Hydrocephalus, but a little attention to the shape of the head soon serves to distinguish. Thus in the rickety head we have the squareness and overhanging of the forehead, a depression or groove in the temporal region, there is elongation of the antero-posterior diameter, and the occipital segment of the head is enlarged. In the hydrocephalic head the swelling is more of a uniform globular shape, the forehead being expanded and instead of a depression in the region of the temporal fossa we get bulging.

Prognosis. I have never seen a child die of rickets alone but in many cases I have felt that rickets was the real cause of death, Slight illnesses from which

healthy children usually recover cause great ravages amongst the subjects of rickets and one of the most prominent of these I have found to be Epidemic or Summer Diarrhœa. Rickets undermines the health of children and so renders them more susceptible to many diseases and increases the severity of all, and of pulmonary complaints and Diarrhœa in particular. In rickety children the size of the thoracic cavity is greatly diminished and so the expansion of the lungs is impaired and their expulsive powers diminished; hence the secretions are retained in the lungs, instead of being expectorated. It is said that the difficulty in expectorating is due to "impaired mobility of the chest wall" and to muscular weakness, but I quite fail to understand how anyone who has handled the chest of a rickety child, or watched the chest movements when a rickety child is suffering from dyspnœa, can take this view, but no doubt muscular weakness does play an important part. Many cases of Bronchitis pass on to Broncho-Pneumonia or prepare the way for the tubercule Bacillus. In the mining villages in the North of England the death rate from Pulmonary Phthisis is low, as far as actual cases amongst the miners themselves, and if the disease were not kept going by these children with weakened chests it might almost die out in the district. Indeed, Pulmonary Phthisis, either fibrous or tubercular, as a disease among coal miners, due to their occupation, is very uncommon,

in fact as a disease Phthisis is probably more uncommon among coal miners than among any other class of workmen in the community. The condition of the lungs found in miners called Anthracosis has been demonstrated now not to predispose to tubercule. The lungs of all coal miners would show post mortem a blackening due to inhalation of coal dust, powder, smoke, or soot from lamps etc., but this blackening is by no means due to a diseased condition of the lungs. I do not think that the presence of soot or coal dust in the lungs of miners has any influence whatever in the production of Phthisis, and where Phthisis, and especially Fibrous Phthisis, exists in conjunction with Anthracosis, it is due not to the inhalation of the coal dust but to the inhalation of dust from the stone or seggar-clay which accompany the coal strata. I have seen this demonstrated clinically in several cases during the last few years in this wise. I have known healthy miners from Northumberland in whom Anthracosis doubtless did exist, go to the gold mines at Johannesburg and after a year or two come home suffering from Fibroid Phthisis or Miner's Rot, as it is called, caused by inhaling the stone dust from the diamond drill as it bores through the quartz in the gold mine. Phthisis among colliers is the same as Phthisis in other people and arises from the same causes, with the exception above mentioned. There is another fact that in a

colliery district the death rate from Phthisis is greater among the females than the males, notwithstanding that there are more males. In this district, which is one of 20,000, there were last year 18 deaths from Phthisis, 8 males and 10 females. 5 of the males were colliers. This has no bearing on the question of Rickets, but I have gone into the question merely to show that tubercular disease is more common in the young members of the community and that in the adult pitmen Phthisis as a disease is rare.

All chest complaints and laryngitis cause great havoc amongst children with rickety chests, and infantile diarrhœa which is most common in hand fed children, and therefore amongst those suffering from rickets, carries away great numbers each warm summer. Convulsions are more common in rickety than in healthy children. Syphilitic subjects are very liable to a severe form of rickets and to the combination a large number succumb. I have seen four cases in which scurvy complicated rickets, all being due to the same cause. Three of the four died. While slight traces of rickets, usually in the head or legs, are common amongst the pitmen, the more obvious deformities have become less common. The fact is that in the survival of the fittest many of the subjects of rickets have disappeared before adult life is reached.

Treatment. This can only be scientific and accurate when we know the pathology of a disease. In the absence of definite knowledge it is often well to theorise in order that one may have a peg on which the treatment may hang until it receives some more definite support. I am convinced that the trouble in rickets is due in great extent to improper feeding, especially the use of proprietary foods. Whilst the composition of these foods leaves much to be desired, when they are compared with milk, which is the normal food for a child, yet it is unlikely that the continuous use of one of these foods, however deficient in any requisite constituent it may be, would suddenly set up an acute febrile disease. It is equally improbable that any foreign ingredient is the cause, as all the foods are at fault and most of them are prepared with the greatest care. It is more probable that the abnormal food, by its prolonged action on the alimentary tract, produces a condition favourable to toxins, probably bacterial in origin, and that the absorption of these toxins causes an acute febrile attack with its resulting symptoms, and that the disease tends to wear itself out in any individual and that one attack confers immunity. I have found this view a useful one to work with.

As Rickets is essentially a preventable disease, I think something might be said on its prevention. All women, if they are in fair health and are not subjects of constitutional disease,

such as Syphilis or Tubercule, ought to be shown how much it is to the advantage of the child to bring it up on the breast for at least the first nine months. If from any cause she is unable to suckle her child then stringent rules should be laid down for the regular and systematic feeding of the child with milk or food of a definite composition. If the mother's health is at all debilitated she should be given a full nitrogenous diet which increases both the Proteids and the fats of the milk. Very often I have noticed small and weakly children which were being suckled by debilitated mothers increase rapidly in weight when the milk was made richer by giving the mother a full nitrogenous diet, such as meat, eggs, cow's milk, and malt liquors or malt extract. The latter I have found to be of special benefit. Sometimes women are unable to nurse on account of fissured nipples associated with which I very often find abscess of the breast. The nipples should be attended to at once. A shield may be used for a few days and the nipples dressed with Boric lotion and simple ointment or Lanoline which I find very soon heals them. The digestion of the mother should be looked to. The diet, besides being rich in nitrogen, should be plain and nutritious. She should avoid all highly seasoned dishes and spices and also strong tea or coffee and instead of these may drink milk or thin gruel. Her nervous system should be kept

quiet and she should not be the subject of fatigue, strong excitement, worry or grief. An infant taking the breast milk under such circumstances may get an attack of acute indigestion, or may even get toxic symptoms with convulsions similar to Ptomaine poisoning. I can remember a child exhibiting these symptoms in a very marked degree in a case where it had had the breast while the mother was prostrate with grief on the hearing of a fatal accident to her husband. The infant was seized with sudden vomiting and diarrhœa with great collapse and convulsions, and it was only after very energetic treatment that it recovered. In no case should the child be brought up on the breast:-

I. When the mother is suffering from Syphilis or Tubercule.

The child would certainly acquire syphilis, whereas it might acquire tubercule, as I think that it might more readily get it from the mother by kissing etc. but as the tubercular ~~mother~~ mother is always weak and debilitated, nursing would do harm both to herself and to her child.

II. Where the mother had had any serious complication on childbed, such as excessive hæmorrhage, eclampsy, kidney disease, or puerperal fever.

III. Where the mother suffers from Chorea or Epilepsy.

Twice last year I attended mothers who had chorea as a complication to pregnancy but in both

cases the symptoms disappeared at the lying-in period and the children were both suckled with no disadvantage to the mother.

IV. Where the mother is the subject of heart disease.

These are the most important conditions in which the child should not be suckled but other less important conditions very often arise, such as extreme debility when the child does not improve on the breast after the mother has had special diet etc. Again cases sometimes arise when the mother seems entirely unfit, not from any debility but from no apparent cause, to bring up her child on the breast. In regard to drugs, I don't think there is any drug that has the direct action of increasing the secretion of the milk. Iron and the hypophosphites are useful, but probably act through their general tonic action.

If it has been determined to rear the child artificially, then cow's milk should be used, as it is the best substitute. It should be so treated as to lower its proteid percentage and to increase the percentage of fat. This can be done by diluting with water according to the age of the infant and by adding cream. The child should be fed at regular intervals, say every 2 hours to begin with, and with a definite amount of milk at a time. The proteid of milk is more difficult to digest than the proteid of human milk and in the stomach coagulates en masse while the proteid of human milk coagulates

in a flocculent manner. I have found that addition of lime water to the milk lessens this tendency to big coagulation in cow's milk. The feeding bottle should be simple and devoid of tube and should be kept scrupulously clean. The hygiene of the child should be considered. Absolute cleanliness and an abundance of fresh air. The child should get nothing but milk for the first nine months, when this may be supplemented by thin oatmeal gruel. After the first year milk should still be the basis of the diet, only as the child can digest more proteid it need not be diluted so much. In addition we can give oatmeal gruel or porridge and thin soup or beef juice. Towards the second year we can give soft boiled or poached eggs. From the 2nd to the 5th year it may have milk, soups, meat, a little potato and eggs, with bread and farinaceous food. It gets then practically the ordinary fare of the house. If these simple rules were carried out faithfully, Rickets as a disease would I am sure become rare.

The general malaise-temperature, sweating and enlarged spleen all point to a toxic condition and I have found the administration of antiseptics the most useful method of treating the disease in the acute stage. I have found the use of such intestinal antiseptics as Calomel, Salol, Liq. Hydrary. Perchlor., Liq. Thymol Co. and even Salicylate of Soda of very great value indeed.

When the diagnosis is made in the acute stage, the treatment should begin without delay. The

child should be put to bed at once and in dealing with children able to crawl or walk they must be kept in bed for at least six weeks, and heavy children for a longer period. Unless the child is exhausted a mercurial purge should be given. I make a point of seeing the resulting motion, as it may give valuable information as to any article of diet disagreeing with the child, and further it will be able to exclude the possibility of worms. Following this I usually prescribe some intestinal antiseptic to which some aromatic chalk or a little opium may be added, if there be diarrhœa. The dieting of the child must be reconsidered. At first milk alone should be given, diluted if necessary, not according to the child's age, but to meet his digestive powers. Again an examination of the stools will afford valuable evidence as to the amount of dilution. Very often in these cases if the milk is not diluted enough, the child passes undigested milk in its stools.

The feeding must be regular and not too frequent. The mother should be told how much milk is to be given and the hours of feeding should be specified. If anything is required between the hours of feeding, water alone or beef juice very much diluted with water or thin beef tea should be given. It is well to tell the mother this, as she is almost sure to give the child something else when the child is thirsty, and these children are always thirsty. I always use fresh cow's milk in preference to the

brands of humanised milk, for, though the composition of the latter is undoubtedly very near that of human milk, it is very difficult to alter its composition to suit special symptoms; further it is very expensive and I doubt if its use would prevent scurvy any more than the condensed milks. A study of the composition of cow's milk will enable one to produce a milk of almost any formula. The proteid element is the one that is likely to give trouble and the 4% in cow's milk must always be reduced for children under a year, as the digestion is usually disturbed. It is not advisable, however, to dilute below 2% or 1½% without first trying the effect of peptonising and pancreatising it. If this is not successful it is better to precipitate the proteid and give whey. In any case, however, one must not neglect to bring the cream up to about its normal percentage. In summer the milk must be sterilised. In beginning treatment with a child under one year I advise the mother to allow the milk to stand in a cool place until she is able to separate the cream, then to pour off half the milk and make up to original bulk with water and then to add the cream which has been removed. I emphasise the importance of adding the cream, as all the patent foods are deficient in fat, and, as they mostly contain an excess of Carbohydrate, I am less particular about adding sugar at first. The effect must be carefully watched. In these rare cases of the child developing rickets

before the ninth month, while it is still on the breast, if the mother is in good health, attention must be paid to the regularity of the feeding. The nitrogenous elements of the mother's diet must be increased. A liberal diet of milk, eggs and meat, and an abundance of fresh air. If the mother be debilitated then recourse may be had to general tonics, such as the Hypophosphites and Iron. If, under this treatment, the mother still remains debilitated and the milk poor the child had best be weaned. In cases due to too prolonged lactation the child must be weaned without delay. In these cases the mother is apt to have trouble with her breasts at the weaning of the child. In quite 50% of cases I have noticed a general mastitis which is no doubt caused by locked up secretion in a generally over stimulated gland.

For two or three weeks I give antiseptics by the mouth and if, after stopping them, I find that the febrile symptoms persist, I start the same treatment again.

Calomel in doses of $\frac{1}{4}$ gr. 3 or 4 times a day is a good drug to begin with. If there is a tendency to diarrhœa I then give Salicylate of Soda with aromatic chalk and Dovers powder or I sometimes give Salol and Dovers.

As the acute symptoms pass off I increase the diet. I continue the milk, but add other articles according to the age of the child. Such articles

very often take the form of white of egg, raw eggs, thin oatmeal gruel, the expressed juice of underdone meat or beef juice which can be obtained in the shops. It is very difficult to obtain a satisfactory beef juice, as regards its proteid constituent, which may either occur as proteid or be available as peptone. Some beef juices contain too great a percentage of water and too small a percentage of proteid and salts, and some are too much of a Multum in Parvo. I am very fond of ordering Carnricks Beef Peptonoids. It contains a high percentage of proteid and salts and is rich in peptones. It requires little digestion and is readily absorbed and is just the preparation to suit the weak rickety stomach. I give an analysis of some of the favourite varieties of beef preparations just to show their percentage composition. I copied it some time ago from an American chemical paper the name of which I cannot recall.

	Water	Organic Substances	Salts
1. Kemmericks Extract of Meat	20.95 P.C.	60.81 P.C.	18.24 P.C.
2. Liebigs Extract of Meat	19.33 "	57.32 "	23.25 "
3. Murdoch's Liquid Beef	83.61 "	15.83 "	0.56 "
4. Valentines Meat Juice	59.07 "	29.41 "	11.52 "
5. Johnston's Fluid Beef	49.49 "	45.32 "	5.19 "
6. Benger's Peptonised Beef Jelly	89.68 "	9.43 "	0.89 "
7. Brand & Co's Essence of Beef	89.19 "	9.50 "	1.31 "
8. Carnricks Beef Peptonoids	6.75 "	87.51 "	5.50 "

Fat in the form of cream, bacon fat or suet is very useful and it is surprising how well it is retained.

Cod liver oil and Parrish's syrup of chemical food are very valuable. During the past two years, however, I have ordered Virol instead of the Cod liver oil. It is a good preparation, very rich in fat and containing a fair proportion of organic Iron. It can be given to Infants in their milk and is well taken also by older children. The dose recommended, however, is too small and is just about half of what the dose should be. It is well to remember that it just ~~almost~~ contains about one half the fat that cod liver oil does.

In addition I order cool sponging daily and have the body gently massaged and rubbed thoroughly with a soft towel. I think there is some advantage in the use of sea water and especially water in which sea weed has been boiled. I find this cutaneous stimulation of much value and a very good tonic indeed for young children. After the first fortnight massage night and morning.

Too much importance cannot be laid on the benefit of sunshine in children recovering from the acute attack. I have noticed very rapid progress when these cases are warmly wrapped up and protected from the winds and laid out in the sunshine for an hour or two every day.

In following out this line of treatment I

think I can claim considerable success. In several cases where the child has been kept in bed and the above treatment carried out I have seen the disease cut short before the epiphyses enlarged, or they have only been enlarged slightly, and on getting the child up I find the bones show no tendency to bend in many cases. Too often my instructions as to rest and dieting are not fully carried out, though, as a rule, the drugs are administered with unfailing regularity. In many of these cases I am convinced that the treatment, though not fully successful, is of considerable value to many of the children, and in the treatment of a very large number of cases I have found the above method more successful than any other I have employed.

In cases complicated by scurvy the child should be put on the prepared cow's milk and in addition the juice of a lemon or orange should be given, disguised with a little syrup or gylcerine. Children with deformed chests should be taught as early as possible to exercise the chest and lungs. This can be done by gentle restrained movements or in younger children by giving them toys such as trumpets etc. In cases where the bones are bending the weight must be taken off them. In such cases I continue to use the treatment above described. In addition, if the bones are soft, the deformity can be remedied and kept straight by suitable apparatus. Up to about 4 years of age

the bones can usually be moulded into good shape, under the influence of an anæsthetic. When straightening the bones you may frequently hear a crack. After straightening it is best to put the legs up in splints and have them massaged daily. One can prevent the child from walking by putting a starch case round the ankle and fixing a strip of metal which projects down the side for a few inches beyond the sole of the foot. A heavy plaster of Paris case is bad as it prevents movement and causes atrophy of the muscles. The deformities of the arm if starting get well when the child begins to walk. If established they can be put straight and fixed up in suitable splints. In one case of Coxa Vara I tried prolonged extension, applied to the thighs, hoping that whilst my extension weights pulled downwards, the head of the femur would be fixed by being caught on the brim of the acetabulum and that the pull on the neck would bring it back to its natural angle with the shaft. My attempt was quite unsuccessful, due, I think, to the laxity of the ligaments, which is very common in these cases, preventing the head of the femur from being jammed in the acetabulum. After the age of six, as a rule the bones can be no longer bent and the child with bad deformities must be handed over to the Surgeon.

I have made a small appendix with a chart of a child suffering from acute Rickets & also with a few photographs of the most typical varieties of Rickety deformities I might have inserted many photographs, but as the deformities are mostly uniform in character I have thought fit only to insert those showing the deformities in any marked degree

DISEASE.

Notes of Case.

Name

Age 11 Months

Diet Cow's milk.

Case Book No.

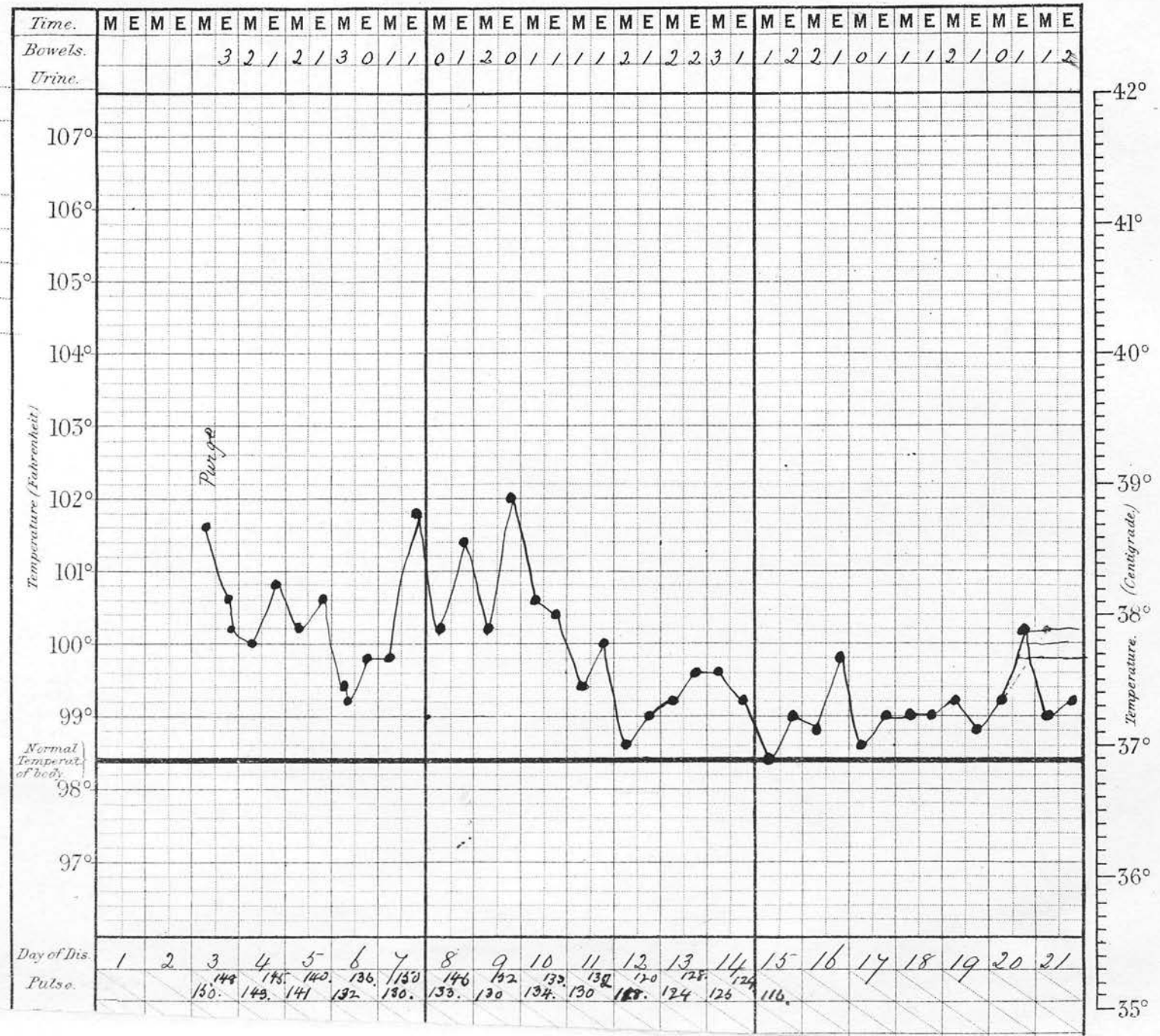
Treatment

Salol.

Aromatic chalk

Dovers powder.

Date of admission



Photograph of a case of Rickets showing the Thoracic deformity and enlarged abdomen

Photograph showing Thoracic deformity The small narrow thorax is well shown in great contrast to the enlarged abdomen The longitudinal grooving of the chest comes out very well & the photograph also shews the marked epiphyseal swelling in the region of the wrist joint.

Photographs of a child thirteen months old suffering from Rickets. The photographs are not quite a success, from the fact that the child was suffering from Bronchitis, & a good expression could not be got. However they are sufficiently good to show the large open anterior fontanelle with the frontal prominence.

Photograph showing extreme mobility of the joints in a Rickety subject.

Photographs showing extreme Rickety deformity. & also showing exaggerated mobility. This girl had also Coxa Vara