ORDINARY RICKETS

WITH SPECIAL REFERENCE TO ITS SYMPTOMS, AETIOLOGY AND TREATMENT

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JOHN FREDERIC HAEGERT,
M.B., Ch.B.

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I N D E X.

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PREFACE.

Rickets being such a common disease to which young children of many countries are prone, and the variety of tissues it affects, make it a subject of great importance and interest.

The causation of the condition, even at the present time, is still a problem that is not apparently clearly solved, as so many authorities on the subject hold widely different opinions.

Recent experiments seem to show that lack of sufficient exercise is an important factor in experimental rickets in animals. And this is of great interest in view of the generally accepted doctrine that rickets is due to dietetic and hygienic errors.

DEFINITION.

Rickets is a disease in which the nutrition of infants and young children is affected, of which the chief result is that the epiphyseal ends of bones are enlarged and the bones so soft that they yield to pressure of various kinds.

SHORT HISTORY OF RICKETS.

The name Rachitis we owe to Dr Francis Glisson of Cambridge. In his work on the subject he offers his readers this name, which is derived from the Greek equivalent of spine, on the ground that this part is the first affected. It is possible that he really adopted it on account of its similarity in sound to the word rickets, which was a popular name in the West of England where rickets seems to have been first recognised.

Rickets is derived from the old English word 'wrickken', to twist. But Comby thinks that rickets comes from the Normandy word 'riquets' and speaks of it as "riquets, épithète appliquée aux personnes bossues et difformes". Whistler has been spoken of as the discoverer of rickets, and to have written in 1684 a thesis which he called Paedosplanchnostercae.

But Dr Norman Moore asserts that there is no trace of this thesis, and that Whistler was not a man to be trusted in regard to his claim as the discoverer of rickets.

Glisson is generally acknowledged to have been the first to recognise and describe it. In Dutch articles on the subject Whistler is not even mentioned, and the discovery of rickets is attributed to Glisson.

Glisson's treatise on rickets was published in 1650 and he is known to have been working on the subject in 1645. The explanation he gave for the prevalence of rickets in his time, was that it was largely due to wealth, luxury and idleness. He included many diseases as the precedent causes.

In Holland the disease is known by the name Engelsche Ziekte and in Germany as the Englische Krankheit.

Indeed it was once thought that rickets spread from England to the Continent. But this idea was misrepresented. The fact is that in 1765 a Hollander, Dr Hendrik van Deventer, thought the origin of rickets was due to an Englishman who was the first to establish a coffee-house in the Hague and later at Leeuwarden in Friesland. By doing this he initiated the custom of coffee and tea drinking in this country. Van Deventer suggested that this

new custom was the prime cause of rickets being found in Holland.

Rickets existed in ancient times, although some of the apparently rickety skeletons have been shown to be really syphilitic. Zambaco found some rickety bones amongst those of the ancient necropolis at Cairo. Professor Lortet recently recorded some instances of apparently undoubted rickets in mummies of sacred baboons found in Thebes.

But G. Elliot Smith found no sign of the condition during his examinations of thousands of ancient human skeletons in Egypt.

Ebstein found twisted bones, which were like rickets, in old Grecian graves.

Soranus states that deformed and twisted legs were sometimes present in Roman town-children, and that in A.D.2 there were several victims of a disease like rickets.

Stiebel considered Aesop was rickety, but Ackermann, who wrote 70 years earlier than Stiebel, states that Aesop, Socrates and Crates had symptoms like rickets and that the disease may have been syphilitic in nature.

AETIOLOGY.

The causation of rickets is generally held to be dietetic in nature, but in reading the literature of it one is struck by the very diverse opinions held by many eminent authorities, opinions which, often, are apparently definitely proved by experiments: so that it must be said that the aetiological factor is not established to the satisfaction of competent authorities in general.

Some 30 years ago it was held that the deficiency of lime salts in Loch Katrine water was the cause of the prevalence of rickets in Glasgow (Sir James Barr). Such a theory was readily discounted by the fact that rickets is not confined to districts supplied by water deficient in lime. Friedleben has further shown that a diet in which lime is deficient will not cause rickets.

1. Diathesis.

Sir William Jenner maintained that rickets was of a diathetic nature, like tuberculosis. By diathesis he meant a certain proclivity to disease. He emphasised the fact that the first child, or even the first two or three children, are frequently free from rickets; and again that a woman who has once had a rachitic child, those that follow are

practically certain to suffer from rickets. However, this may be explained by the progressive enfeeblement of the mother's health by repeated childbearing, or, in the poorer classes, to deficiency in the amount of clothes and food which a large family entails.

Better class children are frequently kept too much in-doors.

Sir Samuel Wilks agreed with Jenner's theory, but with the proviso that diathesis means the state of health that induces rickets, i.e. that defective health during long periods leads to the proclivity to rickets.

He classes rickets with Scorbutus and Tuberculosis as a diet diathesis.

Jenner has pointed out that children of phthisical parents were actually less likely than those of non-phthisical to become rachitic.

Certainly, a child of a feeble mother is very apt to become rachitic if its diet and hygiene are faulty, but the causative factor cannot therefore be said to be diathetic.

In view of many cases of experimental rickets, produced independently of the question of diathesis, and in view of our present knowledge of the probable causes, the theory of Jenner is untenable.

2. Congenital origin.

Parker believed that rickets is invariably congenital. In support of his view he showed the Pathological Society of London a radius with epiphyseal enlargement and an ulna which was much curved. The bones were from a foetus which did not long survive its birth.

Elsässer regards rickets as being due to "congenital constitutional tendency to a slow development of the whole body, and especially of the osseous system."

Bednar, late physician to the Vienna Foundling Hospital, in his book describes typical rachitic symptoms and calls the disease congenital rickets.

Further, Kassowitz and others of the German School consider that rickets is congenital. They based their views on the macroscopic appearances alone, and not on the characteristic microscopical bony changes. Children commonly have soft bones, the abdomen may be protuberant, fontanelles unclosed, and there may be swelling of the costal cartilages, and yet it was on these grounds alone that such a theory was based.

Tschistowitsch and other later advocates of this theory did examine the bones microscopically, but it appears that much of their material was obtained from still-born children.

At that time the pathological anatomy of rickets and osteochondritis syphilitica was imperfectly understood, and therefore the two conditions were often confused: so that it is fair to assume that many of the specimens examined were in reality syphilitic in nature.

As further knowledge differentiated these two conditions, each succeeding report of work done on the subject showed a decreased proportion of congenital rickets. In 1882, Kassowitz considered that 80 per cent of all children born were rachitic; in 1897, Tschistowitsch gives the percentage as 12; whilst in 1902, Escher could only diagnose 1 case as congenital rickets, and that after clinically and histologically examining a large number of rachitic cases.

Escher's findings are strikingly in contrast to the opinions of Parker, Bednar, etc.

I have never seen a case of rickets that could in any way be considered congenital, and I find that many authorities on the subject do not believe there is such a condition as congenital rickets.

3. Heredity.

Most British observers doubt whether rickets is capable of direct hereditary transmission.

Vogel states that he knows many families in

which the parents had distinct signs of rickets, and that all their children became rachitic.

Ritter von Rittershain appears to have traced the cause of rickets to tuberculosis in the parents, more frequently in the father than in the mother.

Jenner doubted whether impairment of a father's health has any tendency to induce rickets, and quite contradicts the opinion of Ritter von Rittershain.

Siegert considers rickets to be hereditary, and dependent on some inherited weakness or predisposition.

Ormerod reports a case of a father who had rickets in childhood and mollities ossium in adult life, and his two daughters suffered severely from rickets. But it is not said whether the diet and hygiene of the children was faulty or not.

I have seen a case of rickets in the first child and not in the second, one of the parents having had rickets as a child. The case in question was in all probability due to faulty diet, as the child was brought up entirely on artificial food. Shortly after the food was altered to fresh milk etc. the child rapidly recovered.

Hausen records the case of a stallion who begot seven rachitic foals. Later two of the mares, who had given birth respectively to three and two rachitic foals, became pregnant to another stallion,

and bore healthy offspring.

The evidence in favour of the hereditary influence as a causative factor in human rickets seems to be slender. At the same time, heredity may play some part in the condition.

Parker considers that heredity is a very important factor, but I very much doubt this.

4. Lactic Acid.

Heitzman attributes rickets to lactic acid. He found lactic acid in the urine of rickety animals. By injecting this acid subcutaneously and also by feeding animals with it, he induced the condition in some. He supposes that the lactic acid is a result of the carbohydrate fermentation and irritates the ossifying tissues. Other observers have been unable to confirm this formation of lactic acid, and have failed to produce rachitic changes by its administration.

Senator considers that this acid acts as an irritant to growing bones.

5. Wegner, of Berlin, showed in 1871 that if lime salts are withheld from the food of young animals and minute doses of phosphorus administered, there resulted a condition exactly like rickets. He supposed that the phosphorus acted as a stimulant to

the ossifying tissues.

6. Bossi has shown that removal of one suprarenal gland from sheep caused osteoporosis, and he suggests that rickets may be explained by some defect of this nature.

In this connection it is interesting to note that Chalmers Watson examined the adrenals of twenty-five healthy wild rats and a similar number of rats from the same source but which were kept in the laboratory on a diet of bread and milk in proportions of proved value. He found that the percentage weight of the adrenals from the confined animals was considerably less than in the controls. His earlier experiments did not show a similar result, and Chalmers Watson concludes that the low percentage in the confined rats was due to want of sufficient muscular activity.

7. Mendel attributed rickets to perverted function of the thymus gland. But there is slender if any evidence of this being the case.

8. The Infective Theory of Rickets.

This theory is supported by Mercote, Edlessen, Torane, Salvatore Forte and others. Silvestri points out that the frequency of rickets in certain

streets, its geographical distribution, the analogy between the yearly curve of rickets, and that of certain infective diseases, the apparent sudden commencement of some cases and its prevalence in insanitary districts, all tend to suggest infection.

Kassowitz suggests that it may be due to the inhalation of foetid organic matter, especially the products of ammoniacal decomposition of urine.

Torane and Salvatore Forte claim to have produced rickets in rabbits by inoculating them with watery and alcoholic extracts of the faeces of children suffering from rickets and diarrhoea. It is singular that though rickets is said to follow on injection of either the alcoholic or watery extract, a mixture of the two is quite inactive. By confining a healthy animal with a rachitic one, Moussu induced rickets in the former: and then, without subsequent disinfection, he confined another healthy animal in the cage and it also became rachitic.

Further, this observer inoculated various kinds of animals with emulsions of rachitic bone marrow, but without any effect. By injecting what he calls a more virulent emulsion, and also confining the animals in the 'infected cage', he was able to induce rickets.

Arton and Agnese inoculated white rats with Diplococcus osteomalaciae and induced symptoms like

rickets.

Although there is certainly evidence that apparently supports the infective theory, it cannot be said to be proved, as in view of more recent research it has been shown that confinement of animals is a factor in the production of rickets in animals. And it can safely be presumed that the animals in the above experiments were all confined in cages.

Further, the interesting experiment of von Hausemann is a proof against this theory. Rickets is not found in Japan, but von Hausemann was able to induce the condition in a wild Japanese monkey, by simply keeping it in captivity.

9. Want of fresh air and sunshine.

It is generally admitted that these factors are of great importance in the treatment and causation of rickets. But Vogel asserts that the want of fresh air is the most important one.

J. M. Logan writes in precisely similar terms as Vogel. His contention is that rickets is most apt to result when the child does not get enough fresh air. It is rare in the country and common in large towns, and the difference in feeding is not great but the difference in the air is immense. It is said that rickets is common amongst the cottars on the West coast of Scotland, but very frequently

there these children do not get the fresh air, as they are sedulously kept in-doors.

Logan states that he has verified this theory in his experience in private practice and also in the Liverpool Children's Hospital.

While one is ready to admit that fresh air is of great importance in the causation of many cases of rickets, it cannot be the most important one. In proof of this I may say that I have recently treated a severe case of rickets in a child who was regularly taken out of doors, and during nearly the whole day the child lay in his cradle out of doors and breathed the purest air constantly. In this case the cause was unquestionably dietetic.

Von Hausemann supports this theory and draws attention to the fact that children born in winter are more liable to rickets than others, presumably because they are not able to be taken out soon to get fresh air, and when they do get it, they are so warmly wrapped up that active muscular movement is restricted.

He caught a wild Japanese monkey and kept it in captivity in Japan with the result that it developed rickets. This is a very striking experiment, as rickets is said to be unknown in Japan.

Clement Bilcher mentions the case of a colt that was probably rickety owing to improper diet and bad

air. When these were remedied and lime water and cream given in addition to its ordinary food, the rickety symptoms all disappeared. This does not in any way prove that fresh air is the important factor, but it certainly lends support to the general opinion that fresh air is of importance.

Von Hausemann's experiment does not afford sufficient proof of this theory, for certainly the animal did not get its accustomed food, and besides, it was deprived of its habitual movements such as climbing, etc.

10. William Ewart considers that rickets is largely a question of the Respiration. He bases his views mainly on his treatment of rickets by judicious recourse to artificial means for restoring what he calls the partly-suppressed respiratory function. For some years he has treated rickets by the prone posture and the use of a special elastic abdominal belt, etc.

But his reasons appear quite insufficient, as other treatment, such as simple cod oil and attention to hygiene produce remarkably rapid cures.

11. Closely allied to the last theory is the one held by Dr Robert Lee, namely, that in England "the "cause of rickets in 98 per cent. of the cases in

"which it occurs is to be traced to those common "forms of lung inflammation which prevail so marked-"ly in our climate."

His contention is that in every case of rickets there is some pulmonary inflammation such as catarrh, etc., and that this leads to imperfect oxygenation of the tissues of the body and thus leads to rickets.

It appears that in his hospital practice over 3000 cases were under his care, and that they practically all showed signs of pulmonary inflammation. He published a list of 50 cases in all of which cough or bronchitis was present, and in some there was broncho-pneumonia.

He lays stress on the intercostal retrocession which is to be seen after the subsidence of the acute pulmonary conditions, this retrocession being due to incomplete pulmonary expansion.

Still states that changes of the thoracic wall almost constantly lead to pulmonary catarrh and atelectasis, but this would seem to show that the pulmonary condition is secondary to rickets and not the causative factor. And this is the view that is generally accepted.

It will be readily conceded that a non-rickety child weakened by pulmonary trouble would readily become rachitic, if exposed to dietetic and hygienic errors.

A strong proof against this theory is that rickets is effectively treated by cod oil (and attention to hygiene when possible) amongst the out-patients of various children's hospitals, and that generally without any change whatever in the hygienic or respiratory conditions.

If this theory be correct, one wonders why the many children one has treated for pulmonary conditions do not become rickety. While practising for a year as assistant in a country town in Yorkshire, I had to treat a great number of children for bronchitis, broncho-pneumonia, etc., but during the whole period I did not see a single rickety child. The children were mostly breast fed.

It would seem that certain districts are much less afflicted with rickets.

12. Climatic Influence.

Rickets is said to be a disease of the temperate zone, and is rarely if ever found in tropical and sub-tropical countries. But it can be shown that this is not the case, as the disease is quite common in Egypt at the present day, though it appears to have been excessively rare there in ancient times. Professor Lortet records instances of apparently undoubted rickets in mummies of sacred baboons found at Thebes.

It has more than once been asserted that Italy is free from rickets, but enquiry into the causes of death in that country shows that, far from being free, it is nearly three times as prevalent there as in England.

Professor Ruata of Perugia, Italy, has studied the question of the incidence of rickets in the various Italian provinces, and gives interesting statistics on the subject. He found that certain provinces, which contain large towns, had a relatively low death rate from rickets, whereas provinces with small towns and villages had a high mortality. So that overcrowding in these cases does not seem of great importance.

A damp climate such as pertains in other provinces similarly shows a much lower mortality than those which are very hilly and dry.

But in Holland, which in general is essentially a damp climate, owing to the immense number of canals, rickets abounds. This fact is very noticeable in travelling here, after being in other countries in Europe and South America. It is quite common here in Holland to see children with severely curved legs, and adults with spinal deformities, both conditions being probably rachitic in nature.

Dutch writers on the subject also state that rickets is very prevalent in Holland. One writer

says that there is more rickets in Amsterdam than any other town, and that in that town alone 1000 cases are treated every year. I have often wondered whether the fact that the houses are nearly all built on very damp soil has any important effect in causing rickets. When the foundations of the houses are being made on long poles about ten feet in length, the foundations appear to be literally in a swamp. Much of the water is pumped out and the space filled up with sand, but naturally dampness will remain. Owing to the flatness of the land in many parts, a proper sewage system is not made in some towns. The watercloset is a mere hole which discharges itself down into a branch of the canal that happens to be nearest. On asking an engineer why these old closets were still retained, he explained that there is no fall to the sea, the land being under sea level, and that the sandy soil is so loose that drain pipes of any size would very soon be put out of repair by the sinking of the sand.

I mention these facts because Senator and others point out that rickets is more prevalent in cold and damp climates.

Reference has already been made to the statement that rickets is seldom if ever seen in the tropics. It is said by Dr Ewart, Professor of Pathology at Calcutta, and several other doctors of

considerable Indian experience, that rickets is exceedingly rare in India. And they mainly attribute this to the universal custom of suckling the children, and to the children being practically constantly in the fresh air. Personally I have my doubts whether many of the native children really do get fresh air in the poorer parts of the town. In 1903 I made a tour through India, and in Calcutta, in a part of it called Chowringee, one was usually conscious of a general foul and unhealthy odour. But in the smaller towns and country the air is quite good.

Dr Evézard states that in the Madras Presidency and Burmah, during a long experience, he only saw two or three cases of rickets: and these occurred in emigrants to those parts, and during the damp season. These children had been brought up on rice or arrowroot. The native population was almost entirely free of the disease, and European children seldom, if ever, contract it.

During my stay in various parts of India I never saw a rickety child.

Rickets is said to be much less prevalent in North America than in Britain. When it does occur it affects chiefly the children of the poorer population, who are badly housed, and many of them are

Italians.

During 1906-1911 in my practice in Southern Chile and Argentine, I only saw one case of rickets, and that was due certainly to dietetic causes, as no fresh food was available. This part of South America is exceedingly healthy. Children are very frequently brought up on tinned milk only, and later on are given mutton soup, etc. The air there is remarkably pure.

Some of the districts are very damp, and there is a tribe of primitive people called Canal Indians who inhabit large portions of the coast where the rain-fall is said to be one of the greatest in the world. I have seen many of these Indians and have not found any signs of rickets amongst them. They live constantly in the fresh air and are going about in the bitterly cold weather in the scantiest clothing. I have noticed that as soon as these Indians become a little civilised and wear clothes, they become tubercular, and rapidly die of phthisis. In this way I have known a sturdy family of them die within a couple of years.

Amongst the Pampa Indians of this part, Rickets is unknown, as far as I could ascertain. I have seen many of them, and examined the skeletons of others, and never came across any sign of rickets.

Two horses that I frequently saw in Chile

interested me considerably, as they had severely curved spines. I attributed this to the probability of their having been foaled late in the season, and as good grass is scarce in winter, the mothers very likely had little or no milk for them. They appeared to have suffered from a disease like rickets.

Bartlett, who was superintendent of Regent's Park Gardens, attributed the common occurrence of softened and deformed bones amongst the young lions to the clay soil and cold locality. Many of these animals died, whilst those in menageries, where the animals are moved about to different places, a great many lions reached maturity.

It is difficult to explain the incidence of rickets in Italy, but at the same time I think that certain climates are not productive of rickets. Where there is abundance of fresh air and sunshine, as a rule, the children are not rickety. Damp cold climates appear to foster the disease, though there are exceptions to this, as has been pointed out.

13. Lack of sufficient exercise.

As recently as 1908, Dr Leonard Findlay has experimentally produced rickets in puppies by considerable restriction in a cage. Control puppies

were similarly treated as regards diet and hygiene, but were allowed to run about freely. By thus restricting the exercise of puppies he has invariably produced rickets, although the food, as he states, was beyond suspicion, the air pure and the kennels kept scrupulously clean.

The instance already quoted of the condition being found in the sacred baboons of Thebes lends some support to this theory, but the captivity in which the animals were kept would also deprive them of fresh air and their natural food.

And the same may be said of the experiment of von Hausemann with the Japanese monkey.

Chalmers Watson suggests that the porridge and milk diet of the puppies, in Findlay's experiments, was not a physiological one; and he has previously shown, by experiments on young animals, that such a diet, if there is a greater proportion of oatmeal diet than as used in Findlay's experiments, will cause interference with the growth and later, marked disturbances in the health of the animals. He agrees with Findlay that confinement and lack of exercise, under certain dietetic conditions, modify the osseous system and produce rickety conditions.

Lack of exercise seems to play an important part in the production of rickety symptoms in animals, but anyone who knows much about animals will

agree that this restriction of exercise means vastly more to animals than to children. Many young animals are from birth able to run after their mothers, this being a provision of Nature for their safety to escape danger.

In Southern Chile amongst the poorer classes the children are invariably bound up with their arms tight to the sides, and the legs straight out. They make a very neat rigid bundle of the child. Their idea in doing this is to make the children grow up straight. They consider loose clothing for a child akin to negligence. And in spite of this lack of exercise, I have never seen a case of rickets amongst them.

A few days ago a friend, who is interested in breeding dogs, related to me the following. He has found that puppies, if kept in a small space, become usually rachitic. He has shown these puppies to a veterinary surgeon who at once diagnosed the condition as "Engelsche Ziekte", and prescribed exercise, and lime salts to be added to the diet. My friend has often noticed that the puppy does not become rachitic if he has another as play-mate.

14. Syphilis has been held by Marfan & Parrot to be the chief cause of rickets. But this has been shown, by experiments and clinical observation, not

to be the case. Certainly a large proportion of the children with rickets whom Parrot treated were syphilitic, whilst others were both syphilitic and rachitic.

Marfan states that rickets can be the effect of hereditary syphilis, but that the authorities who hold this view have not considered it to be the only cause.

Parrot considered rickets to be entirely due to hereditary syphilis, and states that signs of hereditary syphilis are found in 90 per cent of rickety infants.

Marfan agrees broadly with Fournier that hereditary syphilis, like all hereditary or acquired taints, predisposes to rickets, and adds that on occasions syphilis can cause rickets.

It is quite probable that syphilis may act as a predisposing cause, but it is generally accepted that it is by no means the prime factor.

It is a common occurrence to see cases of rickets in which there is not a trace of syphilis in either the children or the parents.

15. Dietetic.

An error in the diet of the child is generally held to be the chief factor in the production of

rickets.

(a) Guérin induced rickets in puppies by feeding them on flesh. But Findlay has been unable to induce rickets by various diets. I can find no confirmation of Guérin's experiments. On the contrary, I have frequently seen puppies brought up on meat alone, without any rachitic symptoms.

(b) Deficiency of lime salts in the diet has been, and is still, considered by several observers to be the chief causative factor in rickets.

Crisp records cases of pheasants, a young ostrich and a great number of young lions who suffered from soft and deformed bones. He attributed this mainly to deficiency of calcium phosphate in the bones, caused by improper diet and lack of fresh air. He appears to consider defective lime metabolism as of great importance.

Chossat produced curvature of the bones in animals by excluding earthy salts from the diet.

Reimers & Boye record bony changes in dogs fed on a diet poor in lime, but they consider the alterations in the bones very different to rickets.

Voit also states that he produced rickets in animals by depriving them of calcium.

Katz saw typical rickets in a fowl which was kept in small cage for some months, and he attributed the condition to the bird getting insufficient

lime.

I have known a Dutch woman treat her rickety child with powdered egg-shell, and the child got better.

But it is generally stated that calcium is of so slight value in treating rickets as to be scarcely worth while giving it.

It is improbable that want of lime in the diet is the factor of any consequence, as farinaceous foods and cow's milk contain much of that substance, and it is on this latter form of diet that rickets is supposed to flourish.

During rickets the lime absorption is certainly deficient.

Friedleben has shown that a diet deficient in phosphoric acid and lime salts cannot produce rickets.

(c) The influence of suckling. Rickets has been attributed to absence of suckling, to suckling at too frequent intervals or not long enough, and to prolonged lactation. No doubt if a child is suckled on milk that is inefficient in its constituents, the child will be weakly and apt to have gastro-enteric trouble and so lead to rickets. And the same may be said of the irregular feeding at the breast.

The fact that a child is starved by getting poor

milk from the mother and is therefore rickety, is contrary to clinical experience. Cheadle says that rickets may ensue on starvation, but this cannot be accepted. A child suckled on good milk is rarely rachitic.

Siegert found 81 per cent out of 845 artificially fed children had rickets, and only 31⅓ per cent amongst those who were suckled for at least 4½ months.

(d) Deficiency of fat in the diet. Bland-sutton considers that the deficiency of fat is the cause of rickets. His experiments, and the successful treatment of rickets with cod liver oil, greatly support this view. By simply adding fat and cod liver oil to the diet of lion cubs he was able to rear them, this being the first time that lions had been reared in the London Zoological Gardens during ten years.

Cheadle & Holt attribute rickets to deficiency of fat and proteid. And Ashby supports this view, but adds that probably the fermentation of carbohydrates may produce toxins, which cause some of the symptoms of rickets.

(e) Over-feeding. Crisp found that many cases of rickets were over-fed, but he does not consider this the important factor, so much as the quality of

the food.

Esser in 1907 affirms that rickets is due to over-feeding, which causes chronic gastro-enteritis and results in deficient absorption of food constituents. He bases his theory on the similarity of the blood in rickets and chronic over-feeding. But this starvation by over-feeding has been proved unable to produce rickets by Baxter, and is contrary to clinical experience.

(f) Starch as a causative factor in rickets. The time of weaning a child is often the start of rickets, as the natural milk is frequently replaced by starchy artificial foods. And a common error in the diet is that the child is given more starchy food than he is able to digest.

Dr Buchanan Baxter has tabulated 120 consecutive cases of rickets and found that 92 per cent of them had been given farinaceous food before the age of 12 months, and further, that in many of them the disease dated from about the time when this food was first given. Dr Baxter records some experiments he made with puppies, kittens, rabbits, guinea-pigs and white mice. Shortly after birth they were given a diet of pure arrowroot jelly with some milk. Soon the starch was passed through the intestines unchanged, as the digestive juices were inefficient in

amylolytic power. Invariably inanition resulted. A smaller proportion of starch proved less quickly fatal than a larger amount. The bones were thinner and more frail than in healthy animals, but they showed no rachitic changes. Baxter attributed the failure to produce rickets to the difficulty he had in keeping the animals alive.

He states that an infant fed on starch alone would die of starvation and show no rachitic changes.

I have a recollection that Prof. Greenfield stated in his lectures that he had induced rickets in animals by feeding them on starch. Unfortunately my Pathology notes are abroad and I cannot verify this.

Both Ashby & Heitzmann consider that the fermentation of the carbohydrates is of great importance in the aetiology. The former suggests that toxins are thus produced, while the latter asserts that lactic acid results.

Baxter says that no decomposition of the starch takes place, as it is passed unaltered and stained with bile. He states that infants and young animals are incapable of converting starch into sugar, by physiological experiment.

It is suggested by him that starch may indirectly be the causative factor by interfering with the assimilation of the fat.

Personally I agree with those who state that there is decomposition in the intestines. The foul smelling faeces and the frequently flatulent abdomen are sufficient proofs.

Starchy foods certainly seem to play an important part in the causation of rickets, as one has almost constantly noticed that the patient has been given this kind of food.

MORBID ANATOMY.

In a typical case of rickets the long bones are felt to the softened, their epiphyseal ends are much enlarged and various deformities of the shafts are evident.

The softness of the bones is due to delayed ossification and especially to the smaller proportion of earthy salts. The deficiency of lime salts is not so much due to their removal after deposition, as to the slow way in which they are deposited.

Analysis of rickety bones shows them to contain 33-35 per cent earthy salts, instead of 63-65 as in health, and the animal matter is said to yield no gelatin on boiling. Besides being softer, the bones are specifically lighter than normal and contain an undue proportion of fatty matter. The cartilage

cells contain a high per centage of water.

The periosteum is generally greatly thickened and is very vascular. The superficial layer can be readily stripped off, but the deeper one is firmly fixed to the underlying bone and can only be stripped off in fragments. Its connective tissue cells rapidly proliferate and become bone corpuscles. Calcification is irregular, so that layers of firm bony tissue are interspersed with others of a fibrous matrix which shows connective tissue or bone corpuscles and medullary spaces. The flat bones, especially of the cranium, show porosity and irregular calcification, the porous portions being chiefly on the surface and edges. Small portions of the parietals, and occipital less frequently, are thinned and transparent owing to deficient calcification and thinning of the parts by pressure. The causes of this craniotabes is fully discussed with the symptoms.

The long bones are seen to be retarded in growth and deformed in various ways. Ossification of parts that are still cartilaginous is interfered with, and bone that is already ossified is rendered soft.

A section longitudinally through a typical rachitic bone shows the following. The cut surface

appears generally reddened from intense congestion, except the parts of the bone next the epiphysis which are paler or yellower than normal. The epiphysis is greatly enlarged, due to an enormous development of the pearly-grey or bluish translucent cartilage, which varies in thickness with the severity of the disease. The layer of cartilage into which the new bone is growing is called the "zone of calcification": that next to it in which the cellular elements are arranged in vertical columns in preparation for the deposit of lime salts, is called the "zone of proliferation". These two zones are greatly thickened and are not distinct from each other as would be in a healthy bone. In rickets the epiphysis shows new bony tissue shooting up irregularly into the zone of calcification, and so gives rise to scattered points of bone with islets of cartilage, calcareous deposits and vascularity, some of these cartilage cells being quite surrounded by bone.

The medullary spaces are formed in unusual places and are even seen in the proliferating zone much beyond the margin of ossification.

In rickets the lime salts are first deposited in the cartilage cells. Very often the cartilage cells are converted into bone. At the same time scattered points of lime are seen in the matrix and

give rise to a dotted appearance in the section of the cartilage.

Similar alterations take place at the surface of the diaphysis.

Under the microscope the cartilage cells are seen to be very active. They are swollen and becoming calcified. Instead of making good bone, the interstices between them become filled with a vascular marrow instead of with natural bone. These medullary spaces are continuous with the channels in the diaphysis. Thus a spongy and very vascular tissue is formed which contains very little bone. Under the periosteum a similar process takes place. So that the shaft consists mainly of spongy lamellae which are only partially ossified.

By absorption the normal enlargement of the medullary canal proceeds, and thus the proportion of true bone with its proper amount of lime salts is constantly lessening. Therefore the bone becomes yielding and prone to distortion.

Strelzoff points out that an additional cause of the yielding is that the osseous trabeculae, instead of being concentric, are radially arranged. Thus external pressure readily distorts such bones.

In the <u>thorax</u> the beading of the ribs and various distortions of the framework may be noted. The

beading of the ribs is more pronounced on the pleural aspect because of the in-drawn condition of the yielding costo-cartilages. The softened ribs and the resulting deformities give rise frequently to Emphysema and atelectasis. In fact, where there is distortion of the thorax these two conditions are always present. The emphysema is chiefly at the anterior pulmonary borders and extends posteriorly for about three quarters of an inch from the margins. Just beyond this emphysematous portion is a line of collapsed lung which separates it from the healthy lung beyond. Each inspiration causes the enlarged rib ends to project further in, and thus compress the subjacent lung tissue, and therefore obviate proper expansion by the incoming air.

The thorax is narrower laterally and the antero-posterior diameter is much increased by the protrusion of the sternum. Naturally, then, the lungs, at their anterior borders, just behind the sternum, become emphysematous to fill up the resulting space.

Owing to the bending inwards of the ribs the angle between them and the vertebrae is more acute, and therefore patches of atelectasis may be found posteriorly. Pulmonary catarrh and plugging of air tube with mucus is another cause of scattered patches of collapse.

The enlarged rib epiphyses further cause circum-

scribed opaque patches on the visceral pericardium and on the peritoneum of the spleen, that on the pericardium being over the left ventricle a little above the apex. At this point the cardiac apex comes in contact with the enlarged end of the fifth rib. That on the spleen is due to rubbing against a similar projection during the rise and fall of the diaphragm. In both cases the patches are limited to the fibrous layer.

With reference to the difference pathologically between rickets and osteo-malacia, in the former the ossification is incomplete and much new material is formed which is imperfectly calcified; whereas in osteo-malacia the softening is the result of removal of the lime salts from perfectly formed bone. Both conditions closely resemble each other in their bone alterations, and it is important pathologically to differentiate them by carefully observing the above distinction, and by other characteristic rickety changes.

The lymphatic glands are frequently slightly enlarged or indurated. They give rise to a "shotty" feeling under the skin. They are found to be larger in the mesenteric glands. The enlargement is due to increase of the fibrous tissue of the glands. They do not caseate like tubercular glands.

The spleen is not always enlarged; in fact, splenic enlargement cannot be said to be a common feature of rickets, though at one time it was held to be of very frequent occurrence by Sir W. Jenner and Dr Dickinson.

In view of recent observations, it appears that any degree of enlargement is rarely, if ever, found in uncomplicated rickets. Starck has shown that splenic enlargement is just as frequent in non-rachitic as in rachitic children.

Eustace Smith states that it is only seen in exceptional cases of rickets.

Dr Fagge has found a large fleshy spleen in many non-rachitic children, and exceedingly seldom did he note any splenic enlargement in rickets.

Howard quotes a case in which the spleen was palpable.

Dickinson considered splenic enlargement to be frequent, and found that the rickety spleen had only ·09 per cent of lime salts as compared to ·27, that of a healthy one. He found that the fibrous tissue was increased greatly, and that the Malpighian bodies did not show waxy degeneration. He concludes that splenic and other visceral enlargements are as much an essential part of rickets as the bony changes. At the same time he observes that where there is much splenic or visceral change, the osseous changes are

seldom marked.

One naturally presumes that many of these so-called rickety spleen were really not due to rickets. Dr Goodhart found only 54 cases of enlarged spleen in about 900 rachitic patients. Of these, 16 were certainly due to rickets, 7 being doubtful and the remainder were due to congenital syphilis, leucaemia, tuberculosis and other unknown causes.

He agrees with Gee and Fagge that when there is splenic enlargement it is due to a cause which antedates it and the bony changes. Gee points out that enlarged spleen during the early months of life strongly suggests syphilis.

There is ample evidence that splenic enlargement does occur in rickets. It has been found post-mortem, and even during life cases have been recorded where the spleen reached below the umbilicus and occupied a third of the abdomen. Children who have this swelling of the spleen during rickets are markedly pallid.

Under the microscope Goodhart found that the enlarged rickety spleen showed few Malpighian bodies and a lack of contrast between them and the pulp, the fibrous tissue of the septa and stroma showing hyaline thickening. Both Goodhart and Dickinson consider the change one of interstitial splenitis.

Sir William Jenner describes thin sections of

the rickety spleen as being glue-like, owing to a peculiar morbid deposit termed albuminoid.

Goodhart points out that this glue-like appearance is due to the increased hyaline fibrous tissue which is also found in chronic enlargement of the spleen from ague and other diseases, and not to some particular product of rickets.

When there is an enlarged spleen in rickets, it is firm to the touch and can be readily cut into thin slices. The surface is deep red or purple in colour with smooth white spots, which are the enlarged Malpighian bodies. The section of it is deep red and mottled, with a pale buff colour, and but little blood can be squeezed out of it.

The liver is seldom enlarged. If there be much intestinal catarrh or other digestive trouble, the liver may become swollen from fatty infiltration.

Severe pulmonary catarrh, by impeding the respiration, may enlarge the liver from chronic venous congestion.

There have been recorded cases of fatty degeneration of the liver from phosphorus poisoning.

The muscles are flabby, pale and small. On microscopical examination they are found to have indistinctly marked striae.

The urine is smaller in quantity during the

period of profuse sweating. It contains a large proportion of calcium phosphate and Lehmann has observed that there may be even six times the normal amount of lime in the urine.

Heitzmann and other observers have found lactic acid in the urine of rickety animals.

Generally the urine is pale in colour and may often contain a deposit of calcium oxalate crystals.

When there has been much acid produced during fermentation, the urine often contains a large deposit of uric acid.

Chronic hydrocephalus, cerebritis and albuminoid disease of the brain are such rare complications that a description of them is hardly required.

The Blood.

Stoelzner states that there is no diminution of the alkalinity of the blood in rickets.

Sir James Barr has kindly informed me that he considers the calcium index of the blood to be diminished, but that no systematic observations have been made on this point in cases of rickets.

Haemoglobin. Most observers have noted that the red corpuscles are deficient in colouring matter. Morse in 1897 examined the blood of 20 uncomplicated

cases of rickets, and found the haemoglobin per cent to vary between 48 and 77. Felsenthal examined 12 cases, in all of which the haemoglobin was diminished.

Findlay has recently examined the blood in 30 uncomplicated cases, controlled by 12 healthy children of the same class of society. He found the haemoglobin slightly diminished, by 5 per cent, in one or two instances. In six it was normal, whilst others were above normal. In all the cases of rickets the average percentage was above the average of the healthy children, and five of the former showed marked increase of haemoglobin, while nine were slightly increased.

Red corpuscles. These are generally found to be paler than normal, variable in size and may show poikylocytosis occasionally. Nucleated reds were found by Morse in three cases of rickets which had splenic enlargement. In one of them the spleen was greatly enlarged and contained nucleated red corpuscles and numerous megaloblasts and normoblasts.

Felsenthal only found nucleated reds in very severe cases. Both Morse and Felsenthal found the number of red corpuscles not diminished or very slightly.

Ashby, Cheadle and Holt in writing on rickets

state that there is always more or less severe anaemia, while Cabot and Ewing consider the degree of the anaemia to be proportionate to the rachitic manifestations. The former says that anaemia is always present and that normoblasts, myelocytes and examples of polychromatophilia are frequently observable.

Pfaundler and Schlossman consider that there is nothing characteristic in the blood during rickets.

Hutchison states that even severe rickets is frequently without any appreciable degree of anaemia.

Still writes that the anaemia is specially marked when there is much splenic enlargement, but Findlay makes an opposite statement, namely, that the size of the spleen bears no definite relationship to the severity of the anaemia.

In the 30 rachitic cases that Findlay examined only 8 showed slight diminution of red corpuscles, and 7 an increase. But a striking feature of the blood examinations is that the red count was higher than in the normal children, and that this is contrary to what has generally been thought.

The Leucocytes. Eustace Smith states that there is a definite leucocytosis, even in rickety children who are not obviously anaemic.

Esser, in 1907, asserted that this is almost always the case, especially in the early stages of

rickets: that the increase amounts even to 30,000 per c.mm., and that the nuclei of the polymorphs were more lobulated.

Findlay found marked leucocytosis in 5 cases, and slight in 13; so that 12 of his patients did not show any increase of leucocytes, indeed 9 of the 12 showed diminution and 3 were normal.

But it must be noted that Findlay's patients varied in age between 12 and 42 months, so that the contention of Esser and others may still be correct, as the early stages are often prior to these periods.

I think that the leucocytosis which is found early in rickets is probably due to either gastro-intestinal irritation or pulmonary catarrh, or both.

Naegeli describes a mono-nuclear cell with meta-chromatic granulations, and considered it to be in nature lymphocytic. It is difficult to say if this cell is hyaline, myelocytic or lymphocytic. These cells are of variable sizes and have a circular or oval nucleus which is often slightly indentated. The nucleus stains more deeply with basic dyes than does the typical lymphocyte. The protoplasm varies in amount and density and contains a variable and usually small amount of faintly staining metachromatic granules.

Some of these cells are markedly basiphile at one part near the circumference like lymphocytes, and

at other parts are hyaline in character.

Findlay found a high proportion of these cells in the rickety children, but is doubtful of their significance and value as a help in the differential diagnosis of rickets. In his most interesting paper on the condition of the blood in experimental rickets I do not see any mention of these mononuclear metachromatic cells.

He produced rickets in eight pups by confinement in a restricted space. The blood showed the following:- in four of the animals there was a steady rise in the amount of haemoglobin and in the number of red cells, in spite of the development of marked rickets. One showed slight diminution in the number of red cells at the time of the last observation. In another the haemoglobin and red cells remained constant. Only two pups developed slight anaemia. Nucleated red cells were seldom found after the onset of rickets, and all the pups showed some variation in the size of the red cells. Leucocytosis occurred in two of the rickety pups, but both could be explained by the post-mortem findings of broncho-pneumonia. In three of the pups the lymphocytes were relatively increased, but in the other five, the polymorphs were more abundant.

Findlay concludes that in experimental rickets there is no marked pathological blood change and absolutely nothing characteristic.

SYMPTOMS OF RICKETS.

In rickets the symptoms are so many and so varied that it is convenient to describe them under two headings, viz., early and later. But at the same time it is necessary to bear in mind that the symptoms do not always keep to any definite order. Usually digestive trouble initiates the illness, but various complications ensuing, naturally the course of the disease is altered in diverse ways.

1. Early Symptoms.

One of the first conditions that occur affects the digestion. The child has a weakened digestion, he frequently passes large sour or foul smelling motions, and may be noticed to have a sour smell generally, this last being due to the condition of the motions, but I think it is also caused by the acid eructations that the child suffers from. The patient does not usually at first have either diarrhoea or attacks of vomiting, but still the motions are, as a rule, more frequent and larger in quantity than usual.

Much farinaceous and curdy material, often fermented, is to be seen in the motions.

The patient, previously bright, is now becoming more and more fretful and takes much less interest

in his toys.

At night he is probably sweating profusely, and this is especially seen on the head and neck, the pillow being even wet. The child is most restless in bed, pushing off his bed-clothes during sleep, and sleeping in all kinds of postures.

The abdomen is often seen to be swollen from flatulent distension and this makes the child cry. For this reason the child may be seen often lying with his head burrowed into the pillow, supporting himself on his knees and elbows, or he may be resting his weight on his chest and knees.

The excessive sweating frequently causes a rash, miliaria, to break out about the head and neck. This miliaria is best seen about the forehead and behind the ears.

Another effect of the profuse perspiration is in the diminution of the amount of urine, which now becomes acid and irritating when voided. It also contains much uric acid sand. Some observers, e.g. Heitzmann, have even found lactic acid in the urine.

Should the digestive symptoms not improve, a previously plump and rosy child may become pale, flabby and listless.

The superficial veins, especially of the face and chest, are usually too well marked. The veins at the base of the nose and temporal regions and

jugulars are seen to be full and visible.

The hair is at times very scanty and thin. The occiput is seen to be bare of hair, as it is rubbed off by the constant oscillatory side to side movement of the head. This movement is probably due to uneasiness of the head or ears, which may even be painful. The bareness of the occiput is a very characteristic symptom.

The child begins to dislike being danced about in the nurse's arms and resents anything like rough handling. This is probably due to the general tenderness of the limbs and body from which it suffers. At the same time, Eustace Smith considers that this tenderness, if marked, usually indicates scurvy; and one's personal experience coincides with this opinion.

Most observers consider general tenderness an early symptom of rickets, but Eustace Smith says that it is rarely seen in uncomplicated cases. He says that the child objects to being danced about, not because of the tenderness, but because the violent movement hurries the respiration, causing increased demand for air, which the yielding ribs cannot satisfy. Personally I think that the general tenderness, which certainly frequently obtains in rickets, is probably the cause of this uneasiness.

The child appears to dislike warm clothing even

in cold weather. This is commonly a cause of the child catching cold. The bowels are kept loose by such repeated chills, and the child is very prone to develop pulmonary catarrh. Indeed, very frequently he has a good deal of cough, so much so that one observer at least, viz. Dr Robert Lee of Pwllheli, maintains that every case of rickets has some form of pulmonary catarrh and claims that this is the causative factor of rickets.

Alterations in the bones vary a good deal.

Baginsky explains this variability by pointing out that the parts affected are the ones which are most actively growing at the time of the onset of the disease.

In some children the limbs are distorted and the chest unaffected, while others have a deformed chest and straight limbs: or both conditions may obtain.

Some cases show marked swelling at the epiphyses, whilst others have more malacia.

Usually the epiphyseal ends of long bones are enlarged, flat bones are either thickened or thinned in parts, and there is a general softening of all.

The epiphyseal swellings are at the point of junction of the epiphysis and diaphysis. The wrists and chondro-sternal joints especially show this enlargement.

Both extremities may suffer in this way and the change is naturally most marked in those parts which are nearest the surface.

Usually the ribs are first affected and then the wrists, and generally epiphyseal swellings are more evident in the upper than the lower extremities.

The softening of the bones is one of the chief causes for the production of deformities of the trunk and limbs, which are so common in early life. At the same time mild cases do not always go on to bone softening and distortion.

2. Later Symptoms, as in a marked case of rickets.

Here the osseous and muscular system call for special attention. Owing to the malacia of the bones and weakness of the musculature, various deformities result.

But in addition to the softening and deformity of the bones, there is an arrest of proper growth and development of them which is evident in nearly all severe cases of rickets. This means that the children are short for their age and undersized, even after the disease has gone. The arrest of growth is evidenced by the short legs, and the small chin is due to the lower jaw being not fully developed. But the incomplete pelvic development in rachitic girls is a most serious matter. Should the

pelvis be deformed or relatively small, the difficulties of parturition may be so serious as to demand Caesarean section or other dangerous operative interference.

The head of a rickety child is typical. It is long antero-posteriorly, and the increased breadth of the forehead gives it a square appearance. The top of the head is flat and has increased width across the parietal bones. The fontanelles are usually depressed, but may be raised. They are delayed in ossification and the anterior fontanelle may remain large and open long after the end of the second year. The posterior fontanelle is usually closed before the onset of rickets. The edges of the flat cranial bones are thickened and thus elevated above the sutures and fontanelles. The sutures may be felt as furrows.

The interparietal ridge commonly seen in ordinary skulls is absent in rickets.

There seems to be no doubt that a rickety skull is larger than normal. Dr Clement Lucas had numerous measurements of rickety and healthy children taken by his house-surgeon, Dr White, and it was found that in rickets the head measurement averaged 21.22 inches, whereas in non-rickety heads the circumferential average was 19.95 inches. And this was the more striking as the non-rickety children

were at least a year older than the others.

But Ritter von Rittershain also made accurate comparative measurements which, unlike those of Lucas, showed that the rachitic skull is not bigger than that of healthy children of the same age, the apparently large cranium being due to the undeveloped face.

The brain is said to be dwarfed like other structures, and Dr Gee further describes an effusion of fluid which fills up the rest of the cranial cavity. Indeed many observers consider a rachitic child to be slow and dull in his intellect. Very often these children are precocious, possibly because being sick they are thrown so much in the society of their elders.

Trousseau held that the yielding of the soft cranium allowed more easy development of the brain and that in consequence the children were intellectual in advance of their years.

Lucas says that he regards the rachitic skull as something to be proud of and a good fortune, as its increased capacity allows of greater mental development. He points out that Thackeray and many persons of distinction in literature and science had undoubtedly skulls of larger capacity owing to rickets.

The squareness of the forehead is due to the

increased cellular cavities in the frontal bones, aided by the increased thickness of the bony structure. Parrot's nodes:—

The bosses on the frontal bones are of special interest. Parrot was the first to describe such bosses and nodes. He attributed all these nodes to rickets and considered them and craniotabes evidences of the invariably syphilitic nature of rickets. Indeed, he considered typical rickety bone changes as merely a modification of syphilis. Apparently he was unacquainted with the histological characteristics which enable rickety lesions to be traced from their beginning.

The opinion of M. Parrot was doubtless due to the fact that the children he saw at his hospital were of the poorest class, and very often certainly syphilitic.

It has been clearly shown, by histological findings, that Parrot's nodes are certainly syphilitic, so much so that even large books on children's diseases do not even mention them in connection with rickets.

Craniotabes. This symptom, which is said to be earliest observed, even at the third month, is fairly common in rickets. It was first described by Dr Elsässer of Neunstadt (Germany), in his book "Der weiche Hinterkopf". It consists in an abnormal

thinning of portions of the parietal and occipital bones, causing them to yield on moderate digital pressure, and to impart to the fingers a sensation like that derived from stiff parchment.

The softened areas are seldom much larger than a good sized pea. Eustace Smith considers them to be due to imperfectly ossified bone, and also probably to the pressure of the brain from inside, and the pillow from outside. Jenner considered these two pressures as the causes of the condition, while Elsässer in his book states that craniotabes is predisposed to by congenital weakness which softens the bones, and that the pressure of the brain is the direct cause.

Barlow considers that syphilis leads to softening, and that the pressure of the brain, then, more easily brings about absorption of bone. The point is so interesting that it is best to quote Elsässer's words on the page already mentioned:

"In craniotabes it is clearly <u>simple pressure</u> which brings about the absorption of the softened bone, its thinning, and its perforation. The pressing mass is no morbid product, no unnatural tumour, no parasite, but the <u>healthy brain itself</u> ..... The mechanical action of the brain is brought about by its regular rhythmical movements, aided by its rapid growth and its own specific weight."

The position of the softened patches is also urged as a proof of this theory of pressure. Now the positions of these softenings or thinnings are not invariably in the positions most liable to the effects of gravity. Even Elsässer, who offered the theory, shows a plate in his book which has craniotabes in another position as well.

I cannot see how the pressure of the brain, which is usually conveyed through the surrounding fluid, can affect one part more than another. Surely the pressure must be practically equal in each direction. Besides, the processes of the dura mater fix the brain to the interior of the skull in such a way that gravity is therefore greatly obviated. It is a question, indeed, if our brains really do gravitate to one side, say when we are lying down.

An aneurism may often erode bone, but a healthy artery never does. It seems probable that the greater number of grooves in adults which fit the brain convolutions are formed not because of pressure, for the adult bones are much harder, whereas an infant's soft bones show very little or none of this convolution grooving. If gravity made the brain form depressions in the skull, how is it that chronic invalids do not show evidence of this, even though they are bed-ridden for long periods?

Mr Parker rather considers these thinned areas

to be due to undeveloped bone. It has been shown that their usual position is along the posterior border of the parietal bones, at points furthest away from the single centre of ossification from which each parietal bone develops. They are not commonly found on the occipital bone, which, if the gravitation theory be correct, ought surely to be the one chiefly involved.

I am inclined to agree with Mr Parker, that craniotabes is due to incomplete development. I have never seen craniotabes in a sheep unless there was abnormal pressure at the point, due to one or more cysts.

There is a tabetic dog's skull in the Museum of the Royal College of Surgeons, England, but it is not said whether the brain was healthy. Possibly it was, as in sheep, due to a cyst or tumour of some kind.

As to the disease which causes craniotabes, opinions vary. Elsässer, Vogel and Steiner all consider craniotabes to be due to rickets, but Friedleben states that it may occur in healthy children who are not rickety. Indeed, he maintains that it is a normal condition during the latter half of the first year of life, and that it is more marked in hand-fed children. He also says that it occurs in animals - of this there is sufficient proof.

But the fact that he mentions it as being specially marked in hand-fed children seems to suggest that those children probably had rickets.

Parker and West in England, and Meigs and Pepper in America also support Elsässer's contention. The first named quotes several cases to support this view.

But in England such eminent authorities as Sir Thomas Barlow and David B. Lees, whilst readily admitting that Parrot's nodes are typical of syphilis and not rickets, yet hold that craniotabes is, as Parrot thought, really a sign of syphilis. They very carefully enquired into 100 cases of craniotabes and in 12 of them no history or sign of syphilis was to be found. In 47 they found signs of syphilis, and of these 35 showed marked craniotabes and 12 only slight. Hence they conclude that fifty per cent. of the children who show obvious craniotabes, and forty per cent. of those in whom it is less prominent, or forty-seven per cent. of the total number of cases, are almost certainly syphilitic.

The children who were believed to be syphilitic, yet who had not craniotabes, were either under three months of age or over ten months; that is to say, they were outside the craniotabic period.

They came to the conclusion that syphilis is by

far the largest factor in the causation of craniotabes, and that marked craniotabes must be regarded as strong evidence for suspecting the existence of a syphilitic taint. But it must be noted that many of the cases they quote had signs of rickets. They say that all their older cases, besides some of the younger, showed some signs of rickets. And they go on to explain these symptoms as being due to faulty diet, as they found that syphilitic children were very apt to be badly fed.

It is their opinion that even if it were proved that craniotabes was the first sign of rickets, it would not invalidate proof that craniotabes is itself the result of syphilis; and that the only conclusion resulting would be that syphilis in children is very apt to lead to rickets.

Again, Virchow, whose opinion must weigh very much, writes: "I have never seen a case of craniotabes in the dead body in which there were not other bones, especially those of the thorax, showing rachitic changes." He speaks of Elsässer's works in terms of praise, and entirely agrees with his views.

Professor Still supports the views of Barlow and Lees. Whilst making observations on congenital syphilis he found that craniotabes was most often present in cases of marked congenital syphilis, or

where the suspicion of the existence of that disease was strong. At the same time he found a number of cases showing craniotabes in which no sign of syphilis could be obtained. He found craniotabes to be very infrequent amongst rachitic children.

Experienced observers have noted that craniotabes is practically invariably associated with laryngismus; and laryngismus is generally admitted to be almost always due to rickets. It has rarely, if ever, been ascribed as being due to syphilis. So that, if laryngismus and craniotabes are thus closely associated, the conclusion must be in favour of the rachitic nature of craniotabes.

The late Dr George Carpenter recently studied this question and he was inclined to consider syphilis as the more important factor, but concluded that both syphilis and rickets combine to favour its production.

The thinning of the hair over the occiput is often well seen, and is probably caused by the rubbing against the pillow, aided by the copious perspiration which soddens the hair.

The hair is frequently late in coming, but as the rickets improves, it gets remarkably quickly thick.

In connection with the anterior fontanelle, it

has been observed that in most cases of rickets a systolic murmur of variable intensity may be heard. Senator, quoted by Eustace Smith, considers it to be due to the ossified membrane being a better conductor than cranial bones. It certainly is rarely heard when the fontanelle is closed. Eustace Smith records a case in which this systolic murmur was so loud as to be heard by bystanders as a purring sound. It was loudest on the right side of the head.

Fleischmann has pointed out that the lower jaw has lost its normal curve, being flattened anteriorly so that the incisors are in a straight line: that at the level of the canine teeth the lower jaw forms a sharp angle and bends abruptly backwards, this being due to the imperfect growth of the central portion of the jaw.

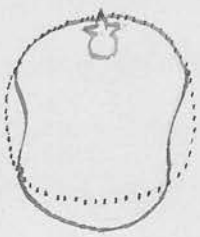
Further, Baginsky describes an occasional asymmetry between the two halves of the bone, so that one side appears higher than the other.

This delayed development of the jaw greatly affects the dentition, and makes the face look small.

Dentition is arrested as soon as the facial bones become affected by rickets; so much so, that teeth may be much delayed in their eruption, and when they do erupt they usually appear very irregularly and tend to be of bad quality from imperfect development of the dental enamel. They frequently

blacken and decay.

The chest in rickets is deformed in a characteristic manner, owing to the giving way of the softened ribs and the yielding of the costo-cartilages. The latter are unable to resist the external pressure of the atmosphere. Therefore when the chest is expanded on inspiration the weak parts give. Thus the middle or weak point of the arch of the rib gives way and tends to push the sternum forwards.



Cross section of rachitic chest (Gee). Dotted line represents normal chest for the same age.

This causes a grooving laterally and from the projection forwards of the sternum gives rise to 'pigeon breast' shaped thorax. These grooves extend from the second and third ribs to the hypochondrium, and the lowest parts of them are formed by the ribs outside the costo-cartilages. So that along the inner side of each groove the enlarged ends of the ribs are seen like a row of beads. This beading or rickety rosary is very typical of rickets.

But the pigeon breast and the grooves are most marked in those cases complicated with pulmonary catarrh, i.e., some form of pulmonary obstruction which makes the intra-thoracic pressure less and

causes increased respiratory exertion.

In such cases Harrison's Sulcus is well seen, one on each side of the thorax. They are transverse and beginning about the level of the xiphisternum they are directed outwards and slightly downwards. Very seldom do they extend as far as the mid-axillary line. They are limited to the most yielding part of the thorax, i.e. to where the thorax is widest. Lower down than the sulcus the liver and other abdominal contents form a support and so obviate the chest from being further indrawn. These sulci are sometimes represented by a transverse furrow at the level of the epigastrium. This furrow is especially marked in patients who have suffered from much pulmonary catarrh. These children have an impediment to the entrance of the air, because the smaller tubes of the lung are narrowed by the congestion, and there may be pulmonary collapse.

Rokitansky considered this groove to be due to the traction of the diaphragm, but Sir William Jenner has shown this not to be the case. The weakened and nearly unsupported ribs give way at the line because each inspiration in-draws this part, and the pulmonary condition greatly increases the effect.

The spinal column is frequently distorted. In some children the cervical curve may be so increased

as to cause the child's head to lean backwards upon the shoulders and gives rise to a characteristic attitude. And the weight of the head and shoulders, as the child sits bending forwards, is apt to produce kyphosis, which may be so marked as to simulate vertebral caries. But this deformity at once disappears when the child is held up under his arms, as the weight of the limbs and pelvis come into play. The spinal curvature may be forwards, lordosis, or if the patient has been sitting or walking much there may be scoliosis. In these cases the deformities are due to the weakness of the ligaments, muscles and also to the yielding vertebrae. Unless measures are taken to improve the disease, these deformities may become permanent. Many of the cases of twisted spine that are seen in adults owe their origin to rickets.

The pelvis may be altered in various ways, according to the age of the child, to his usual attitude, and whether he is able to walk or not. The distortion is frequently such as to make the pelvis an irregular triangle, and is due to pressure of the limbs on the acetabula. The sacral promontory is unduly projected forwards. Pelvic deformity, if permanent, is a very grave matter for the future in female children, as it may seriously interfere with parturition. Even in early life pelvic deformity

may greatly interfere with the success of operations for vesical calculi, etc.

The limbs are usually thickened about the joints and the shafts are frequently over-curved.

The Humerus is said to be frequently curved outwards at the deltoid insertion, this being due to the weight of the hand and forearm when the arm is raised (E. Smith). Personally, I think the weight of the forearm is not sufficient to cause this, but that it is due to the child putting his weight on the arm when sitting, trying to stand or crawling on hands and knees. For we find that the radius and ulna are also curved outwards. It is interesting here to note that Mr Lucas considers the fact that the wrists show more marked epiphyseal enlargement than other long bones to be due to the child crawling on his hands and knees, and so pressing on the lower ends of the radius and ulna.

The femora and tibiae are curved outwards and forwards. The knees can often be seen to be wide apart, this being partly due to the child sitting cross-legged. In fact these various deformities of long bones are mainly due to their inability to properly support the weight they are required to, and the muscular force applied to them. But the weight that the soft bones are called on to support is by far the more important factor.

The lower third of the tibia commonly show an abrupt curve, which has its convexity outwards and forwards.

In cases of extreme distortion of long bones, partial or greenstick fracture may result and the clavicles have much accentuated curves.

Muscular Symptoms. The muscles are, in some cases, markedly feeble, but the joints also are lax on account of the loss of tone in the muscles, and the ligaments are slack.

The child may be unable to sit up, and it can be frequently noted that the lower extremities appear quite powerless.

Reference has already been made to the effects on the thorax of muscular weakness.

It appears to be still uncertain whether the muscles are structurally altered, but microscopical changes, such as indistinct striation and fatty infiltration of them, have been recorded.

The myalgia and general tenderness of the muscles has already been written about. This tenderness, which is usually general, is especially marked in cases complicated with scurvy or some acute inflammatory affection. In fact in every case of rickets where tenderness is marked, one should carefully look for symptoms of scurvy and enquire about the child's diet.

The abdomen is nearly always unduly prominent, even where there is no apparent disease of the liver or spleen. This "pot-belly" is due to the feebleness of the abdominal muscles, to the intestines which are frequently distended with flatus, and to the fact that the shallow pelvis projects the abdominal contents upwards and forwards above the level of the pelvic brim. Careful palpation and percussion will eliminate enlarged spleen, liver or hydronephrosis as causes of this "pot-belly".

Congenital cystic kidneys, though often large, do not generally cause this pot-belly condition. Palpation will usually reveal their presence if they are enlarged.

The appetite is variable. Some children have a poor appetite, while others are always ready for their food. Even if the patient is still passing large amounts of putty-like motions, he will usually take his food well.

There is no marked sickness, but often one notices that these children eructate part of their food. This eructated fluid is generally of a sour odour.

In children who have been very badly dieted, attacks of diarrhoea are frequent, the excreta being slimy, very offensive, and sometimes greenish in colour.

The complexion of the rickety child is pale and the lower eyelids may be discoloured. This latter is especially seen in scurvy rickets. The mouth may be of bluish tinge. The superficial veins of of the face and jugulars are usually very prominent.

Breathing is often rapid, as disease of the thorax is almost constantly associated with bronchitis and atelectasis.

The lymphatic glands are in many cases slightly enlarged and give rise to a "shotty feeling" under the skin. These glands are to be felt along the anterior and posterior borders of the sterno-mastoid, in the axillae and groins. They tend to get smaller as the child improves, but one has seen them persisting for a long time after the rickets has been cured.

In rickets that is uncomplicated there is not much fever. But if there are complications, such as pulmonary catarrh, etc., the temperature may rise considerably. Even the cutting of a new tooth may send the temperature up, as it often does in healthy children.

The amount of wasting varies with the severity of the attack. In a mild case the child loses his rosy cheeks and becomes pale. He remains fat from over-nourishment of the subcutaneous tissue and is flabby.

In a severe case he will frequently become much wasted, and the belly is very prominent.

In conclusion it may be said that the most prominent symptoms are profuse sweating at night, increased fretfulness, enlargement of the long bones at their epiphyseal ends, prominent abdomen and distortion of the long bones of the thorax and limbs.

COMPLICATIONS are many, but the two chief ones are pulmonary catarrh with atelectasis, and severe diarrhoea. Both of these are probably due to the patient being very sensitive to chills. The child is perspiring too freely and is so restless in bed that he is very apt to catch a chill.

Very few rickety children are without cough, indeed Still considers that practically every child who has his thorax affected by rickets, has in addition bronchitis and atelectasis.

The softened ribs and distorted chest readily give rise to bronchitis, which results from the atelectasis and emphysema. The narrowing of the smaller tubes further increases the atelectasis and so aggravates the catarrhal condition.

This lung collapse is very dangerous and many

fatal cases are due to it.

Severe diarrhoea is also a source of grave danger, as the child is much enfeebled by it. The direct cause of it is often a chill, but the predisposing causes are probably the altered and unhealthy intestinal secretions, enlarged lymphatic glands, congested viscera and diseased bones. The blood producing organs being deranged, it is quite obvious that the secretions must be affected.

Certainly the faulty diet of the patient also is an important factor in the production of this diarrhoea.

Besides these complications there are various nervous ones, perhaps the most important being laryngismus stridulus. It appears uncertain whether this is due to laryngeal catarrh or to irritation of the recurrent laryngeal nerve by pressure of the tracheal rings, or pressure on the vagus by bony enlargement about the jugular foramen. Certainly, there is probably laryngeal catarrh in association with the bronchitis, and taking into account the fact that the infantile glottis is frequently a mere chink, a slight catarrh even would cause spasm of the larynx. But in view of the probable explanation of tetany, it would appear that the spasm is really due to nerve irritation.

Laryngismus is said to only occur in rickety

children, but Eustace Smith contradicts this in his paper on the subject. He quotes several cases of it in non-rickety young infants. In each case he found a common feature, namely, that they all suffered from extreme nasal obstruction due to adenoids and post-nasal catarrh. He considers that in rickets it is due to reflex nerve influence caused by catarrhal irritation in the naso-pharynx.

It is sometimes associated with spasm of the oesophagus in its upper part.

Closure of the glottis may be due to exhaustion of air in the trachea, the soft parts of the rima glottidis falling in and not contracting as in spasm.

Dr Marshall Hall proved this to be possible by experimental exhaustion of the tracheal air. Hence Dr Vivian Poore suggested that the stridor might be due to this cause and not to spasm. A laryngoscopic examination during an attack would settle the question, but it is not possible to carry it out.

Laryngismus may be a cause of much anxiety and requires careful treatment. Some cases are so severe and obstinate that even antiseptic and sedative sprays, with chloral and bromide internally are unable to cope with the condition. However, as a rule, it generally yields to prompt treatment.

General convulsions may frequently be met with in cases of rickets. They are probably reflex

nerve spasms which originate from gastro-intestinal irritation.

Tetany is sometimes seen in rickety children. Oliari, analysing 1,500 cases of rickets, found that 1,144 had no spasms, 80 were doubtful, and of the 276 with spasms, 167 had convulsions, 96 tetany and laryngismus, and the remainder, other forms of spasms. He found that out of 337 children brought to hospital for convulsions, 266 were rickety: that is, 78.9 per cent of the convulsion patients were rickety, and 18.4 per cent of the 1,500 cases of rickets showed convulsions. He says that the greater the deformities, the less liable are the children to have spasms.

Dr James Burnet has pointed out that tetany, especially in rickets, and also in other diseases, is commonly associated with gastrectasis. In the three rachitic cases that he quotes he found the stomach markedly dilated. In one of them it even extended to midway between the pubes and umbilicus.

Trousseau has shown that after tetany has disappeared, it can be reproduced by firmly constricting the arm or leg in the grasp of the hand for half a minute or a little longer. In this way the typical tetany position can be reproduced in some cases long after, even weeks later, the spontaneous tetany has gone. It appears uncertain whether pressure on

the nerves or vessels is the cause of this phenomenon.

Facial irritability is commonly associated with tetany and laryngismus. A gentle tap over the facial nerve will cause contraction of the corresponding facial muscles and platysma. At the same time tapping the motor nerves of the arm and leg may show a similar contraction of the muscles they supply.

Zonular cataract, where some of the strata of the lens between the nucleus and the cortex become opaque, occurs sometimes in cases of convulsions due to rickets, etc. The reason of this is not known.

Chronic hydrocephalus may result where the brain of the rickety child is small and fluid is effused to fill up the cranial cavity. There is seldom much effusion of cerebro-spinal fluid and therefore this is rarely of grave import.

It is interesting to note that a weak rickety child is not specially susceptible to tuberculosis. Indeed rickety children have been shown to be freer from tubercle than others who are non-rickety. At the same time a child with rickets may become tubercular.

Ritter von Rittershain has asserted that a large proportion of the fathers of rachitic children were tubercular, but Sir William Jenner had similar

investigations made by Dr Edwards, which appeared to show that the children of phthisical parents were even less likely to become rachitic than those of others.

Chronic cerebritis and albuminoid disease of the brain have been described by a few observers. But even if they do occur as complications, they are excessively rare.

Scurvy is very apt to complicate and be mistaken for rickets. The child suffering with scurvy rickets is remarkably sensitive to even gentle handling, is easily bruised and is liable to haemorrhages under the skin, conjunctivae, and from the nose. Nearly every case of it has been fed on solely artificial foods.

Enlargement of the spleen is probably not a common occurrence in rickets. Professor Still could only collect 42 cases of enlarged spleen, and in 24 of them the rachitic nature of the ailment was doubtful.

It is not an essential feature of rickets, and the subject is discussed more fully under the pathological heading. When there is much splenic enlargement, the patient is markedly pale in the face and mucous membranes.

DIAGNOSIS OF RICKETS.

The diagnosis of a typical case of Rickets is not difficult to even an inexperienced observer. The enlarged wrist joints and tendency to outward curvature of the legs generally decisively point to Rickets.

But it is very necessary to avoid the mistake of diagnosing as true Rickets what was called Acute Rickets but now Scurvy or Barlow's Disease. In this condition the inflammatory and sanguineous effusions sub-periosteally are very apt at first to be mistaken for Rickets.

In discussing the question of diagnosis, it may be convenient to divide cases into mild and marked.

1. In a mild case of Rickets, the salient points are :

(a) The epiphyseal enlargement of long bones.

(b) The tardy and irregular dentition, and

(c) The inability or backwardness in walking.

On seeing a child with enlarged wrists, it is as well to ascertain the number of teeth it has, and at the same time ask about its walking powers.

Even before there is evidence of articular swelling, one can usually correctly suspect the onset of

rickets. The child is often fat and flabby, and may even be rosy-cheeked, but on enquiry one finds that he sweats profusely about the head and neck at night, and is very restless, so much so that the bed-clothes are with much difficulty kept over him.

The motions are often large and putty like, and the child has symptoms of gastro-intestinal irritation, e.g. acid eructations after food.

The tenderness of the limbs and body generally may be due to scurvy as well as to rickets.

Congenital syphilis can usually be readily separated from rickets, as the former shows snuffles and rashes which are characteristic. Also syphilis tends to produce more extensive and diffuse thickening of the lower end of the diaphyses than does rickets. And, moreover, syphilitic bone lesions are destructive, leading to separation of the epiphysis from the shaft, and to abscess formation in various parts of the limbs and body.

It is practically certain to be a case of Rickets when a child of say 8-12 months shows enlarged wrist joints, irregular dentition, and if his limbs helplessly double up under him when he is held up on his feet.

2. Marked Cases: Here the child shows marked enlargement of the wrist joints, the chest is deformed and may even be pigeon-breast shaped, the legs

are typically curved and on examination the ulnar and radius may be found markedly curved. The ribs are beaded at their sternal ends and Harrison's sulcus may be seen.

The posture is often typical as the child may be seen sitting with crossed legs which he does not care to use for walking. He may even place his hands before him on the ground with a view to supporting his feeble spine. But other cases of rickets, who are getting stronger, tend to run about freely.

In those who have apparent complete uselessness of the legs, one must guard against diagnosing Infantile Paralysis. The characteristic tenderness of the limbs in these cases and the fact that they can move them, differentiates them from Infantile Paralysis. In Infantile Paralysis pinching of the limb produces no movement. But in severe cases of Scurvy Rickets the child will not move the limb if it is pinched, as it knows too well that the slightest movement is excruciating.

The shape of a rickety child's head may be of some value in diagnosis. The head is elongated from before backwards and is typically square about the forehead.

A skull that is hydrocephalic is usually globular. The late closure of the fontanelles, especial-

ly the large one (anterior fontanelle) is typical of Rickets, but whether it is raised or depressed is of very little value as both Rickets and Hydrocephalus may show either the one form or the other.

Eustace Smith says that a depressed fontanelle is compatible with a fairly copious intra-cranial effusion.

The spine may be curved antero-posteriorly or laterally. The former curvature disappears if the child is lifted up under the arms so that its body weight may act.

Lateral curvature must be diagnosed from cases of pleurisy with effusion or one sided empyema. This is usually readily done, as there are typical signs of Rickets, and cases of effusion or empyema can at once be diagnosed by auscultation and exploration with a suitable syringe.

Rickets can be readily differentiated from the epidemic anterior poliomyelitis that appears from time to time in England and Germany, as the paralyses are not accompanied by swelling of joints and other characteristic signs of rickets.

The essential pathological difference of rickets and osteomalacia will clear up a diagnosis, but in life these two conditions may be confounded. A careful survey of the symptoms and mode of onset will generally greatly assist the diagnosis.

In scurvy rickets the general tenderness is very marked and there is invariably a history of the child being fed solely on artificial food.

PROGNOSIS.

One can usually give a very hopeful prognosis, as improvement under treatment is early marked in uncomplicated cases.

If there is pulmonary catarrh in the form of bronchitis with atelectasis and a deformed thorax, the case becomes a serious matter and our prognosis must be guarded. Similarly in those cases that have splenic enlargement with its frequent profound anaemia, the prognosis may be bad. Some of these cases may gradually get weaker and die. Severe diarrhoea is similarly very unfavourable.

Laryngismus stridulus in children with a markedly infantile form of epiglottis is sometimes a cause of sudden death.

Still says that many cases of death in rickets are due to general convulsions, but he also states that it is difficult to tell whether the fatality be due to the laryngeal or the general convulsions.

Eustace Smith, on the other hand, believes that general convulsions have rarely any ill-effects.

Personally, I incline to Professor Still's opinion.

Rickets complicated with syphilis may be given quite a hopeful prognosis, as both the conditions, if without serious complications, are amenable to treatment.

The age of the patient matters very little in prognosis, except that the older the case the less likelihood is there for complete recovery from the distorted limbs. At the same time it is very remarkable how greatly - even apparently badly affected bones and joints - these distortions are improved under treatment. Large joints are frequently seen to be getting smaller and even normal, crooked bones straighten, and a deformed chest will recover itself to a large extent.

Many cases that are not brought in good time for treatment leave serious defects in the chest, limbs and pelvis. The last mentioned may prove very serious indeed in girls who later on may bear children.

We can, therefore, generally give a good prognosis in most cases of rickets, but it is perhaps wise to be a little guarded as regards the complete disappearance of deformities or disfigurement.

The progress of rickets is slow and where insanitary conditions continue the child is liable to get worse and die of pulmonary catarrh.

When improvement sets in, it is frequently surprisingly rapid, especially under careful treatment.

TREATMENT OF RICKETS.

Whatever the aetiological factor or factors may be, it is generally conceded that rickets is best dealt with by careful attention to the hygiene and diet of the patient, with probably the use of cod liver oil in some form or another.

It is essential that the doctor should carefully find out the conditions in which the child is living; and enquire about the amount of food given per meal and if this is too much, or of unsuitable quality, it should be put right. Frequently mothers are very lax about keeping to a properly graduated amount of food, and also neglect to have regular times of feeding for the child. This should be attended to.

It is greatly to be deplored that nowadays so many children are not suckled. Mothers often do

not wish to be troubled with suckling, and in this way probably do the child a grave injustice. A child suckled on the natural food as supplied by a healthy mother who has milk is very unlikely indeed to become the subject of rickets. Unfortunately also, many women are unable to suckle their children either through ill-health or not having any proper supply in the breasts. Such women have to use cow's milk or one of the many artificial foods. And these children who are brought up artificially are chiefly the ones that supply cases of rickets. So that one must know what kind of food the child receives and find out by various enquiries if it is suitable.

Very often the food used by the better classes is quite good and well digested for a long time, and then, because the child has been kept so strictly on preserved foods, without the addition of fresh milk and the fresh juice of suitable fruit, it develops gastro-intestinal symptoms - the stools begin to be putty like and large in amount. At this stage were the diet altered to one with some malt in it, and fresh juice of fruit (orange) given, the danger of rickets and scurvy would be safely avoided, provided the hygiene of the child be also good.

I think that every tin of preserved food, such as the well-known Allenbury's, Mellin's, etc., should

have instructions in bold type, preferably of a different colour to the ordinary instructions, on them saying that it is absolutely essential to give the child some fresh fruit juice and fresh milk besides the food being used.

The utensils used in feeding should all be carefully cleaned after use. Feeding bottles of the type with the long and frequently bacillus and dirt laden tubes should be interdicted. They can seldom be properly kept clean.

Rickety children who have been fed on bread, arrowroot, cornflour, potatoes and other starchy foods will at times be better dieted with such things as freshly prepared beef-juice and gravy, and gradually milk can be again given satisfactorily.

Naturally, the diet of a rickety child varies greatly with its age. But many cases that come for treatment are about 11-12 months of age, and they can usually digest good milk well and are old enough to be given good gravy, custard pudding, broccoli and cauliflower; whereas children of 18 months or more may eat well pounded underdone meat with well cooked cauliflower and gravy.

Eustace Smith gives the following excellent diet:-

Breakfast: A breakfast-cupful of milk, with one or two teaspoonfuls of Mellin's Food dissolved

in it.

At 11 a.m. a breakfast-cupful of milk, alkalinised by fifteen drops of Liq. Calcis Saccharata.

Dinner at 2 p.m.: A good tablespoonful of well pounded mutton-chop, with gravy and a little crumbed stale bread; or a good tablespoonful of the flower of broccoli, well stewed with gravy until quite tender, thin bread and butter (or buttered Dutch rusks), and toast water to drink. To this one would prefer to add the juice of a good sweet orange, given after the meal.

Tea at 6 p.m.: Same as breakfast, or a lightly boiled egg if no meat has been given.

In many cases starch is given in excess to artificially fed children, and if this is found to be the case it should be reduced in amount and malt added to the diet. Many proprietary foods bear this in mind and have the starch digestion aided by malt. Should the food have no malt we may add with benefit such a preparation as Hoff's extract of malt ℥ī-ĪĪ t.i.d. Malt is here of distinct service.

Frequently by altering the diet in this way a case of rickets will greatly improve.

Practically all cases of rickets are preceded by digestive trouble or bowel complaint, and unless the child is improving we find the looseness of the bowels or other intestinal derangement is still

present when the patient is brought for treatment.

As regards the hygiene of the child, it must be impressed on the parents that sunshine and plenty of fresh air are also essential for the patient. He must, if possible, be regularly taken out of doors for some hours, say one to two hours in the morning and a similar amount in the afternoon. This cannot be done by many people who have to work for their living, whereas the better off can be advised to give their children as much fresh air as possible. The women who have to work during the day can at least have the child in a cradle or box out of doors part of the day. It is not easy for the poorer class, who live often in anything but salubrious surroundings, to give the child the amount of pure fresh air he should have.

When the child is taken out, he should be warm beforehand and carefully wrapped up. The belly should have a warm flannel binder, and it should be seen that the diaper is not wet when he goes out. In this way the child will not catch cold, unless the cart he is taken in be insufficiently sheltered. The cheaper forms of mail carts have little or no protection from the wind or cold, and in bitterly cold weather the child is very apt to come home shivering and liable to catch cold. A hot water bottle placed at the foot of the cart is very useful

for infants.

Attention to the condition of the diapers has been mentioned, but it is often necessary to instruct the parents that the child must not be allowed to lie, as they frequently do, for lengthened periods with wet and dirty diapers; the child often lying in the midst of foul and ammoniacal odours which are certainly detrimental to his health.

The bed should be always kept dry and sweet, in spite of the extra trouble it entails on the mother or nurse.

Each morning, at least, the mattresses and bedclothes should be aired thoroughly. The sheets should be frequently changed and any waterproof used under them should be washed and aired also.

The bedroom should be well ventilated. A small fire in cold weather ensures a good circulation of air. A lamp would answer the same purpose, but personally I do not like paraffin lamps in bedrooms for heating purposes. I consider them very dangerous unless they are really first class ones. I have known a gentleman nearly asphyxiated with one that worked well usually, but on that occasion was faulty.

The above measures will usually ensure improvement, and are of probably much greater importance than the medicinal treatment. Attention to the unwhole-

some conditions under which the failure in nutrition has taken place should be our first duty.

Drugs are often of very great service, but they should take the place of complement to the above treatment.

If the child has diarrhoea or too frequent motions, a drop of Tinct. Opii will counteract the undue peristalsis and relieve colicky pains, and should the child eructate, as they often do, mouthfuls of sour fluid, a few grains of sod. bicarb., e.g. grs. ii.-iv. in a little aq. anisi will be useful.

The following prescriptions are useful for this acidity, and if required a drop of tr. opii may be added to a dose or two.

℞: Sod. Bicarb. grs.xxiv.
Spt. Chloroformi ♏.xxiv.
Aq. Anisi ad ℥ß
Sig. ℥i. t.i.d.

℞: Ferri et Ammon. Cit. grs.xxiv.
Sod. Bicarb. grs.xlviii.
Tr. Nucis Vom. ♏ xxiv.
Glycerinae ℥iv.
Aq. Anisi ad ℥ß.
Sig. ℥i. t.i.d.

When the bowels are in good order it is well to give some cod liver oil, preferably a natural oil, as the white over-purified ones are not so effective. Jong's cod liver oil is a good preparation.

Children cannot readily digest fats, and infants

especially must not be given too much cod oil. A good plan is to give about 10-15 drops three times daily after food, and carefully watch the motions. If the stools show no traces of oil, more may be given with advantage, increasing the amount by a few drops each time. As soon as oil appears in the motions, the child should have a little less.

Southwarth considers olive oil an efficient substitute for cod oil when the latter is badly borne in hot weather. He also attaches much value to the juice of oranges in the treatment of rickets as well as in scurvy.

Phosphorus. The advantage of giving phosphorus is denied by some authorities, e.g. Eustace Smith does not even mention it in his treatment of rickets. Goodhart and Still find it entirely without decided value.

But at the same time other equally eminent men do place much faith in it. Kassowitz and other Continental writers have published a large series of observations upon its value.

Some American writers favour the administration of phosphorus in practically all cases of rickets. J. J. Thomas and A. F. Furrer of Cleveland, Ohio, publish a series of 100 cases of rickets, and their routine prescription is :-

℞: Elixir phosphori ℥v.
Ol. Morrhuae ad ℥ii.
Sig. ℥ß t.i.d. p.c.

Southwarth lays great stress on the value of it. He has had excellent results by giving gr. 1/200 of phosphorus in ♏xxx. of cod liver oil.

Personally I never use phosphorus and prefer to use calcium lactophosphate in combination with the cod liver oil. And I attach far more importance to attention to diet and hygiene, massage and the administration of cod liver oil than to other drugs.

At the same time phosphorus has been much used of recent years and many eminent men maintain that there is no question but that free phosphorus has marked effects on the growth of bone.

Kassowitz, already mentioned in this connection, found that even in advanced types of rickets speedy recovery always resulted by giving phosphorus, and that the bones became hard in four weeks in most cases. Many others confirm these results, e.g. Wegner and Berg.

Hazard's solution as devised by Thompson consists as follows:-

℞: Phosphorus 1 grain
Absolute Alcohol 350 minims
Glycerin 2 ounces
Spt. of Peppermint 10 minims.

Five minims of this is given to children of 3 years of age.

Berg states that double this dose should not be used, as there is a danger of causing fatty degeneration of the liver cells.

Whitla prefers the following prescription:-

℞ Olei Phosphorati (B.P.) mxl.
Olei Morrhuae ad ʒvi. Misce

Sig. ʒi. t.i.d. p.c.
(For a child one year old.)

Dawson Williams also considers it of use in the acute stage in fat children who are tender and perspire too freely.

In Holland, where rickets is so prevalent, it is interesting to note that phosphorus is not used very much. Professor Stokvis of Amsterdam never prescribes it, although he considers it a rational treatment. Presumably, from what he remarks to his students, he objects to it because cases of phosphorus poisoning have resulted in the practices of Leo and Nebelthau, who have treated thousands of cases of rickets with cod oil and phosphorus, phosphorus poisoning being found post-mortem. These deaths occurred in January and March 1902.

An unpleasant case of phosphorus poisoning is cited in the "Annales d'Hygiène", 1891, p.517, in which a certain doctor was sued for damages for causing the death of a rickety child aged 2 years. During the proceedings it was clearly shown that the

patient only received an ordinary amount of phosphorus and well within the limits of proper dosage. Fortunately, however, the doctor was exonerated from all blame, although there was ample evidence post-mortem of phosphorus poisoning.

Leo states that even the smallest doses of phosphorus are liable to cause poisoning, and he advises great caution in its use.

Nebelthau's fatal case resulted after taking only six teaspoonfuls of cod oil and phosphorus, the mixture containing 10 mgrms. phosphorus in 100 grms. of cod liver oil. This child was 2 years old and died of acute phosphorus poisoning. The post-mortem findings showed less phosphorus than was given.

Criticising the fatal cases of Leo and Nebelthau, Ungar states that there was insufficient evidence of phosphorus poisoning, but at the same time it would be very difficult to prove that it was not.

Hryntschak and other observers do not consider phosphorus of any value in rickets, and they also state that those who do exhibit phosphorus should exercise great care in its administration.

The last mentioned person further says that generally phosphorus and cod liver oil mixtures contain very little, if any, phosphorus, and he entirely rejects phosphorus as a specific for rickets.

Monte and Zweifel quite agree with these observations of Hryntschak.

In conclusion it may be said that it is probably unnecessary to use phosphorus, as there are certainly safer and still more efficacious methods of combating rickets.

Iron is of use as a general tonic, especially if the child is anaemic.

I seldom use it in treating rickets, as the anaemia, which is apparently present, rapidly improves with proper diet and plenty of fresh air.

When it is required, syrupy preparations are probably best avoided, as they are apt to cause fermentation and acidity in the digestive tract. But Still greatly favours the syrup of calcium lactophosphate and iron lactophosphate. He uses 3ß-ī well diluted.

The following are useful:- Iron Wine ♏ xx.-xl., Ferri Sulph. Exsicc. grs. ii.-iv. or Tr. Ferri. Perchlor. ♏ v.-xv.

In any case, care must be taken that the iron does not interfere with the digestion and regularity of the bowels.

Calcium salts were formerly much used in order to supply the deficiency of lime. But it is now known that the child generally receives plenty of

lime with the milk, etc., but is unable to make use of it. Many still use calcium salts, e.g. calcium lactophosphate. Eustace Smith says that they are useless in practice, as the benefit of them is so inappreciable.

Quinine is seldom required in treating rickets. The tannate of quinine is the best to use, in doses of gr.i.-ii.

Cod liver oil and malt extract are the most useful.

The Maritime treatment has been much advocated in France and Italy. The first hospital built specially for this purpose was erected in Cette 1847. This was followed by many similar establishments in Bercq (France), to which numbers of Parisian children are sent for the cure of rickets. In 1850 Germany and Austria followed the example of France.

In several Continental countries, e.g. Holland, France and Italy, the medical profession consider sea air as of great value for rickets.

In Genoa the children are taken each day, in the summer, and bathed in the sea; after which they are placed for a variable time in hot sand baths in the open.

Experience of this form of treatment is there considered to be the most efficacious.

Lercux states that even the worst cases of rickets are absolutely cured by long residence and treatment at the sea-side, and that the earlier the case the more rapid the benefit obtained. He adds that the children improve in appetite, and at the same time receive better food.

Cartier has written a special treatise on the maritime treatment of rickets, and is a strong advocate of it.

Cazin, a French physician, writes very descriptively of the effects of this treatment: "Town children, even of the most sickly appearance, alter in an astonishing manner. The skin gets firmer, the eyes brighten, the limbs become stronger, the appetite improves, the digestion stronger, habitual diarrhoea stops, breathing becomes freer; in one word, they become entirely altered for the good."

Varekamp, of Zandvoort, Holland, attaches great value to sea water and sea air. After bathing, the children are encouraged to play about on the sands so as to get as much sea air as possible.

In Zandvoort there are only early cases of rickets, all being attributed to artificial feeding. Soon after these patients are sent out to the sea-side they are cured.

Dr Robert Lee of West Drayton, England, kindly writes me that ozone is now being used in the

treatment of rickets, but I regret that I only know of this maritime treatment, in which ozone may be a curative factor.

Massage. As soon as the local tenderness in the joints and general tenderness of the body have gone, it is very helpful to carefully and firmly massage the flaccid muscles and body as a whole. A little simple warm Ol. Olivae will do as a lubricant. Care must be strictly taken to have a warm hand and to begin massage gently, so as to have the confidence of the child. After a time the child appears to rather enjoy the massage, provided it has not been hurt at the outset by roughness.

The spine, arms and legs should receive special attention, and I think the prominent belly in rickety children is greatly helped by abdominal massage, as this seems to be partly due to flaccidity of the recti and other abdominal muscles.

I have found sponging the child with cold sea water all over the body, arms and legs very beneficial. The child should be at once properly dried and dressed after it, and he will not catch cold, but rather get sturdier.

As the strength improves he may have tepid and later cold baths, or a tepid or cold douche of sea water may be applied to him while he sits in the bath.

Electricity may be useful to tone up the muscles, but in the cases I have tried it I did not find it of any use.

Bronchitis being often present in patients with rickets, and often being very serious in nature, it requires special attention. The child's chest should be rubbed each night at least with warmed oleum camphorata. Should the oil cause a skin eruption, it should be replaced by warm oil (olive) to which, later, camphorated oil may be added. A prescription such as the following will be found useful as a stimulating expectorant:-

℞

Ammon. Carb.	gr.xxiv.
Potass. Bicarb.	ʒii.
Ext. Glycyrrhizin	ʒβ.
Aq. ad	ʒiii.

Sig. ʒi. 3 or 4 hourly.

Convulsions are best treated by hot baths, with application of cold cloths to the head.

Chloral and bromide of potassium are of distinct value here, and for a very young child of a few months the following prescription I have found useful:-

℞

Potass. Bromidi	gr.xxiv.
Chloral Hydrat.	gr.xxiv.
Glycerine	ʒβ.
Tr. Lavandulae Co.	ʒβ.
Aq. ad	ʒiβ.

Sig. ʒi. 3 hourly.

The quantity of bromide and chloral must be increased according to the age of the patient and the severity of the convulsions.

More fresh air should probably be advised and the body sponged with cold or tepid water night and morning. At the same time much care must be taken to avoid draughts, which are so liable to cause bronchitis which is so often fatal.

Bossi has shown that the removal of one suprarenal from sheep caused osteoporosis and suggests that rickets may be explained by some defect of this kind, or at any rate that adrenalin may be of some value in its treatment. I have not heard of any cases treated with adrenalin.

Vaccine treatment. Artom and Agnese have produced symptoms like rickets by inoculating white rats with diplococcus osteomalaciae. It has been suggested that rickets might be treated with a vaccine of this organism.

William Ewart advocates the prone posture and the use of a special elastic abdominal belt. The latter is for the unduly protuberant belly and to aid respiration. He considers that rickets is largely due to suppressed respiratory action, and suggests that judicious recourse to artificial means to stimulate the partly suppressed respiration is

the line of treatment that is of great service.

For the copious perspirations Eustace Smith recommends painting the affected parts, e.g. head and neck, with lin. belladonna. If this is good I should recommend that it be used with great caution, as the child may rapidly get belladonna poisoning. Recently I saw an adult who had marked atropism through frequently painting his joints with a weak solution of this preparation.

Probably the cold sponging, already recommended, would help this profuse sweating. In any case, one finds that under treatment the child improves and this troublesome sweating stops.

The limbs being weak and the bones soft, the child must not be allowed to walk early. Often these patients are fat and flabby, and therefore if they walk early their weight causes the limbs to bend and deformities result. Once the bones are strong the child may stand and walk a little, but he should not be allowed to run about constantly, as he is apt to do. To prevent them from running about a pair of light splints, projecting beyond the feet, will be found useful in out-patient practice, where often the parents will not see that the child is kept off his feet.

But the confinement of the limbs cannot be good

for the muscles, which are apt to atrophy and get flaccid. In cases of actual deformities, suitable splints are very useful.

If the joints are slack some use firm bandages. These are useful, but much more can be gained by proper massage regularly done, and cold sponging of the parts.

For the various deformities that require surgical treatment the surgeon is frequently required. But usually early cases do not require the surgeon, as even unsightly deformities become wonderfully normal under treatment.

Surgical Treatment.

Serious deformities of the bones of the extremities may require operative treatment. If the bone is still soft it may be fractured and then set in a good position. But frequently the bone is better divided with an osteotome and then set properly. Or it may be necessary, as in the case of the femur, to remove a wedge of bone so as to get better apposition of the bony ends.

Spinal curvature of various types are best treated by appropriate gymnastics, e.g. such as are recommended in Roth's treatment of spinal curvature. It may be necessary to use spinal supports, such as a plaster jacket or poro-plastic, but usually the

spine is better treated with massage, gymnastics and electricity, aided by cold sponging of the parts.

Even severe cases of knock knee can be greatly improved without resort to osteotomy. A firm splint being placed on the outer side of the thigh and leg, an elastic bandage may be used to bind the limb to the splint, and in this way tend to correct the deformity. If the child is young, this treatment with massage and suitable splint is quite successful.

Much outward curvature of the tibiae can frequently be corrected by an internal splint and elastic bandage. In young children this curvature can be greatly remedied without resort to osteotomy.

SUMMARY OF CONCLUSIONS.

1. Glisson was the first to describe rickets.

2. Cases of rickets occurred in ancient times.

3. Uncomplicated rickets is not a fatal disease.

4. Rachitic skulls are frequently larger than normal, but this is not universal.

5. Parrot's nodes are syphilitic and not rachitic in origin.

6. Craniotabes is more common in syphilitic children, but it also occurs in rickets, and in a few apparently healthy children.

7. General tenderness of the body is frequently seen in rickets, but if marked, strongly suggests scurvy.

8. Enlargement of the spleen is not common in rickets. When there is a much enlarged spleen, it strongly suggests syphilis.

Aetiology.

9. Deficiency of lime salts in the diet is not the causative factor in rickets.

10. Diathesis has very little if any influence in inducing rickets.

11. Rickets is not congenital.

12. Heredity may have some influence in a few cases.

13. There is insufficient proof that lactic acid is the causative factor. I do not think it is.

14. Disease or deficiency of the supra-renals may be of some influence in causing rickets, but at present there is insufficient evidence.

15. The thymus has not been shown to have any important influence in rickets.

16. I do not believe that rickets is infectious. There is ample evidence to the contrary. But in view of Artom and Agnese's experiments, it may be shown that rickets is caused by some organism resembling the Diplococcus Osteomalaciae.

17. Want of fresh air and sunshine are of great importance in pre-disposing to rickets.

18. I am not satisfied that there are sufficient proofs that rickets is due to respiratory insufficiency and inflammatory conditions of the lungs.

19. Climate is of importance. In general, damp cold climates predispose to rickets, but there are exceptions. Warm countries are not necessarily free from rickets: e.g. it is prevalent in Egypt.

20. Faulty hygiene is of considerable importance in predisposing to rickets.

21. Lack of sufficient exercise is a very important factor in experimental rickets in animals. But I doubt if it is of such importance in children. Exercise means much more to a young animal than to a very young infant.

22. Syphilis may predispose to rickets, but it is not the chief causative factor.

23. Rickets is due to a dietetic error, and is predisposed to by faulty hygiene and various other conditions mentioned.

24. Starvation does not cause rickets, but a child who is suckled on inefficient milk is weakened, and therefore liable to become rickety if he is fed on farinaceous food.

25. Deficiency of fat in the diet is of importance, but it is not the prime cause of rickets.

26. Over-feeding is not the chief factor in causing rickets.

27. I think that feeding a child on more starch than he is able to assimilate is probably the most important factor in the aetiology of rickets, but other predisposing causes, already stated, are also of great importance.

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28. There is nothing distinctive in the blood changes.

29. Cod liver oil, preferably one that is not over-refined, and attention to the diet and hygiene of the child are the chief points to bear in mind in treating rickets.

30. Phosphorus is not essential in the efficient treatment of rickets. It is, however, useful in some cases. It must be used with caution.

31. Pure olive oil may be of use to replace cod liver oil in certain cases, for instance in hot weather.

32. The maritime treatment of rickets, accompanied by massage, is of great value in treating rickets.

33. Calcium salts are generally said to have very little influence in the treatment of rickets, but I think that some cases seem to derive benefit from the lactophosphate of calcium and iron.

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