Rickets

A consideration of the Symptoms, Morbid Anatomy and Etiology.

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by

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I

Rickets

II

Synonyms — English, Rickets;
Latin, Morbus Anglicus, Articuli Duplicati;
German, Die Englische Krankheit
Doppelgliederder Zwiewuchs;
French, Rhacitisme, Maladie Anglaise;
Italian, Rhacitide,

History — The name primarily derived from the Old English verb "wrikken", to wrist or twist away, arose no doubt from the application of a term commonly applied in the County to inanimate objects, to a disease whose most striking characters are contortion & deformity. The ill formed child with the soft & loose-jointed framework of its body bent and giving way was called rickety, just as a dilapidated table or chair was styled, & the condition received the name of "The Rickets". Dr Glisson, who first described the affection in the 17th century, proposed the common term "Rickets" into "Rhachatis", partly on account of the similarity of the sound, partly as a derivation from the ~~Asm~~ Greek "ῥάχις", a spine, or

the ground that the dorsal spine is one of the first parts attacked, This, rather than Trousseau's derivation from the Norman word "riquets", appears to be the correct origin of the English name, Rickets was recognised on the Continent soon after Glisson's description of it in England, when it was supposed to have spread; but it has no doubt existed in Europe from early times as an accompaniment of civilisation, & it is now becoming common in the younger countries of America and Australia.

Description — Rickets is a general disease of early childhood affecting the nutrition & development of the whole of the growing organism.

The most marked physical changes are seen in the bones. Ossification, which is still in active process, is both retarded and perverted.

The bones grow irregularly, and, remaining largely cartilaginous and soft, yield under traction & pressure, thus giving rise to various distortions & deformities.

The evolution of the teeth is delayed, their structure is fragile, imperfect, & subject to early decay. This striking affection of the osseous skeleton has concentrated attention upon one special part of the disease, & led to too narrow a view of its pathology. Rickets has until recently been regarded as chiefly a disease of bones; it has been thus classed in the text-books, & its etiology & pathology considered almost entirely in this relation.

The effect of growth & nutrition is, however, by no means limited to the bony framework of the body; all the chief structures are involved.

The muscles are wasted & remarkably enfeebled, the ligaments are relaxed, the mucous membranes exhibit an abnormal tendency to catarrh. The brain is functionally backward, while the reflex nervous system shows increased irritability, so that various forms of spasm & convulsion are readily excited. The blood is impoverish-

ed,

its red corpuscles being diminished to such a degree that in some cases the anaemia is well marked.
Lastly, in some cases there is fibroid enlargement of the liver & spleen, & lymphatic glands.

Symptomatology—

The appearance of a child suffering from well marked rickets in its ordinary form is distinctive.
The square forehead with rounded boss-like projections on the frontal bone, the enlarged ends of the long bones—the deformities produced by the giving way of soft & yielding bones, such as the contracted chest, the deformed ribs, the curved spine, the protuberant abdomen, the bent arms & clavicles, the bowed legs, or knock-knees, the yielding ankles, the defective decaying teeth, the stunted growth—are features which cannot be mistaken. In many instances the child is plump or even fatter than normal, others again are thin, wasted & puny.

In addition to these peculiarities of out-ward form, the rickety infant is pallid, flabby, & feeble, Muscular debility is indeed, one of the most constant & characteristic features of the disease, although little stress has been laid upon it. In severe cases the child, at two years of age or later, is unable to stand or walk, or even to sit up; if it has been able to walk it has lost its power. Such cases are not infrequently mistaken for paraplegia, Sir W. Jenner, relates an instance in which the patient, a girl of six could not change her position in bed or lift her arm an inch without assistance.

The feebleness of muscle & softness of bone interfere with the action of respiration, & the lower portion of the chest falls in laterally with each contraction of the diaphragm; the feeble intercostals are unable to lift the soft ribs, which give way under the pull of the phrenic contraction aided by the external atmospheric pressure. This

inefficiency of the respiratory mechanism. greatly increases the danger of the pulmonary diseases to which rickety subjects are extremely prone.
The affection of the bones in severe cases is sometimes accompanied by marked tenderness, so marked that the child cries when handled, a sign possibly of the commencing scurvy with which rickets is occassionally associated.
Another prominent symptom of rickets is profuse sweating, especially of the head, & during sleep; this is evidently accompanied by a sense of heat, for the child throws off its bed-clothes at night & lies uncovered regardless of the cold; There is, however, not only no pyrexia, but the temperature is, as a rule, sub normal, unless there be concurrent scurvy or some inflammatory complication, such as broncho-pneumonia.
The abdomen is prominent owing partly to depression of the viscera, partly to the gaseous distension of the intestines & the feeble condition of the abdominal wall

Moreover, the bowels are liable to be relaxed, the stools being loose & offensive; sometimes, in the absence of biliary colouring matter, almost white; sometimes green, sometimes of darker colour, & slimy.
There is a tendency to bronchial & laryngeal catarrah, so that bronchitis & laryngitis are frequent complications.
Nervous symptoms form a marked feature of the disease, Sir Wm Jenner first noted the exceptional tendency to convulsion, &, a close dependance of infancy upon rickets was subsequently established by Dr Gee.
Reflex excitability is exaggerated, & probably the cerebral control of the imperfectly developed higher centres is also defective, so the various forms of spasm are readily set up by slight causes of irritation.
Laryngismus stridulus, tetany, and general convulsions stand in special relation to the richety state.

Laryngismus Stridulus — is rarely met with apart from rickets, of which, although not universally present is a common symptom.

In this condition reflex apparatus of the glottis so hypersensitive that spasm is excited by slight stimuli; not only by crying or laughter but even by a breath of cold air, sudden movement, or the emotional disturbance produced by fright, anger, or the vexation of cross purpose, such as the refusal or removal of a toy.

Attacks are especially apt to occur on the child just waking from sleep, particularly in the early morning.

The laryngeal spasm is marked by a sudden arrest of respiration, which lasts usually only a few seconds, & as suddenly ends with a long crowing inspiration as air is again drawn through the narrowed glottis.

The spasm varies in intensity; sometimes there is merely a slight crowing sound with each inspiratory

effort for a brief space; & the muscles of respiration may remain fixed until the child is cyanosed; occasionally death from actual asphyxia occurs before the spasm is relaxed.

An attack of spasm of the glottis is not infrequently the precursor of a general convulsion, & must always be regarded as a significant indication that the condition of the nervous system has become unstable, & is one in which convulsions would readily be set up.

Tetany — is a curious state of painful muscular contraction, & a tonic spasm, chiefly of the hands & feet; it is also closely & especially associated with rickets. In these cases laryngismus is a constant accompaniment, & tetany often follows an attack of diarrhoea, to which rickety subjects are prone,

It is true that tetany occasionally arises in adult life as a sequel of exhausting diseases, as in women during lactation, & as a sequel to diarrhoea or typhoid fever.

Such cases are, however, comparitively rare, & tetany is especially an affection of early childhood & of the rickety state. (In the "Brit. Med. Jour, of May 1st, 1897. epitome (350. pp72)) Kassowitz, at the Wiener Medicinis-cher Club (Neurol. Centrabl., March 1897.), showed a child with latent tetany & rickets, in whom complete recovery followed the administration of phosphorus. In reference to this case he discussed the relation of tetany in childhood to rickets. The gastro-intestinal origin must be considered improven; Kassowitz has never seen tetany occur with chronic gastro-intestinal catarrh in the absence of rickets, & the seasonal variations of tetany are exactly the opposite of those of gastro-intestinal disorders, tetany being most frequent in the winter, while the gasho-intestinal troubles are most common in the Summer. The frequency of tetany does, however, vary directly with the frequency of rickets. The close relationship of these two conditions is explained by the rachitic

II affection of the skull, which produces hyperaemia not only of the cranial bones, but also of the meninges & cortex, & so causes undue excitability of the motor centres & a tendency to convulsive movement. The exciting cause of the tetany spasm is some noxious substance entering by the respiratory tract from the foul air in the dwellings of the poor.

In tetany the thumbs are first affected, being adducted & drawn into the hollow of the palm, with tips pressing forcibly against the middle phalanx of the third finger, & even driven into the skin. The fingers are drawn together & overlap, & the palm is hollowed, so that the hand becomes cone-like "the accoucheurs" hand of Trousseau. The wrist is slightly flexed, & in severe cases, when the muscular spasm is great, the back of the hand & wrist may become purplish, swollen, & even slightly oedematous, from the pressure of

the contracting muscles on the venous circulation.

The feet are affected in like manner, but usually in less degree, the toes are addducted, flexed, & overlapping, & the fore part of the sole of the foot is hollowed & concave by the drawing inwards of its borders; the dorsum is arched, & in severe cases swollen, congested, & shining from pressure, like the back of the hands.

In some cases the spasm is said to extend to the muscles of the trunk, causing slight opisthotonus, & occasionally those of the jaws, causing trismus; if this does ever occur, it is rare, & beyond the experience of Dr Cheadle. (Vide Allbutt. Vol iii, 1897.).

The muscular cramps are painful, & when extreme acutely so; this we learn from the statement of adults, & it is noted, accordingly, that a child who suffers from severe tetany cries incessantly, & screams when handled.

The tetanoid state persists during sleep, & even under chloroform. It continues a considerable time, often for weeks, & is

apt to recur, Remissions occur from time to time. These, however, are not complete, some degree of rigidity still remains.

The spasm can be excited afresh by a pressure upon the main artery or nerve – probably on the latter – for the motor nerves are in a state of hyperexcitability, not in the limbs only, but in other parts also. Thus, as first pointed out by Dr Barlow, the sharp drawing of the forefinger over the skin in front of the ear, where the facial nerve emerges, in a child suffering from tetany is followed by the contraction of the facial muscles.

Percussion of a muscle causes alike contraction in it. The electrical excitability of the nerves of the most affected parts is also increased both to faradism & Galvanism; & as Erbe has shown, the mode of reaction to the voltaic current is reversed, contraction being first excited by positive instead of by negative closure, & a prolonged "tetanus" contraction following both anodal & cathodal closure – the only condition,

according to Allbutt Vol iii. 1897. in which anodal "tetanus" has been observed in man. It is said this increased neuro-muscular irritability may sometimes be found in rickety children who are free from attacks of tonic spasm. A case I have, at present, under observation shows the association of tetany and Laryngismus Stridulus with Rickets. L. S. age 4 years & 3 months. Male Sex;. Present condition, pale, anaemic, feeble & very thin, chest bulging, with enlarged joints at the junction of the cartilages & the ribs, weight 26 pounds; History given by the Mother, that she was only able to give the breast for the first six weeks, afterwards various artificial foods were given. At three months old, diarrhoea set in, accompanied by a "convulsion" ~~?set~~ in which the hands & feet were very much drawn; this, in all probability, would be the commencement of Tetany, & there have been slight attacks occasionally since,

He was unable to walk until the age of two years & five months,
Laryngismus stridulus, first came on at the age of four months, but no other attack until three years & nine months old, which I witnessed, and two more since: These, along with the unmistakable signs of rickets, suggests the case, as one commencing with that disease; tetany & laryngismus stridulus following.
The symptoms of Rickets according to Sir William Jenner, Bart:, G.C.B. in his clinical lectures (1895), are—
"a general cachexia which very rarely manifests itself before the fourth month; usually between the fourth & tenth months. One case a boy was three years old before symptoms manifested themselves, and a girl age nine years. It is rare, however, for the general cachexia to first manifest itself after the child has passed its second year.
At the outset the child is dull and languid; its skin is hot; it is thirsty;

it is drowsy, or sleeps little; its appetite is lost; if it has begun to walk, it is taken off its legs; it lies about, is unwilling to play or to be amused.
The bowels are irregular, - confined, or more commonly relaxed - the stools being usually of a dirty brown or leaden colour, & most offensive, & peculiar resembling that of rotten half decayed meat.
In all these symptoms there is nothing diagnostic, they might arise from indigestion, from improper food, or from tuberculosis.
Commonly, however, there are certain symptoms present viz, profuse perspiration of the head, or of the head, neck, & upper part of the chest, especially when the child sleeps, or at the breast, or little increased exertion.
The superficial veins of the scalp are generally large & full, & sometimes the carotid arteries may be felt strongly pulsating. The abdomen & upper extremities at the same time are usually dry & hot.

The second symptom, the child kicks the bed-clothes off.
The third symptom, is general tenderness.
As the disease progresses, the child gets a peculiar staid & steady appearance; its natural lively expression is replaced by a pensive, aged, languid aspect; its face grows broad & square; & when placed upright, it sits "all of a heap", its spine bends & the muscles are too weak to keep it erect. Its head seems to sink between its shoulders, its face is turned a little upwards.
The general cachexia is sometimes very severe, at others extremely trifling.
The bone deformities commence. If the attack be attended with severe general symptoms, the softening of the bones usually precedes, & is out of proportion, for some time, at least, to the enlargement of the ends of the bone. The younger the child also, the softer are the bones.
At this time the appetite is often good, but the bowels are deranged; the stools

being either dark & fetid, or white, or the food is passed as it is eaten.
The muscles lose power & waste as the disease progresses; but the loss of power is infinitely greater than can be accounted for by their diminished size.
The abdomen is larger than natural, because:- 1st the chest is smaller & the diaphragm more depressed than in health
2nd, The liver & Spleen are often larger than natural.
3rdly. The capacity of the pelvis is diminished.
4thly The muscles of the abdomen & intestines are less powerful even then they are in their normal condition; & moreover, derangement of the digestion is always present to favour the excessive formation of flatus.
Writers state that the intellect of the rickety child is precocious, but this is an error, for thrown necessarily much in the society of adults, catch their expressions, phrases, & ideas, hence is thought, to have larger intellect.

They are nearly always deficient in intellectual capacity & power. They are not idiots, they offer no signs of idiotcy, they resemble rather children of low intellectual capacity & power much younger than themselves, & it retrogrades as the rickety diathesis progresses. The teeth are always retarded in their development, & fall from their sockets very early; or decay early.
The back, arms, & sides of the face are very often covered with downy hair. The anterior fontanelle is frequently opened till the child is three or more years of age.
Thomas Barlow, M.D. F.R.C.P. & Judson, S. Bury, M.D. M.R.C.P. in the "Cyclopaedia of the diseases of Children. Keating. Vol ii." state that the earliest manifestation of rickets consists in beads at the junction of the ribs with the costal cartilages, (ros-ary), & may be detected at three months, may be identified as early as one month, & we have found them on dissection of still-born children.

The free margins of flat bones of the skull are unduly soft, & round spots of local thinning may be detected on occiput & parietals & even exceptionally on the frontals, to these Elsässer, who first discovered the condition, gave the name of craniotabes, such spots are most frequently met with about the third month; they may be found up to the eleventh, but do not appear after that period.

Symmetrical lenticular swellings form on frontal & parietal bones, in front & behind the anterior fontanelle,

these masses of soft, vascular, bony growths shine through the thin pale scalp, & in some cases cause a characteristic pale-bluish-looking swelling. There is no local heat or tenderness over these areas.

Two types of rickety skull are given. The first, presenting a broad, square forehead. crown flattened, the antero-posterior tracing of the outline of this form of skull is polygonal,

whilst that of a hydrocephalic skull is almost circular.

The second variety or type of rickety skull is dolio-cephalic, the frontal region, not very broad, is prominent in the middle portion, the top of the skull is flattened, upper part of occiput projects considerably.

Whatever shape, the head is generally larger, than a head of healthy child of same age.

Fleischmann has pointed out the alveolar border of the upper jaw tends to assume a beak-like shape, the antero-posterior axis being lengthened whilst the outline of the lower jaw becomes somewhat polygonal, & its anterior surface turned slightly inward. The bone has lost its normal curve, the incisors are quite in a straight line; then at the situation of the eye-teeth the jaw forms a sharp angle & bends abruptly backwards.

Baginsky describes in addition an occassional want of symmetry between

the two halves of the bone which gives the appearance of one side being higher than the other, the effect of this delayed development is late teething. Among the earliest symptoms of rickets according to A. H. Tubby, M.D. Lond., F.R.C.S. Eng. in his book on "Rhacitic Deformities." are tenderness of the bones, the child crying out when moved, disordered digestion, sweating of the head, & bronchitis, the child, if it has commenced to walk is "taken off its feet" & some bending of the bones may be seen at this stage; he quotes Dr Gee, who states that a child who is not idiotic & weakened by some recent disease, & who cannot walk at the age of eighteen months, is either richety or paralysed. The complexion is of a peculiar earthy tint. & the child sits in a heap. At this time, too, "beading" of the ribs & enlargement of the radial & ulnar epiphyses are found. The spleen & occassionally the liver are felt to be enlarged. & the abdomen is distended

At the annual meeting of the British Medical Association held at Edinburgh, 1898. Wm Tubby spoke of the occurrence of a pad on the dorsum of the foot in rickets (Vid Brit Med Jour Oct 15th. 1898. pp 1137-38). In the course of examining a large number of rickety children, he noted a peculiar appearance on the dorsum of the foot, & occasionally of the hand, but more often seen on the foot, this appearance has not hitherto been described.

It is well known that in infants the dorsum of the foot is somewhat prominent & the soft tissues are fuller, but the appearance & feel of this part are totally different in healthy & rickety children.

In the latter, the soft tissues are decidedly thicker than in healthy children at this spot, & the swelling may be a flattened elevation or dome-like. Its colour is that often seen in the skin elsewhere in rickets, & may be compared to that

which would be produced by the injection of white wax, slightly tinted to a yellowish shade, beneath the skin so as to give it a distinct tint & a semi-transparent look.
As to the consistency of the swelling, if in a healthy child the skin of the dorsum of the foot be pinched up between the fingers, it can be moved independantly of the subcutaneous tissue; but in many rickety children skin & subcutaneous tissue are quite inseparable, & only a more or less thick fold composed of both can be made. It appears in many cases as if solid oedema were present in the skin & subcutaneous tissue. The pad, however, is not due entirely to changes in the subcutaneous tissue, but in some cases originates in swelling & thickening in the neighbourhood of the epiphyses of the metatarsal bones; hence the pad is more frequently seen rather towards the anterior than the

posterior part of the dorsum. When the bones are so affected, distinct beading may be felt about the epiphysial lines, & doubtless the perichondrium & periosteum are thickened as well. But to return for a moment to the consistency of the swelling. In early cases of rickets it is often semi-fluid, In cases of a few months' duration it is semi-solid, & in those of more than a year's duration it is hard and resistant.

I have tabulated 100 cases of rickets, but it is somewhat difficult to fix the date of onset of the attack, so it has been customary to ask the mother the following questions; When did the child's head begin to sweat at night persistently? When did he cry as if he were tender all over? When did he appear to be weak in his back & to go off his legs? When did the motions become evil-smelling & the abdomen to swell? By noting the answers to these questions—

Some, of course, being in the negative — it has been possible to fix the date of the onset of the attack with some degree of accuracy. Taking one hundred tabulated cases of rickets, 86 had dorsal pads & 14 none. The 86 have been divided into three classes:

(a) Those children in whom the pad was marked, but was semi-fluid, & no affections of the bones could be felt. Of the 86, 20 cases came under this category, the average age being 17 months & the duration of the rachitic attack 2 months. This statement bears out another observation, namely, that before the signs of rickets are seen or felt in the bones, the skin & subcutaneous tissues generally are affected, being puffy & of a tallow-candle appearance.

(B) Those in whom the pad is well-marked, nearly dome-shaped, and solidly oedematous to the touch. Of the 86 cases, 49 presented this appearance; the average age was

24 months, & the average duration of the attack was 11¾ months.
(c) Those in whom the thickening was mainly bony, the swelling being of a firm, hard, resistant character, & beaded. Of the 86 cases, 17 came into this category, their average age being 4 years, & the duration of the disease 24 months.
Although only 100 cases have been tabulated, yet a very large number of other cases have been examined and have appeared to me to bear out the preceding observations, & to indicate that if cases be watched over a long period the pad goes through three stages, namely, semi-fluid & confined to soft tissues, semi-solid with some implication of the bones, & firm, resistant & irregular, due entirely to the fact that the bone lesion is the last to disappear.
In conclusion the following points are advanced.
1 In a large proportion of cases of rickets the feet show definite dorsal

pads, & in a smaller proportion of cases the same appearance is seen on the backs of the hands.

(2) This pad is of varying consistency and origin.

(3) If the disease be of recent duration – that is, two to six months – the thickening is subcutaneous and is semi-fluid.

(4) If rickets have existed from six to eighteen months, the pad is best marked, & is made up of thickened subcutaneous tissue & periosteum & overgrown epiphyses.

(5) At a period of eighteen to thirty-six months after the onset of the disease, the subcutaneous pad disappears concurrently with the general flabbiness & undue pallor of the skin, & leave the bony changes well-apparent to the touch.

Such are the symptoms & physical characters of rickets in its full and complete expression.
All these features are not, of course, present in every instance, The disease is met with in every degree, from the smallest indications to the extreme forms.
Slight cases of rickets are liable to be overlooked, This is especially the case with the children of the better class, amongst whom slight cases are by no means uncommon. Now & again the signs are extremely unobtrusive; A little bending of the ribs, a prominence of the frontal bone, or soft edges of the flat bones, may be the only manifestation of rickets as far as the bony skeleton is concerned. Indeed the rib-beads constituting the earliest signs met with in the foetal state may be found alone in the early stage.
The concurrence of head sweats, or Laryngismus, or convulsions, or a tendency to catarrh, will be sufficient to establish

the existence of rhacitis.
In a more pronounced case there may further be some enlargement of the wrists, the girdle contraction of the chest, the bending of the long bones of the limbs. In the most complete & severe form the distortion of the skeleton may be extreme, & the whole series of other symptoms affecting the muscles, mucous membranes, & nervous system which have been described may perhaps be present; but the concurrence of all in the same subject is extremely rare.

Morbid Anatomy.—

Sir W. Jenner describes the bone changes as exhibiting extensive preparation for ossification & imperfect performance of the process,
Dr Cheadle's (Vide Allbutt Vol iii. 1897) observations agree in the main with the opinion that, as shewn by Kassowitz, the rickety process is not merely a defective ossification but is accompanied by a hyperplasia.

Sir Wm. Jenner, in his Lecture iii, 1895, says that, Mr Paget in propounding his theory of inflammation dwells on the fact that concerned in the process of nutrition, are four agents - any one of which being deranged at a particular spot, derangement of the others necessarily follows; & he points out that, when inflammatory action is established in a part, all four are in an abnormal condition - that inflammation is a disease of nutrition, rickets then is a disease of nutrition. In rickets, moreover, there is necessarily no pathological exudation or new formation; there is, so far as we know, merely a change in quantity & arrangement of normal structures & secretions. This is true not only of the bones & muscles, but of the secretions of the skin & kidneys. Rickets, then, is essentially & purely a disease of nutrition, not of one part only, but of the whole body. But if we admit this as proved, we have advanced a very little way on the road to

the discovery of its itimate nature. But, as little as we have progressed, we certainly are in advance of those who still regard rickets to be merely a chemical abnormity of the bones viz, a deficiency of the earthy salts.

Dr Eustace Smith in his second edition on "Diseases of Children" says the changes in the bones are affected in three ways, growth retarded & irregular ossification of parts still remaining cartilaginous interfered with, & bone already ossified is softened.

Dr. G. Sims Woodhead, in his "Practical Pathology" 3rd Edition, says- Rickets must be looked upon as a disease of an essentially constitutional character, the result of malnutrition, It is most frequently observed in badly nourished children from one year old & upwards, the period during which the bones in which the changes associated with rickets are most marked, are being most rapidly developed, These changes are best seen in the long bones, especially at the point of jinction with the various cartilages.

Mr. A. H. Tubby in his book on "Rachitic Deformities" say the bones in severe cases pass through three stages; (1) Stage of congestion. (2) stay of softening; (3) Stage of sclerosis. It is in the first and second stages that the deformities occur, which in the third stage become fixed. The lesions which arise are as follow—

1 The Cranium (Vide Allbutt Vol iii).

The skull well exhibits the three great features of perverted ossification; viz, delay, atrophy, & hyperplasia. The margins of the bones remain membranous; spots of thinning, the "craniotabes" of Elsasser, are perceptible in the parietal & occipital bones; & small bosses or swellings form symmetrically on the frontals, on the parietal & occipital, & even on the temporal bone.

The atrophic lesions of craniotabes consist of wasting & thinning of the inner table, so that the inner surface of the bone, when the dura mater is removed, presents shallow depressions;

some of these, in extreme cases, extend through to the pericranium, while the bone around is thin & elastic. These thinned areas have been variously attributed to pressure of the enlarged brain, & to that of the pillow; but they are probably patches of bone which have grown imperfectly. The elevations or bosses are developed chiefly from the outer table of the skull, & consist of red highly vascular, spongy material which yields to pressure. These bony growths, in some instances, become more or less completely absorbed; but in most they become organised, & remain as rounded projections or bosses which are characteristic, or they may spread diffusely into more general thickening of the bone; in either case they largely assist in giving to the rickety skull its special character.

The chief features of this skull are as follow: The forehead is broad, square, & projecting, the eminences on each side being prominent & thickened; the top is flattened so that the head looks square or in some cases oblong, the parietal & occipital protuberances being well marked; a groove runs along the site of the suture between the two halves of the frontal bone, & is continued along the vertex to the anterior fontanelle, or beyond it, to the occiput. In some instances there is a distinct want of symmetry, as if the head had been twisted askew, so that the frontal region projects on one side & the occipital on the other. The head generally is larger than normal, the face by contrast looking smaller; & the upper jaw is narrowed & elongated. The anterior fontanelle is large, & it remains open longer than usual; instead of being closed at eighteen or twenty months, it may be still more or less patent at two or three years of age; a slight opening may indeed be detected as late as five years.

(A child, I have had under observation for some time, shows quite an exception to this rule, N. D. female, now aged one year & seven months, with all the symptoms of rickets well marked, has had, to my knowledge, the anterior fontanelle closed the last four months, when I first saw it; & the mother stated, she had noticed, it closed for some time before, ~~before~~ then.).

The closure of the sutures, except that between the two portions of the frontal, is delayed also; & there is irregular thickening of the margins of the bones, notably in front of the anterior fontanelle; deep grooves, often mistaken for open sutures, are found in the temporal region & elsewhere.

The teeth come late; in extreme cases none may have appeared at ten or eleven months; they often come through in irregular order; they are fragile and deficient in enamel, subject to early decay, and fall out before their time.

Sir Wm. Jenner, in his lecture ii, 1895 says the rickety head is distinguished

(1) Anterior fontanelles remain open long time (healthy child, closes before end of 2nd year)

(2) Thickening of bones, just outside sutures (sutures deep furrows)

(3) Relative length of antero-posteria diameter. (In Keating Vol ii two types are given by Dr. J. Barlow & Judson. S. Bury. viz the polygonal, in its antero-posterior tracing with a broad square forehead & flattened crown. The second form 'dolicocephalic', in which the frontal region is not very broad, is prominent in the middle portion, the top of the skull is flattened, upper part of occiput projects considerably).

(4) By the height. squareness & projection of fore-head. The two first due to the affection of the bones, the two last chiefly to disease of the cerebrum.

In consequence of arrest of growth of the bones of the face & sinuses, the forehead as mentioned by Mr. Shaw seems to project more than it really does. Guerin supposed that the rickety deformities were developed from

below upwards, eg, inferior esthemities before trunk, But this is not the case.
Mr Suby remarks that the circumference of the head is often much increased. in some cases owing to hydrocephalus.
(In the discussion on rickets at the London Pathological Society in 1881, an opinion was expressed that rhactic heads are smaller than the normal. but extended experience disproves this)
With regard to the defective development of the bone, especially in those parts exposed to pressure, the result is more or less circumscribed areas which are yielding to the touch, & gives the impression of the so-called "egg-shell crackling"; this condition known as craniotabes, is met with also in congenital syphiles.
In a case which came under observation at the Evelina Hospital, the whole vault of the skull was so softened, that pressure at any part produced an indentation.
That craniotabes is due to the effect

of pressure on softened bone is supported by two facts. It is generally seen in the occipital region in infants, i.e. where the head presses on the pillow; while in richetty monkeys the thinnest bone is met with in one or other parietal region, owing to the habit these animals have of resting in the sitting position, with one side of the head against a support. A prominent square forehead, with fulness of the lateral aspect of the frontal bones, obliquity of the upper wall of the orbits, & prominence of the eyes, are characteristic of richets. In the parietal regions the eminence on the central part of those bones is exaggerated, & taken with the enlarged frontal eminences, the hat-form appearance results. This is, however, better marked in congenital syphilis.

In the face the chief error of development is in the lower jaw. Makins observes (vide Treves System of Surgery, vol i p 368) the "development of the outer wall of this

bone being defective, interferes with the acquisition of the proper arch formed by the body, the incisor teeth being arranged transversely, & from them the remaining teeth diverge obliquely backwards, the alveolus being somewhat inserted posteriorly so that the teeth point inwards, the lower margin of the body is inverted (Heischmann). The teeth appear late, & are defective in enamel, readily becoming carious".

The Thorax

In the first place the shape of the chest (which is "constant, Vide Sir W Jenner Lect) is modified by the softness of the rib bones & their yeilding~~ing~~ to pressure, & the great determining cause of the thoracic deformity is atmospheric pressure; aided by the elasticity of the lungs. Rokitansky has maintained that this deformity of the thorax is the consequence of want of power in the inspiratory muscles, but there is no correspondence between

the points of insertion of the muscles of inspiration attached to the outer surface of the chest wall, & the points of recession. Again the diaphragm is said to cause the circular recession by its direct action — by drawing in the receding parts at each contraction, but the line of recession does not correspond to the point of attachment of the diaphragm. But it does correspond to the upper margin of the liver, spleen, & stomach; & is produced — as the longitudinal furrow is — by atmospheric pressure; the parts of the parietes below being prevented receding by the organs mentioned.

One depression runs transversly from the lower end of the sternum across the chest on each side to the posterior margin of the axilla, about the line of attachment of the diaphragm.

When any obstruction to respiration occurs, & in aggravated cases under ordinary conditions of respiration the chest is further drawn in along this line with each inspiration.

This is a result of the yielding of the ribs (which are softer than the cartilages) in the direction of least support. Another depression runs obliquely down the front of the chest along the line of junction of the ribs with the cartilages; & at the bottom of this depression, or outside it, are seen the "beads" or enlargements of the growing ends of the long bones. which collectively form what is called the "rosary" & which is the earliest of all the bone changes; they have been recognised at birth, & in some instances even in the foetal skeleton. The beads are most marked in the lower ribs, & examination after death shows that they are more prominent on the inner than on the outer aspect of the bone. A slight degree of beading, however, may be abnormal, They are generally symmetrical on the two sides, & no vestige of them remains in adult life.

Posterior nodosities in severe cases form near the angles of the ribs

(which appear to arise from partial fractures) they are less symmetrical, vary in position on sucessive ribs, sometimes present on only one side, angular rather than nodular.

The result of the giving way of the soft bones of the chest under atmospheric pressure is the projection of the sternum forward. This projection is rounded, not acutely angular as in the true 'pigeon' breast, which may arise independently of any rickety softness of bones, it is rare in children under twelve months old, except subjects of atelectasis & congenital malformations of heart, & most commonly found in children over two years of age, who have suffered from whooping cough, of repeated bronchitis, or of post nasal adenoid growths. When these obstructions to respiration occur in rickety subjects, the sternal protrusion becomes more extreme.

In addition to these changes in the thorax proper the clavicles are often thickened and more curved than normal; in some instances they present "green-stick fractures" or imperfect splintering with consequent thickening of the shaft.

Sometimes a subluxation of the inner end of the clavicle occurs in rhachitic children. With displacement of the bones, there is frequently some scoliosis, & inasmuch as the subluxation is on the ~~concave~~ "convex" side of the scoliotic curve, it is probable the scoliosis may be the immediate cause owing to the forward & upward tilting of the shoulder on the convex side, thus pushing the inner end of the clavicle out of place. (Vide. Tubby "Rhachitic Deformities, 1896). Gilbney (Int Clinics vol iv. 1893. p 239) in a clinical lecture, alludes to several cases, & says "Lately we have a way of curing these that I is very good, his attention was first called to it by G Stimson who had injected alcohol, two or three injections setting up inflammation around the articulation, then binding the parts, with sucessful results.

The scapula in severe cases is curved, so that the posterior aspect is convex in conformity with the convexity of the back.

The spine. The back is rounded, owing to the relaxation of ligaments & the inability of the enfeebled muscles to keep it erect; sometimes there is a slight lateral curvature, & the forward lumbar curve is increased. In the early stage both these curves can be made to disappear by traction; but when the child begins to sit up, permanent deformity may be established if the rickety condition persist unrelieved. The softening of the vertebrae & weakening of the muscles & ligaments was suggested as an explanation of the cause of rhachitic torticollis. Phocas of Lille reports in the Revue d'Orthopédie January 1894. three cases of torticollis in rickety children of ten, fifteen, & eighteen months. The head was strongly inclined to the left, the chin was elevated, & the face turned a little to the right. There was no contraction of the sterno-mastoid, & the head

was easily replaced, but resumed its vicious attitude when support was withdrawn. Later, the head was thrown back, & this backward tilting was harder to prevent than the lateral. Pain was not severe, but the children were peevish & resisted examination. The lateral deformity lasted about three months, & the posterior also disappeared after a time under appropriate general treatment, & the use of a rubber collar to keep the head in position.

The pelvis, Sir Wm. Jenner, says in Lecture ii the rickety pelvis is not constant in shape, said to be oval, but more frequently triangular.

Mr Tubby says, the conjugate is decreased owing to the prominence of the sacro-vertebral angle, In ~~many~~ consequence of the ~~iss~~ thrust inwards of the head of the femur at the acetabulum on each side, the lateral aspect of the pelvis is flattened, & the pubic arch is diminished. The tubera ischiorum approximate unduly. The ventera ilioum expanded by weight of viscera.

Dr Cheadle states that the flat bones of the pelvis are thickened irregularly as elsewhere; when the body is supported erect on the limbs, the pelvic arch, compressed between the weight of the body acting downwards from above through the spine & the upward resistance of the thigh bones, gives way; thus a a general result the pelvis becomes narrowed by the pushing forward of the sacral portion towards the pubes, & is more shallow.

The long bones of the limbs - Bones of the upper extremities - The lower ends of the radius & ulna are thickened, both the epiphyses itself & the junction of this with the shaft; thus is constituted the enlargements of the wrist, which is one of the earliest signs of rickets. Similar but less pronounced changes may be found at the upper end of these bones & at the upper & lower end of the humerus, being more marked at the latter. The changes observable are at first confined to these; but later, as pressure comes into play, & the child,

in squatting or crawling, begins to lean its weight upon its hands, the shafts of the bones of the upper & fore-arm become curved & twisted. The force of gravity appears to be aided & modified by the support & traction of the muscular attachments, which are very well marked, being drawn out during the soft stage. In Fig 138 rhachitic deformities. Subby. the radius is seen to be prominent at the upper part, More frequently both bones are bent just above their inferior extremities.

In the Boston Medical & Surgical Journal. Vol i, 1897, two cases are reported, which were read before the Warren Club, Nov 3d, 1896 by J. S. Stone M.D. Boston, showing the cause of bowing of the left forearm in rhachitic children. Case I. S. T. aged 14 months, of Jewish parentage, was brought to the out-patient clinic at the West End Nursery on July 8, 1896. She was breast fed, but also had always been given whatever she wanted to eat, including tea, coffee, cake & candy,

She had never had any illness, except a cold during the preceding winter. She was in fair general condition, though presenting well marked evidences of rickets of a moderate grade. The radial epiphyses were considerably enlarged. The anterior fontanelle admitted a finger. There was no marked rosary or bowing of the legs. The left forearm presented at the junction of the upper & middle thirds of the shafts of both the radius & ulna, an angular backward bowing of about forty degrees, a little greater in the radius than in the ulna. The motions of the elbow were normal, excepting that the deformity slightly increased pronation & limited supination. The right forearm was normal. The mother associated the deformity with a fall from a chair four months previously. This gave rise to no symptoms, for several days, when the deformity was observed. The child was just beginning to walk, careful enquiry showed great improbability of green stick fracture, which the deformity suggested.

Case II M. E. C., aged 2½ years of Irish parentage, was brought to the out-patient clinic at the Childrens Hospital in the service of Dr Lovett, through whose courtesy the case is reported. As a baby she had been healthy, & was breast fed. At nine months she had diarrhoeal troubles, followed by whooping cough, & for a year she had been rachitic. She had a square head with a closed fontanelle. There was marked retraction at the sides of the chest, a considerable rosary, a prominent abdomen & slightly enlarged spleen. The radial & tibial epiphyses were markedly enlarged. There was slight anterior & outward bowing of both tibiae, at the junction of the middle & lower thirds of the shaft. The curves of both clavicles were exaggerated. There was the long rachitic curve of the spine. The left radius & ulna were bowed backwards, the deformity causing a sharp angle of about forty degrees just about the middle of the shafts of the bones, There was no limitation of motion. The right radius & ulna were normal, The deformity was

first noticed as the child was beginning to walk. There was no history of any injury whatever.

Photographs were produced, showing the nature & cause of the deformity. Each mother was right-handed, & in helping her child to walk, grasped the left hand & wrist. The bones of the fore-arm bent exactly where her little finger pressed against them. The "green-stick" fractures previously spoken of is not uncommon in the bones, & in rare cases complete fractures have been met with. In severe cases thickening of the ends of the metatarsal bones & phalanges has occassionally been observed.

The bones of the lower limbs — In these the earliest & most characteristic change, often indeed the only one to be found in minor examples of the disease, is enlargement of the lower end of the tibia from thickening of the epiphyses & its junction with the shaft. In the more pronounced cases the upper end of the same bone, & both extremities of the fibula & of the femur, are similarly affected in

varying degrees. When the child begins to crawl or walk about the results of weight pressure begin to appear, as in the case of the arms; & the long bones become bowed. At first the tibia alone grows a little concave on its inward aspect; this increases if the child is allowed to go on walking & standing, & the femur becomes bowed in like manner. In some of the more extreme cases the tibia undergoes a forward curve just above the ankle, & the femur is arched forward in similar fashion. This is probably the result of pressure on the femur upwards & on the feet backwards as the child is carried in the arms.

Coxa vara (incurvation of the neck of the femur) in the majority of cases is due to rickets infantile & adolescent. In the latter the disease is said to be "local" in its manifestation (Vide By 'Royal Whitman', observations on, 'Bending of the neck of the femur' in Adolescence. Trans Amer Orth Assoc. Vol vii. pp 270–293.)

The rhachitic Attitude is an exaggeration & a persistence of the attitude of the infant when learning to walk. The rhachitic child stands with the feet wide apart, the thighs flexed, the knees bent, the back arched, & the shoulders thrown back, Much light is thrown on this by Arbuthnot Lane in Guy's Hospital Reports Vol xxix., p 32. But some of the lordosis is due to the prominent abdomen of rhachitic children, in addition to the cause given by Lane. Such are the general features of the bone affections & deformities in rickets. Some of these abnormal conditions dis-appear with time & growth. In adult life the beads on the ribs are no longer recognisable, the enlargements of the ends of the long bones undergo more or less complete convolution, & the deformity of the chest tends gradually to become corrected; but an antero-lateral depression below the nipples persists to adult life in some cases.

The curvature of the spine & of the long bones slowly straighten, & if moderate, disappear altogether; although in more pronounced cases they remain during life.
The contraction of the pelvis, however, when extreme, remains; & it forms not infrequently a serious source of difficulty & danger in parturient women. The bossing of the skull is toned down, although when the hyperostoses are large, they remain in a modified form, & are prominent during life; the square projecting forehead & thickened margins of the sutures also continue as permanent indications of the extinct fault of structural growth which gave rise to them. When the rickety condition is severe growth is defective & stature short. & the bones stunted & deformed, owing to the fact that ossification sets in so rapidly that the epiphyses unite at an earlier date than usual, & the child remains dwarfed.

Histological changes – The morbid changes in the more intimate structure of the bones consist essentially of modifications of the normal process of ossification; in excessive formation of cartilage & of the proliferating layer of the periosteum, in retardation of the development of bony tissue in these, & in perversion of the process. Bone is formed irregularly instead of by orderly advance of the ossifying column, & it is imperfectly calcified. With this perversion of the formative process there goes also an absorption of bone already formed. In the long bones these abnormal conditions are exemplified by the greatly increased vascularity of the cartilages – vessels invade the cartilage from the periosteum, & in severe cases enlarge so greatly that they look like haemorrhages or blood spaces; & also by the excessive development of the proliferating zone of active growing cartilage cells at the epiphyseal end. The cells are increased in number & crowded together; sometimes they actually replace the matrix.

There is also abnormal shallowness of the columnar zone where the cartilage cells are being arranged into columns preparatory to their evolution into the medullary spaces of the forming bone. Further these ~~are~~ spaces are themselves irregular, and some advance obliquely in the shaft, thus impinging upon & destroying adjacent columns.
In extreme cases this latter zone may be quite wanting, & spongy bone approaches the columnar zone of cartilage.
The bone formation takes place not only around the spaces but also above & below them, & small areas of bone may even be found in the unchanged cartilage.
The cut surface (which may easily be done with a knife, the bone being soft) is exceedingly red & vascular, the bony lamellae are thin & friable, & it is evident that rapid absorption is going on; The medullary portion of the shaft is more vascular, than normal, the interspaces larger. The marrow is relatively large in amount, red & gelatinous instead of yellow & fatty.

The formed bone itself undergoes changes; the vessels enlarge & new ones form, which press upon the septa between the medullary spaces & cause this partial absorption, so that the spaces open into each other, Thus the strength of the bone is impaired, & the slight fractures occur, already described. As the septa of the formed bone are eaten away fresh bone is formed which is deficient in lime.

This deficiency is due (according to Dr Cheadle Vide Allbutt Vol III, 1897) not to removal of that material as at first supposed, but according to the later observations of Hassowitz, to defective deposit; the result is that rickety bone contains only 32 to 52 per cent of lime as compared with the 63 to 65 per cent in normal bone.

Sir Wm Jenner in his Lecture on rickets says:- From the experiments of Lehmann, & Marchand, the animal matter of rickety bones, differs, in some cases, from that of healthy bone;

for, in some of their experiments, the bones yielded no gelatine in boiling.

The result of analysis of several observers, show that the bones of healthy children yield about 34 parts of organic & 66 of inorganic matter; & those of richety children yield about 79 parts of organic & 21 of inorganic matter.

With regard to a "deficiency" of the earthy salts, this view of the pathology of rickets is altogether erroneous (Vide Sect. III) & seems to him to be proved by the fact, that not only is there insufficient deposition of the lime salts in the growing extremities of the long bones, but there is an error in position of the small amount deposited there. The earthy matter is found in the cells instead of the matrix. And yet further, not only is there an insufficient quantity of the lime-salts & error in position of those present, but there is absorption of those deposited ere the disease began; for bones previously hard soften. The lime is taken up from from the well-constituted shafts

of the long bones & from the flat bones, enters the blood, & is thrown out of the system by the urine. The agents concerned in the nutrition in the bones not only do not take the lime from the blood, but they take the lime from the bones.

It is not probable that there is any lack of lime in the blood seeing that one secretion from the blood, viz, the urine was found in Marchands experiments, to contain six times its normal quantity of lime-salts. Again if it be a fact as shown by Lehmann & Marchand viz - that the bones don't yield gelatine on boiling, it shows some far deeper change in the nutrition of the bones than a mere want of lime.

These analyses, according to Drs Barlow & J. S. Bury (vide Keating Vol ii) & Cheadle (vide Allbutt vol iii) Rehn & Seemann (quoted by Goodhart, 5th ed, 1894, pp 63-7). have not been verified by the later observers, so that the observations of Karowitz, as previously stated, are most likely correct.

As the active process subsides, vascularity of the growing portion lessens, & the spongy bone tissue becomes hardened & condensed by the further formation of new bone rich in lime salts.

So far the description refers to intra-cartilaginous ossification; but changes from the normal occur also in the other form of bone formation, namely, in that from the periosteum.

The periosteum strips off the shaft of long bones more easily, but not cleanly off; for fragments of softened bone adhere to it, & is more vascular than in health.

The outer fibrous layer is thickened, & the inner proliferating layer, in which the bone structure is developed, is the seat of active hyperplasia; the amount of the subperiosteal bone varies from the normal to as many as five or six layers (Cheadle) Virchow has counted six to ten; usually two to three (Keating Vol II), It varies also in density & in the degree of calcification from spongy, limeless tissue to normal osseous tissue.

The periosteal formation, which is the sole form which obtains in the flat bones, shows excessive hyperplasia of the proliferating layer & imperfect ossification of like character. but where there is a pressure. eg. the weight of the brain upon the softened tissue, the bone does not develop, & the skull at that point (occipital or parietal bone) remains very thin. This hyperplasia is generally regarded as the result of an inflammatory process, a view which is supported by the fact that with the subsidence of the active rachitic state more or less induration of bone — sclerosis follows.

Microscopic examination — According to Tully, shows the deeper or osteogenetic layer of the periosteum to be chiefly affected, & the layers of spongioid bone beneath to consist of calcified islets arranged radically to the surface of the diaphysis. Beneath the islets of the bone are large red medullary spaces,

G. H. Makins. Treves system of Surgery. Vol i. p 365. says. The cartilage of the epiphyses under the micros-cope shows excessive & irregular increase of the cells & loss of definition in the columns. In the spaces between the columns much vascular round-celled material exists. Away from the growing line masses of calcified cartilage are seen, & still further much embryonic bone is present.

G. Sims. Woodhead. in his "Practical Pathology" 3d Edition, (after staining with. Chromic acid, Osmic acid, Picro-carmine & Iodine) observes as follow— (x50). In the thickened belt of cartilage with the irregular calcareous & bony layer beneath, there is an enormous proliferation of the cartilage cells, some of which have a regular arrangement. but by far the greater number are grouped without any attempt at arrangement, either as regards columns or size, & in many cases there is comparatively little matrix. In the small yellow opaque points calcification is going on

both in the matrix & in the capsules of the cells. The patches of calcified cartilages are not arranged regularly, but crop up indiscriminately. Blood vessels also make their appearance at irregular intervals in the cartilage, & closely following them, appear spaces similarly to these met with in ordinary bone, many of which are lined with a regular layer of pink cells or osteoblasts, & thus true bone, or a structure which very closely resembles it, is formed. Even in the midst of the bone, formed in this position, mazes of irregular cartilage cells may be still seen.

(x300) The above appearance when observed more closely, the great irregularity in the size of the cells, in the matrise, & in the calcification of the matrise; the calcification of the cartilage cells at certain points, their proliferation & apparent transformation into osteoblasts. Even when true bone is formed, it appears to be laid down

without any attempt at order, or regularity, & bone, calcified cartilage, & true cartilage are mixed up, apparently indiscriminately. Here we see then, that the chief points to be noted are the enormous & irregular increase of cartilage, with irregular & deficient bone formation.

(x50) The piece of the shaft examined. Under the fibrous layer of the periosteum there is a great increase in the number of small round cells or osteo-blasts, which form a thick deeply stained layer. In the deeper part of this cellular mass a few trabeculae, partly fibrous (stained pink) & partly calcified (stained green), may be seen. These trabeculae form an open net-work, & are seldom or never perfectly ossified; they consist rather of a calcified fibrous matrix. Beneath this osteoid tissue (very like that seen in an osteoid sarcoma) comes the true bone, somewhat loose in texture & irregular

in structure, in some cases almost like spongy bone, In this tissue the numbr of vessels & osteoblasts is always very great, but the osteoblasts are not markedly increased in number.

(X300) pp 471 - The pink round cells, or osteoblasts, along with numerous blood-vessels, are readily seen, not only beneath the fibrous layer, but between the granular looking trabeculae, & in the space between the osseous trabeculae. In normal bone these osteoblasts grow slowly, form around them periplasts, which gradually become calcified. In the case of rickets, however, these osteoblasts are formed in very large numbers, but any periplast which they may form is always small in quantity & only imperfectly calcified; there is also fine fibrillation of the intercellular substance, parts of which remain fibrous in place of being converted into bone. In the flattened bones, such as those of the skull, a process similar to that goes on under the periosteum of a long bone is met with.

It is evident from the above description that, although there is great proliferative activity in the cells, both of the cartilage & of the periosteum, calcification & ossification go on slowly & incompletely. Although little new bone is formed, the process of absorption goes on as usual (sometimes even more rapidly than normal, as the increased quantity of granulation tissue, & red cellular marrow aids in the process): but as the old trabeculae are absorbed new ones are formed to take their place. At the end of the developmental period, however, or when the disease gives way to treatment, the bones, when ossification does set in, may become very thick & strong from the fact that the osteoplastic tissue is present in such large quantities.

The bones may be stunted & deformed; this is owing to the fact that ossification sets in so rapidly that the epiphyses unite at an early date than usual, & the child remains dwarfed.

Ligaments. These suffer seriously in their nutrition; although the exact nature of the histological changes have not been investigated, they are obviously enfeebled, & like the bones, yield undue traction & pressure. Thus the ligamentous structures which bind together the bones of the feet give way as the rickety child begins to bear its weight upon them, & flat foot results. In like manner the ankles relax under the pressure & the feet splay outside-ways; the knees ligaments give way & knock-knee results; the supporting ligaments of the vertebral column yield, it beomes convex or curves laterally; & thus throughout all joints the laxness & feebleness of the binding ligaments & tendons prevail & alter shape & symmetry.

Muscles Dr Cheadle states that the muscles are also profoundly affected, This is shown by the feebleness, which, as has been stated, is in some instances so great that the child may be unable to stand or walk, keep the trunk erect, or in esctreme cases

to move in bed, or even to raise the head.
If the structure of the muscle be examined
they are seen to be flaby & wasted, &
under the micros-cope the striation
is blurred and indis-tinct.
Sir W. Jenner (lect iii) says the muscles
are small pale flabby & soft; & when
examined under the micros-cope, the
fibres are found to be singularly colour-
less & translucent; the transverse striae
very delicate, sometimes scarcly to be
made out; & not able to detect olein
in the fibres.
The Skin - The skin may exhibit nothing
abnormal except some slight pallor;
but in severe cases the anoemia is well-
marked, & in these instances there is also
pigmentation, The subcutaneous fat is
frequently increased; indeed in the
majority of cases of mild rickets the
child is as plump or more plump than
in health. In a minority of cases where
there is some general failure of health
or a concomitant cachexia such as con-
genital syphilis, the fat is deficient,

the child puny & emaciated.
This emaciation & pallor (Vide, Jenner, lect. iii), are the two great features, during life, of albuminoid infiltration. The anoemia is often most remarkable; & if, a little serosity, effused in the cellular tissue, the child has that transparent, waxy, greenish yellow tint, which is sometimes seen in the anoemia of young women. Now & then there is decided anasarca; the face as well as the extremities, the hands as well as the feet being oedematous.
The existence of excessive sweating would seem to indicate the presence of some morbid products in the blood which acts as a stimulant on the glandular structures of the skin; or perhaps the morbid stimulation of gland function may act through the nervous supply, & be a consequence of that hyperexcitability of the reflex nervous system which so conspicuous a feature of the disease, & is exemplified by the tendency to laryngismus etc (Vide Allbutt Vol iii. 97).

Mucous Membranes - These, throughout the whole respiratory tract & the whole extent of the alimentary tract, are especially prone to catarrh; so that laryngitis, bronchitis, gastritis, & enteritis are common complications. There is marked hyperplasia. The changes in the minute structure of the mucous lining, which are at the root of this abnormal tendency to catarrhal inflammation, have not been made out. All that can be said is that it is due to that vital instability & deficient resisting power which results from imperfect nutrition.

Lungs. As a result of the falling in of the chest wall the lungs suffer mechanically. As previously shown, the depression is brought about partly by the pressure of the atmosphere & partly by the traction of the diaphragm upon the softened bony framework; & this is most effective where the thoracic wall is weakest, namely, at the junction of the ribs with the cartilages, & where the wall is least supported by the viscera.

Tracts of collapsed lung are found beneath the lines of the grooves of depression, especially under the beads; & with this a compensating emphysema is developed over the anterior borders, where the forward projection of the sternum removes support & promotes distension of the air vesicles.

"Sir Wm Jenner lect II, says the emphysema is that variety called insufflation."

In addition to this collapse from direct pressure, there may be more extensive collapse from mucous obstruction when bronchitis has occurred. When cough is violent as in whooping cough, the collapse may be so great as to endanger life; & not infrequently, indeed, in that affection it is the complication which determines a fatal issue.

The general catarrah of the mucous lining of the bronchi & respiratory tract in many cases has been already mentioned, but the exact changes of tissue which they betray have still to be determined.

The Heart - In the case of the heart, also, whether in rickets any definite histological changes take place in the muscle has yet to be ascertained; but in view of the great feebleness of circulation which attends the rickety state there can be no doubt that this organ shares in the general malnutrition. The position of the heart in the chest is somewhat altered, the apex is pushed a little out towards the left, by the falling in of the thoracic wall (Vide Allbutt Vol III). (due to sternum forced forward & relative positions changed, Jenner Led II). White patch of thickened visceral pericardium is found, a little above apex, on left ventricle, where it comes in close contact with a bead on the rib, (in adults a friction patch is found at the centre of anterior surface of right ventricle)

Dr Hodgkins theory of the patch in rickets was due to pressure aided by movements of heart. Dr Wilks in lectures on Pathological Anatomy terms it "Attrition Theory".

The Liver In the majority of cases the liver is of normal size, although the margin may be low down in the abdomen, owing to depression of the diaphragm by the contraction of the thorax. In certain instances, which, however, in the experience of Dr Cheadle (Vide Allbutt Vol III), are rare exceptions, the organ is distinctly enlarged & harder than normal. This is due to a ~~fibroid~~ diffuse fibrosis, with slight increase of cellular elements & a deficiency in earthy salts, as in the bones.
The hyperplasia is probably due to the chronic hyperaemia set up by obstructed circulation through the lungs, the result of contraction of the thorax combined with feeble heart power, collapse & emphysema, which retard the outflow from the portal circulation. Such hyperplasia is readily set up in child-hood, a period when the formative process is active, Dr Cheadle has seen such fibrosis in marked degree in a child as the result of chronic bronchitis & emphysema. Or the hyperplasia may be irritative

in origin, due to the circulation in the blood of some peccant material, such as has been held to be the active cause of hyper-plasia in the bones & of the hyper-activity of the sweat-glands. In cert-ain instances the hyperplasia is of syphilitic origin, especially when accompanied by great enlargement of spleen. Sir Wm Jenner (in lecture iii) states that the liver & spleen are often larger than natural, but not the whole of the liver infiltrated.

Dr Dickenson's observations (vide Bristowe 4th Edition) seem to show that a morbid process, in some respects like that in the bones, goes on in the kidneys & lymphatic glands, & especially in the liver & spleen of rickety children; & that these organs become enlarged & indurated, & the seat partly of interstitial development of fibroid tissue, partly of overgrowth of the glandular elements. The changes are transitory, & are accompanied by a deficiency of the earthy salts. They are quite distinct from amyloid or lardaceous degeneration.

The Spleen & Lymphatic Glands.-

In Allbutt Vol iii. Dr Cheadle states -
With the enlargement of the liver there is commonly also enlargement of the spleen, due to a corresponding fibrosis with increase of cell elements & decrease of earthy salts. As in the case of the liver, the hyperplasia depends either upon the mechanical hyperaemia due to obstructed pulmonary circulation & feeble cardiac power, to irritative stimulation, or to congenital syphilis. In cases where the enlargement is great, sometimes indeed so excessive that the lower border of the spleen extends below the umbilicus, it is almost certainly due to congenital syphilis. The appearance of such a spleen after death cannot be distinguished from that of the organ in so-called cachexia, the origin of which is usually syphilitic, sometimes perhaps malarial in origin.
The lymphatic glands in these cases are likewise increased in size, indurated, & fibrous from like interstitial & cellular change.

Sir W. Jenner in his "Clinical Lecture ii on Rickets" states that the Spleen is increased in size, may be trifling a extreme, it is not adherent to parts ~~adherent~~ adjacent, the capsule is scarcely, if at all thickened, the anterior border is sharp, & firm to the touch, & smooth on surface, the weight, having regard to Size, is considerable. The substance is tough but elastic, the cut surface is remarkably smooth & translucent, & appears as if infiltrated with glue, Only a little pale blood can be expressed from its surface. Usually the organ is pale red, occasionally purple. the most transparent parts almost colourless.

The Splenic corpuscles are sometimes more readily seen than in a healthy Spleen. The cut surface of the glands is singularly pale & transparent; compact, Smooth, Folerally moist, &, to the unaided eye, uniform in appearance. The substance is tough, & the gland heavy in proportion to Size. In rare cases, it is purplish in colour, instead

of being pale. The glands thus diseased are never very large, usually vary in size from a pin-head to a sweet pea. they are not tender, & rarely, if ever inflame, & are free from adhesion. There is no increase in the number of white corpus-cles.

Dr Goodhart in "Diseases of children pp 423." states that in rickets the Spleen is large, its capsule perhaps a little thick, its substance firm, pale or dark-coloured, & under the micros-cope the fibrous septa of the organ are thickened. he has seen hyaline thickenings of the septa which might be called fibrotic in four cases which he has examined. As is well known, an albuminoid change has been des-cribed by Sir. W. Jenner as peculiar to rickets, but this can only occur in the more esctreme cases, & it is decidedly uncommon. he has never seen it (& Dr Gee only occasionally) On page 660, he states. The albuminoid disease of all these vis-cera has been de-scribed as a glue-like change peculiar to this disease, but the actual change—

& it is more common by far in spleen than liver, &, he thinks in lymphatic glands— is an increase in the fibroid material which constitutes the connective tissue of the organs, & it differs in no respect from that of the chronic enlargement of the viscera met with sometimes in ague etc. The disease of the spleen, commoner though it is than that of the liver, cannot be called common, At most (p660) he has only notes of forty-four cases, & in twenty four of these the rhachitic nature of the general ailment was doubtful. It would seem, therefore, that it can hardly be an essential of rickets, & probably Dr Gee is correct in considering it due to some pre-existing conditions, which, perhaps, it shares in common with rickets. The lymphatic glands undergo some change. probably of a fibroid nature, & reveal this by an indurated, scarcely enlarged condition. It is supposed, though without adequate proof, that this change is of a similar nature to that which the spleen and liver undergo.

Dr Eustace Smith in "Diseases of Children Second Edition pp 136-153", says that the alterations in the liver. Spleen & lymphatic glands are present only in exceptional cases.

Dr Dickinson (quoted by Drs Eustace Smith, Bristowe, 1st Ed pp 931. Goodhart Diseases of Children pp 423 & 660) states that in the spleen, the interstitial change may become so hypertrophied that the trabeculae are as thick as the spaces they enclose, in the meshes the corpuscles are crowded together. Increase in size seems due to a chronic congestive process, which causes a large development of hyaline fibroid material. In his Diseases of the Kidneys p. art ii pp 468. he says the rickety condition is profoundly different from the lardaceous, though the two have a superficial resemblance: it is distinguished from the lardaceous by the deficiency not of potash but of lime. The saline constituents in 100 grammes of fresh Spleen are contrasted in the healthy, lardaceous & rickety, as follows.

Saline constituents in 100 grammes of fresh Spleen.

Healthy

Cases.	Salts of the earths	Salts of the alkalies	Composition of the salts of the Alkalies				
			Potash	Soda	Phosphoric acid	Sulphuric acid	Chlorine
I. Healthy Spleen	·274	1.054	·322	·115	·118	·215	·096
2. "	·121	1.084	·301	·230			
3. "	·142	1.092					

Lardaceous

Cases	Salts of the earths	Salts of the alkalies	Composition of the salts of the alkalies				
			Potash	Soda	Phosphoric acid	Sulphuric acid	Chlorine
1. Lardaceous Spleen. Slightly affected. T. Jackson. Cause unknown	·216	·772	·194	·227			
2. Lardaceous decidedly, but not extremely. Elizabeth Moss. Probably Syphilitic.	·141	·982	·124	·239			
3. Highly lardaceous. Child with enormous vomicae.	·258	·658	·218	·115	·07	·049	·105
4. Not extreme. Abscess of ovary. Phthisis.	·201	·886	·245	·184	·258	·034	·09
5. Extensively lardaceous. Disease of Spine, with discharge.	·354	·699	·203	·190	—	·071	·099

Rickety

Case.	Salts of the earths	Salts of the alkalies	Composition of the salts of the alkalies				
			Potash	Soda	Phosphoric acid	Sulphuric acid	Chlorine
i. Rickety enlargement of spleen Ellen Smith.	·094	·989	·283	·196	·055	·025	—

VIII

The Brain & Spinal Cord.

According to Dr Cheadle, the increased size of the head in rickets is suggestive of a brain larger than normal; but this increase of size is due chiefly to thickening of the cranial bones. There is no evidence of increased skull capacity except in cases of separation of sutures when hydro-cephalus is present. The actual condition of the brain, even as to size, is a matter of uncertainty; & of the histological changes we know little.

There can be no doubt that the nervous structures share in the general malnutrition, but whether this gives rise to increase in bulk, as in the growing ends of bones, or to diminution, as in the muscles, is not clear.

According to some observers, the brain is smaller than normal, & the extra space in the cranium is filled by effusion of fluid into ventricles; according to others there is enlargement, due to increase of the neuroglia – a fibrosis such as that found in other organs.

Sir Wm. Jenner, in lecture iii. states that the so-called hypertrophy of the white matter of the brain seems really to be albuminoid infiltration of that structure. This transparent substance presents neither blue, violet, nor crimson reaction with iodine & sulphuric acid, such as are said by Virchow to be characteristic of lardaceous infiltration.

Dr Gee (Vide "On convulsions in children" St Barth. Hospital Reports, Vol iii. p. 109.) has recorded two cases, however, in which the brain was very heavy. A boy aged 2 years & 9 months highly rickety, & suffering from convulsions; the body weighed 17¾ lbs., the brain 59 oz.; the average at this age being 38.71 oz.

A girl of the same age, & also rickety, weighed 15½ lbs, & the brain 42½ oz., the average being 34.97 oz. In both cases the brain appeared to be perfectly healthy.

Dr. Hilton Fagge alludes to one case that came under his own notice, the case was thought to be one of advanced hydro-cephalus, until an examination after death

Showed the brain filled up the cranial cavity. and to six others cases under Dr. Fletcher Beach of the Darenth Asylum.
Dr. Beach has found a uniform granular appearance in the white matter under the micros-cope, with nerve cells scattered sparsely throughout, & an infiltration of the tissue with leucocytes. The increase in size was evidently due to the large amount of granular matter (Vide "The Principles & Practice of Medicine". By the late Charles Hilton Fagge. Edited by P. H. Pye Smith, M.D. Vol. i. pp. 556, 750.). (Dr Eustace Smith, in "Diseases of Children" 2nd Edition pp 130. states that the brain is sometimes small & shrunken, so that fluid is thrown out to fill up the space) Dr Goodhart in "Diseases of Children pp 498. says he is disposed to emphasize the remark of Dr West that an undue importance has been attached to it, as though it were of common occurrence, more so than will be found in practice. & thinks it is rare, he has not hitherto met with such a case.

The Blood In the Boston Med & Surg Journal, Vol i. p 369. 1897. a study of the blood in rickets is given by John Lovett Morse, A. M., M.D., Boston. He states that the blood of infants under two years differs in certain of its characteristics from that of adults.

The number of red corpuscles is about the same or a little larger, averaging a little over 5,000.000 per cubic millimetre.

The number of white corpuscles per cubic millimetre is somewhat larger, averaging from 10.000 to 12.000.

The relative proportions of the various form of leucocytes are also considerably different. The limits as given by Gaudolin, are as follow: (Jahrb. f. Kinderheilk., 1893, N. F., XXXV, 187.)

Small mononuclear	50 to 70 per cent
Large "	6 " 14 " "
Polynuclear neutrophiles	28 " 40 " "
Eosinophiles	1 " 10 " "

That is the proportion of mononuclear, or unripe forms, is about three times as great as in adult life, while that of the polynuclear neutrophiles, or over-ripe

form, is only half as large.
The mononuclear cells, moreover, are not merely lymphocytes but vary much not only in the size of the cell as a whole, but also in the size of the nucleus & in the amount of protoplasm.
Finally, an increase in the number of eosinophilic cells, even if considerable, seems to be of less significance.
Anaemia of various sorts is very common in children. It develops more easily & more frequently as the result of various morbid conditions & diseases in them than in adults. This is because the tissue changes in them are more rapid as the old tissues have not only to be nourished & replaced but new ones formed. Hence any disturbance of nutrition is more serious & results in more rapid & significant changes in the blood.
The classification of the anaemias of children is at best a vexed one.
That of Ettorti is the most complete, but seems unnecessarily complicated.
They, as those of adults, may be roughly

divided into the primary & secondary. The primary being subdivided into simple anaemia, chlorosis & pernicious anaemia. Chlorosis is not a disease of early child-hood. Warner, however, has noted that in the "simple anaemias" of childhood the percentage of haemoglobin is diminished to a much greater extent than that of the red globules". he (Mouse) has also noted this in the anaemia secondary to various diseases. (New York Med Rec 95 XLVII, 231)
Although cases of so-called progressive pernicious anaemia have been reported in young children, they are all open to criticism, & it is very doubtful if the condition occurs in them.
The simple & secondary anaemias may be divided into the mild & severe forms (anaemia levis, & anaemia gravis).
In the former the diminution in the specific gravity, haemoglobin, & number of ~~red corpuscles~~ erythrocytes is slight, & the red corpuscles show non-histological changes. In the latter, the diminution in the specific gravity, haemoglobin, &

& number of red corpuscles, is marked & the histological changes in the red cells are often considerable. The differences, however, are merely in degree, & only show a greater or less amount of disturbance of the blood forming organs. Either may or may not be accompanied by leucocytosis. In all but the mild anaemia without leucocytosis the spleen may be enlarged. It is to these cases that the term "splenic anaemias" has been applied. They do not merit a special name, however, as there is nothing characteristic in the blood condition, & an enlarged spleen may be associated with a normal condition of the blood. Much confusion exists concerning the so-called anaemia infantum pseudo-leukemica. The description of this blood condition as given by Luzet & Alt. Weiss, is as follows; Constant diminution in number of erythrocytes; constant more or less marked diminution in haemoglobin; poikilocytos; very many nucleated red cells mostly of abnormal forms &

many showing karyokinetic figures; polychromatophilia of the nucleated red cells & of many of the non-nucleated; pretty marked leucocytosis, always polymorphous. In addition there is always splenic tumour & more or less enlargement of the liver.

Those who consider this blood condition characteristic are divided as to the limitations of the disease. Some, who consider it always primary, would rule out those cases in which it develops secondary to rickets, syphilitic, or other anaemias, while others would include them.

Others think that there is nothing characteristic in the blood condition, but that it is merely a very severe anaemia, & not typical of any disease. Fischl (Zeitschrift. f. Heilkunde, 1892, xiii, 277. Weiner Klin. Wochenschrift. 1894, vii, 241.) has found the same type of blood in cases of rickets both with & without splenic enlargement. Moreover, cases of progressive anoemia with enlarged liver & spleen do not always show this condition of the blood. Whether the condition known as anaemia

infantum pseudo-leukæmica is to be considered as a separate disease or as merely a severe form of anaemia gravis with leucocytosis must therefore be left for the future to determine.

It is generally recognised that leucocytosis develops more quickly & to a higher degree in children than in adults. While in adults the increase of white cells is almost entirely in the polynuclear neutrophiles, this is not the case in children.

In them the leucocytosis is sometimes ~~sometimes~~ due to the increase of lymphocytes, sometimes to that of the large mononuclear forms, sometimes to that of the polynuclear neutrophiles, & sometimes even to that of the eosinophiles.

According to Weiss (Jahrb. f. Kinderheilk, 1893. XXXV, 146), the lymphocytes are much increased proportionally in affections of the gastro-enteric tract, whil the mono-nuclear cells of all sorts, as well as the transition forms, are proportionately increased in those of the respiratory tract,

When there are complicated tissue changes

& severe organic disturbances the proportions of the various forms of leucocytes show wide variations, He concludes that the proportions of the leucocytes correspond to certain tissue conditions & alterations & are thus characteristic for certain diseases, only in so far as quite definite tissue changes occur in these diseases. As the same pathological changes occur in many diseases, & different ones in the same disease, it is evident that the same histological blood condition is not always to be found in the same disease.
Monti (Wiener med. Wochenschrift, 1894. XLIV, 401. Weiner med. Press, 1894. XXX. 1552. Archiv. f. Kinderheilk. 1884-85, XVIII, 161.). has found that in normal children there is a constant relation between the specific gravity of the blood & the amount of haemoglobin. They vary directly. This relation is not constant in disease, however, & may vary in various ways.
Although the blood must have been examined in many cases of rickets, the literature of the subject is very meagre.

No work worthy of mention seems to have been done in this direction before the beginning of the present decade. Since then, however, several observers have reported short series of cases, although no one seems to have undertaken a systematic investigation of the subject. Monti found all the forms of anaemia described by him in his classification in rhachitis. As a rule, he found the more severe forms in the severe cases. No definite connection between the clinical course of the cases, & the condition of the blood could be made out, however. A greater proportion of the severe than of the mild forms showed splenic tumour. A greater proportion of the cases, with splenic tumour, showed leucocytosis than did those without. Certain cases with very large spleens showed no leucocytosis, however, & others without splenic enlargement showed a high grade. He concludes that "rickets as such does not cause any peculiar anaemia, but that according to the severity of the

rachitic process & the associated involvement of the blood-forming organs very different forms & gradations of chronic anaemia may exist."

Felsenthal (Archiv. f. Kinderheilk., XV, 78.) examined the blood in 12 cases of rachitis between the ages of nine months & two years, nine of which were mild cases with little or no enlargement of the spleen. He obtained the same results in all. The number of red corpuscles was normal or almost normal, & there was no evident relation between the number of corpuscles & the severity of the case. The haemaglobin was diminished in all cases & always to a greater extent than the red corpuscles.

The number of white corpuscles was always increased. He considered the oligochromemia as the most striking feature. He found that the red corpuscles often showed a considerable variation in size, & that nucleated forms, mostly normoblasts, & never very numerous, occurred

IX

in the severe cases. The majority of the white corpuscles were small & large mononuclear.

Weiss. (Jahrb. f. Kinderheilk. 1893, XXXV. 146). examined the blood in six cases, He found that all severe cases of rhachitis show a typical leucocytosis, the principal part of which is to be attributed to an increase of the mononuclear cells. The transmision forms are also numerous, so that the polynuclear cells appear relatively diminished.

Gaudobin. (Jahrb. f. Kinderheilk., 1893, N. F., XXXV, 189.). found the same condition in the blood of rickets as that of other forms of retarded development. He concludes that rickets as rickets causes no peculiar pathological change in white cells, & that any change in them is dependant on concurrent involvement of the internal organs.

Luzet. (Etudes sur les Anémias de la Première et sur L'Anémie Pseudo-leucemique. Thèse, Paris, 1890. Archives Générales de Médicine, 1891, p. 597. La France Médicale, 1891, XXXVIII, 741).

thinks that the blood in rickets associated with splenic enlargement may show all stages from mild anæmia to leukemia. He thinks that the splenic tumour causes leucocytosis, increases the anaemia, & makes the prognosis more gloomy.

Koch (Pediatrics) reports the results of the examination of the blood in two cases of rhachitic anaemia, one without & one with splenic tumour. The diminution of red corpuscles & of haemaglobin was more marked than in the latter. It showed no leucocytosis, however, ~~than~~ while the former did.

Hock & Schlesinger (Centralblatt f. Klin. Med., 1891, XII, 893, Haematologische Studien., Leipzig u, Wien, 1892.), has found that the specific gravity of the blood in rickets is entirely independant of the severity of the rhachitic process, but varies with the anaemia.

Felsenthal & Bernhard (Archiv. f. Kinderheilk., XVII, 333.), have also shown that it varies with the amount of haemoglobin.

Morse (Boston, Med & Surg Journal, Vol i., 1897, p 369) examined the blood in twenty cases of active, uncomplicated rickets in infants under two years of age. The blood was in every case taken from a lobe of the ear, & examined with a Thomas-Zeiss apparatus.
It was diluted (1-200) with a 3-per-cent salt solution coloured with methylene blue. The red corpuscles in one-tenth of a cubic millimetre & the white corpuscles in two cubic millimetres were counted.
The haemoglobin was estimated with a von Fleischl instrument. Cover-slips were in all cases made at the same time, hardened with equal parts of alcohol & ether, & stained with Ehrlichs' "triple stain". A differential count of at least 300 white corpuscles was then made, the classification recommended by Ehrlich being used. As far as possible the blood was taken about noon, & in most cases about three hours after food. The cases may be roughly divided into three groups according to the severity of the process;

those in which the manifestations are mild, those in which they are more severe, but in which there is no splenic enlargement, & those in which there is splenic tumour. The first group comprises 9 cases; the second, four; & the third, seven. The data of the individual cases are as follow:—

Mild Cases

Case I. Male, negro. Eight Months. Breast for six weeks, Mellin's food since. Marked sweating of head for two months. Always little cough. Bowels usually regular. W. D. & N. Hally. Holds up head. Head good shape. Anterior fontanelle widely open. No teeth. Sits alone feebly with marked kyphosis of weakness. Moderate rosary. Moderate retraction of ribs at diaphragm with flaring below. Heart & lungs normal. Abdomen large but soft. Liver normal. Spleen not palpable. No enlargement of epiphyses. No glandular enlargement. No irregularity in size or form of red corpuscle.

Case II Female Irish. Seven months. Breast for three months. Then Horlick's malted milk. Head sweats. Restless at night. Tendency to diarrhoea. W. D. & N. Hally. Pale. Holds up head. Head normal. No teeth.

Sits alone feebly with moderate kyphosis of weakness. Moderate rosary. Heart & lungs normal. Abdomen full & soft. Liver & spleen normal. Slight enlargement of epiphyses at wrists. Slight general glandular enlargement. No irregularity in size & shape of red corpuscles.

Case iii Female, negro. Seven months. Breast for five months, Condensed milk since. Head sweats. Rolls head. Kicks off clothes. Vomits occasionally. Large, fat child. Head normal. No teeth. Sits alone with fairly straight back. Slight rosary. Heart & lungs normal. Abdomen little full & tense. Liver & spleen normal. Slight enlargement of epiphyses at wrists. Little bowing of tibiae. Slight general glandular enlargement. Moderate variation in size of red corpuscles. many being large, No poikilocytosis. One normoblast seen in two slides. Several of the large mononuclear leucocytes very large & containing a very large nucleus.

Case iv Male. Irish. Thirteen months. Breast for ten months. Now arrowroot & milk. Pertussis at seven months. ~~Sits~~. Sits. creeps, & stands alone. W. D. & N. Hally. Pale. Head a little large. Fontanelle normal. Six teeth.

General hyphosis of weakness. Moderate ros-ary. Heart & lungs normal. Considerable enlargement of epiphyses at wrists. No glandular enlargement.

Case V. Female, negro. Thirteen months. Breast off & on. Mellin's for eight months. Then milk, also a little "table food". Never well. Very fussy. Head sweats a good deal. Cannot creep or stand.

W. D. & N. Flesh firm. Head rather large, but of fair shape. Fontanelle three-quarters of an inch square. No teeth. Sits almost straight. Moderate ros-ary. Heart & lungs normal. Abdomen large & rather tense. Large umbilical hernia. Liver & spleen not enlarged. Moderate enlargement of epiphyses at wrists. Slight knock-knee. Some general glandular enlargement. Very slight variation in size & shape of red corpuscles.

Case VI Male. Irish. Twenty-two months. Still on breast. General diet since fourteen months. Always well. Does not stand.

F. D. & N. Flabby. Pale. Frontal & parietal eminces large. Fontanelle one-half inch square. Ten teeth. Sits with fairly straight back. Marked ros-ary. Slight retraction at diaphragm.

Heart & lungs normal, Abdomen, Liver &
Spleen not enlarged, Cannot stand. Epiphy-
ses at wrists, & ankles much enlarged. Anterio
bowing of femora & outward of tibiae.
No glandular enlargement.
No variation in size or shape of red corpuscles.
Case VII Male. negro. Seventeen months.
Still on breast. General diet since twelve months.
Always fairly well. Head sweats. Fussy.
F. D. & N. Flabby. Head rather large. Fontanelle
nearly closed. Six teeth, Sits fairly straight.
Moderate ros-ary. Heart & lungs normal.
Abdomen rather large but soft, Small
umbilical hernia. Liver & spleen normal.
Cannot stand. Epiphyses at wrists large.
Slight bowing of tibiae. Slight general
glandular enlargement.
No variation in size or shape of red corpuscles.
Case VIII. Female. negro. Eight months.
Condensed milk one month; cows' milk,
three months; malted milk, four months.
Head sweats a great deal, sleeps poorly.
Diarrhoea for a long time.
F. D. & N. rater flabby, Head normal. No teeth.
Cannot sit alone, Moderate ros-ary.

Heart & lungs normal. Abdomen not enlarged.
Liver & spleen normal. Epiphyses at wrists enlarged.
Tendency to knock-knee. No glandular enlargement.
Very little variation in size, but not in shape of red corpuscles.

Case IX. Male. negro. Fourteen months.
Never nursed. Oatmeal & milk. Then general diet.
Head sweats. Restless at night. Always little cough.
Constipation. Weak. Tries to stand.
P. D. & N. Head large, flat on top, Anterior fontanelle not widely opened. Ten teeth. Back straight.
Marked rosary. Sternum prominent. Marked drawing-in at diaphragm. Heart & lungs normal.
Abdomen large & soft. Liver & spleen normal.
Epiphyses at wrists considerably enlarged.
Knock-knee. No glandular enlargement.
A good deal of variation in size of red corpuscles.
Numerous microcytes & macrocytes. Moderate poikilocytosis. Numerous very large mononuclear cells with large, pale nucleus & wide, pale protoplasm.

Seven Cases

Case X, Female. Irish. Six months.
P. D. & N. Pale. Craniotabes. Moderate rosary.
Heart & lungs normal. Abdomen large & soft.
Liver extends four centimetres below costal border.

Spleen not palpable. Epiphyses at wrists large. No glandular enlargement. Moderate variation in size of red corpuscles. Slight poikilocytosis.

Case xi Male. Italian. Two months. Condensed milk. Small & emaciated. Skin dry. Pale. Sutures open & bones movable. Slight rosary & retraction at diaphragm. Heart & lungs normal. No enlargement of epiphyses. No glandular enlargement. Marked variation in size of red corpuscles. Macrocytes much more numerous than microcytes. Marked poikilocytosis. Three macroblasts & two normoblasts seen in counting 500 whites.

Case xii. Male. Italian. Nineteen months. Breast for a year. General diet since. Sweats a good deal. Diarrhoea for a month. Quiet. Sits alone. Cannot creep or stand. P. D. & N. Much hair. Pale. Head rather large, but not abnormal in shape. Anterior fontanelle open, one-half inch in each diameter. Four teeth. Marked general kyphosis, not disappearing on suspension. Marked rosary. Heart & lungs normal.

Abdomen very large, tense & tympanitic. Liver & spleen normal. Epiphyses at wrists large. Large glands in groin only. Slight variation in size of red corpuscles. Occassional microcyte & macrocyte. A very little poikilocytosis. Numerous very large mononuclear white cells.

Case xiii. Male. negro. Two years. Brought for "general weakness", Head sweats. Tendency to looseness of bowels. Sits alone Cannot creep or stand. P. D. & emaciated. Head large & forehead square. Anterior fontanelle open, an inch each in diameter. Ten teeth. General kyphosis of weakness. Pigeon breast. Marked rosary. Slight retraction of chest at diaphragm. Heart & lungs normal. Very large abdomen. Umbilical hernia. Liver & Spleen normal. Epiphyses at wrists large. Inguinal glands enlarged. others not. Very little variations in size & not in shape of red corpuscles.

Cases with Splenic Tumour

Case xiv. Female. Irish. Nine months. Breast & general diet. Constipation & Losing weight. Restless at night. Kicks off clothes. Head sweats. Sits alone. F. Dr A. Habby.

Rather pale. Parietal eminences very large, frontal somewhat so. Fontanelle almost closed. No teeth. Sits straight. Slight ros-ary. Heart & lungs normal. Abdomen little full. Liver extends an inch below costal border. Spleen just palpable. Epiphyses at wrists a little large. No glandular enlargement. A little variation in size & shape of red corpuscles.

Case XV. Male. Norwegian. Twenty months. Always cow's milk. Now fair diet. Measles at one year. Very few digestive symptoms. Constipation. Sat up at nine months. Just beginning to creep. Does not stand. Brought because he cries when stood on feet. Not tender to touch. F. D. & N. Habby. Pale. Large head. Fontanelle widely open. Sixteen teeth. Sits with marked kyphosis in lower dorsal region, which is only partially obliterated on suspension. Small chest. Marked ros-ary. Heart & lungs normal. Abdomen large & soft. Liver large. Spleen palpable. Epiphyses enlarged at wrists & ankles. Anterior & lateral bow-legs. General glandular enlargement. Moderate variation in size of red corpuscles, but no microcytes

nor macrocytes. Very little poikilocytosis.
Case XVI Male. Italian. Nine months.
Breast & general diet.
Measles & diphtheria at six months, followed by running ear. Subjects to attacks of vomiting & diarrhoea. Head sweats badly. Sits alone.
T. D. & N. Baby. Pale. Large square head. Fontanelle very widely open but not tense. No teeth. Back straight. Small chest. Marked rosary. Heart & lungs normal. Abdomen large & tense. Liver normal. Spleen palpable. Epiphyses at wrists and ankles enlarged.
No glandular enlargement. No variation in size or shape of red corpuscles.
Case XVII. Female. Irish. Fifteen months.
Now on milk & cracker only.
Brought because of "weakness in legs". Sits alone. Doesn't creep.
T. D. & N. Pale. Head a little large. Fontanelle almost closed. Seven teeth. Back straight. Marked rosary. Heart & lungs normal. Abdomen full. Liver normal. Spleen distinctly palpable. Large epiphyses at wrist. Legs held loosely & rotated outwards at hips.

X

Tendency to talipes varus. No spasm. Knee-jerks present & equal. No glandular enlargement. Moderate variation in size of red corpuscles. A few macrocytes & numerous microcytes, Slight poikilocytosis.

Case XVIII Male. Irish. Five months. Never gained. Cries constantly, Sleeps very little. Frequent vomiting. Constipation. Small but not much emaciated. Marked pallor. Head negative; can hold it up. Cannot sit alone. Marked rosary. Systolic murmur over whole praecordia. Heart otherwise normal. A few râles here & there in both chests. Abdomen lax. Liver normal. Spleen palpable below costal border, as large as last phalanx of thumb. Epiphyses at wrists slightly enlarged. No glandular enlargement. A very little variation in size & shape of red corpuscles. One normoblast seen in counting five hundred white corpuscles.

Case XIX. Female. Russian. Twenty months. Breast & patent foods at first. Now general diet. Always digestive disturbances. Very restless at night. Sits & stands; cannot walk.

T. D. & N. Hally. Pale. Large, square, flat head. Anterior fontanelle nearly closed. Eleven teeth. General kyphosis of weakness. Pigeon breast. Marked retraction at diaphragm. Very marked rosary. Heart & lungs normal. Abdomen large & soft. Liver not enlarged. Spleen palpable below costal border, seven centimetres laterally & three centimetres vertically. Marked enlargement of epiphyses at wrists. Moderate bowing of both femora & tibiae. No glandular enlargement. Considerable variation in size of red corpuscles. Numerous macrocytes. Moderate poikilocytosis. Occasional normoblasts.

Case XX Female. Irish. Seventeen months. Condensed milk & barley water. Pale for a long time. Rapid loss of flesh in last three months. Tumour in abdomen noted two months ago, as large then as now. Just beginning to sit up alone. No vomiting. Constipation.

J. D. & emaciated. Marked pallor with yellowish tinge.
Fontanelle not closed. Four teeth.
Rosary. Heart & lungs normal.
Abdomen very full & tense.
Umbilical hernia. Liver not enlarged.
Spleen fills left half of abdomen, reaching nearly to median line & to within half an inch of anterior superior spine. No ascites.
Epiphyses enlarged. No glandular enlargement. Purpuric spots on abdomen. Urine negative.
Marked variation in size of red corpuscles. Many macrocytes & microcytes, the former being the more numerous. Moderate poikilocytosis. Moderate number of nucleated red corpuscles, megaloblasts being the most common form. Occassionally free nuclei. No abnormal forms of white corpuscles.
The results of the blood examinations are here given in tabular form:—

The figures in these columns represent percentages.

No	Age in Months	Type of Disease	Haemoglobin	Erythrocytes	Leucocytes	Small mononuclear	Large mononuclear	Polymuclear neutrophiles	Eosinophiles	Morphology of Erythrocytes
1	8	Mild	60	4.170.000	9.600	34	4	61	1	Normal
2	7	"	57	4.280.000	9.300	36	3	60	1	Normal
3	7	"	60	5.048.000	14.700	61	4	33	2	Moderate variation in size of normoblasts.
4	13	"	67	4.936.000	18.800	24	14	58	4	Nearly normal
5	13	"	48	5.024.000	17.900	47	8	44	1	Nearly normal
6	22	"	70	5.144.000	10.700	52	4	42	2	Normal.
7	17	"	64	5,102.000	14,000	49	6	43	2	Normal
8	8	"	67	4.840.000	10.200	53	4	41	2	Slight variation in size
9	14	"	62	5.528.000	9.000	32	16	51	2	Considerable variation in size. Moderate poikilocytosis.
10	6	Severe	65	4.704.000	13.900	48	4	48	0	Moderate variation in size. Slight poikilocytosis.

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The figures in these columns represent percentages

No	Age in Months	Type of Disease	Haemoglobin	Erythrocytes	Leucocytes	Small mononuclear	Large mononuclear	Polymuclear neutrophiles	Eosinophiles	Morphology of Erythrocytes
11	2	Severe	52	3,508,000	9,200	69	05	25	1	Marked variation in size. Marked poikilocytosis. Macroblasts & normoblasts.
12	19	"	67	5,290,000	7,200	29	17	53	1	Slight variation in size Slight poikilocytosis
13	24	"	64	4,604,000	5,500	39	15	45	1	Nearly normal
14	9	Spleen palpable	77	4,724,000	22,000	63	4	31	2	Nearly normal
15	20	"	73	4,448,000	14,400	49	6	40	5	Moderate variation in size Slight poikilocytosis
16	9	"	60	4,068,000	14,500	34	3	56	7	Normal
17	15	"	64	4,936,000	11,900	39	6	48	7	Same as No 10
18	5	Spleen considerably enlarged	57	4,519,000	15,900	44	4	47	5	Slight variation in size " " " shape Rarely a normoblast
19	20	"	68	5,158,000	13,200	51	5	40	4	Considerable variation in size Moderate poikilocytosis Occasional normoblast
20	17	Spleen very large	60	3,556,000	12,400	28	10	55	7	Marked variation in size. Moderate poikilocytosis Numerous nucleated forms

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An analysis of these cases shows that the number of red corpuscles was in all cases normal or but slightly diminished; ~~& always proportionately~~ that the percentage of haemaglobin was always diminished, & always proportionately more so than that of the red corpuscles; that there was a leucocytosis in a little more than half the cases; that this leucocytosis occurred more frequently in these cases with Splenic Tumour than in those without; that the amount of the leucocytosis was independant of the presence, or absence, or size of the Splenic Tumour; & that the histological changes in the red corpuscles increased, as a rule, with the severity of the case, being most marked in those with the splenic Tumour. The approximately normal number of red corpuscles & the absolutely & relatively diminished proportion of haemoglobin agrees with the results obtained by Felsenthal. It must be remembered this condition, however, as has already been noted,

is not an uncommon one in the anaemias of child-hood.

The results as regards the white corpuscles do not agree with those of Felsenthal, who found a leucocytosis in every case, but rather corroborates Mont's conclusions that leucocytosis may or may not be present. & that it is more frequent in the cases with splenic tumour. They also confirm his observation that certain cases with very large spleens have no leucocytosis, as the case with the largest spleen had but little more than the normal number of white corpuscles. The average of the different forms of leucocytes in the nine cases without leucocytosis gives the following proportions, which are somewhat different from those given by Gaudobin for normal blood;

Small mononuclear ...	43 per cent
Large mononuclear ...	8 " "
Polynuclear neutrophiles ...	47 " "
Eosinophiles	2 " "

The average proportions in the eleven cases with leucocytosis, as well as in the six

cases with enlarged spleens, are:

Small mononuclear .... 45 per cent
Large mononuclear .... 5–6 " "
Polynuclear neutrophiles ... 45 " "
Eosinophiles ..... 4–5 " "

That is almost the same as in the cases without leucocytosis. These results do not agree with those of Weiss & Selberthal, who found the increase in the mononuclear & transition forms, but rather with Weiss's general statement that when there are complicated tissue changes the increase may be in any or all of the different forms of leucocytes.

It is noticeable that eosinophiles cells are considerably more numerous in the cases with splenic tumour.

The association of nucleated forms & of variations in the size & shape of the red corpuscles with an almost undiminished number is of interest.

Conclusions

The results obtained in these cases, together with those obtained by others seem to justify the following conclusions:

Most cases of rickets are accompanied by anaemia. This anaemia may be of any form & of any grade of severity. The severity of the anaemia varies in a general way with the severity of the process.
The most common form is that in which the number of red corpuscles is normal a nearly normal, & the percentage of haemoglobin both absolutely & relatively diminished. The anaemia may or may not be accompanied by leucocytosis. Leucocytosis occurs more frequently in the cases with splenic tumour than in those without. It may be due to an increase in any or all of the varieties of white corpuscles.
The specific gravity varies with the amount of haemoglobin.
Finally, there is no form of anaemia found in rickets which may not be found in other conditions, & no form of anaemia found in other conditions which may not be found in rickets.

In the Cyclopaedia of the diseases of Children, Healing Vol iii, Drs Barlow & Bury state that the anoemia is of the chlorotic variety, the red corpuscles are diminished, and there is no marked increased of leucocytes. Dr Cheadle in Allbutt vol iii. 1897, states — The changes in the blood in rickets are imperfectly known. Anaemia is present, & as a rule it is proportionate to the other changes characteristic of the disease. When, however, the anaemia is extreme, it is associated with enlargement of the Spleen, & is often out of all proportion to other changes; the red corpuscles are diminished in number, the shape & size irregular, & the haemaglobin diminished. This extreme anaemia, coinciding with Splenic enlargement, is probably the result of the Special cachexia of congenital Syphilis; although it must be allowed that such connection cannot be traced in all cases.

Dr Goodhart in "Diseases of Children" 5th Ed. 1894. p 660. remarks that the condition of the blood in rickets has received but little attention. Chemically it has practically received none. He has made numerous microscopic observations of the blood of rachitic children, & the changes are certainly remarkable.
In some there is a simple deficiency of red corpuscles; in some a deficiency of colouring matter; in some the blood is crowded with a granular detritus; & in others the corpuscles are represented by four or five different sizes.
Dr Dickinson in his book on "Diseases of the kidney" has tabulated four cases. The number assigned to the red corpuscles in a square is the average of several countings, usually of six. With regard to the white, since from their usually small number the average in one square cannot be expressed as a whole number. it is stated as a fraction, of which the denominator gives the number of squares counted, usually 20. The averages at the end of each table give the red for one square, & that of the white for twenty.

Number of Corpuscles in $\frac{1}{31.375}$ of a cubic millimetre of Blood in cases of Splenic Enlargement, not Leucocythæmia

Name	Age	Case	Total	Red	White	White to red 1 to —	Appearance of Corpuscles
Annie Vincent	1 yr	Rickets, with enlarged spleen & liver Very anaemic in look. Under Dr. Barlow	—	105	$\frac{3}{5}$	169	Blood looked watery
Boy	10 months	Rickets, Large spleen, Pallid, cachectic, almost chlorotic look. Under Dr. Barlow.	—	70	$\frac{2}{5}$	177	Ditto ditto
Samuel How	1 yr	Enlarged spleen from rickets. Glands also somewhat enlarged. Under Dr. Gee.	—	102	$\frac{2}{5}$	256	Blood looked watery. Child was troublesome, neither of these estimations quite satisfactory.
" "	—	Another examination at a week's interval	149	—	—	—	Ditto ditto
Fred Bryant.	16 months	Rickets. Spleen greatly enlarged. - as low as crest of ilium & to within an inch of umbilicus	—	89	$\frac{3}{5}$	145	Blood watery corpuscles natural
		Averages	—	103	10 in 20 Squares	—	

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The Urine The analysis of the urine has yielded very diverse results. The great excess of lime salts stated to exist in it by early investigators, has not been found by the later. Marchands experiments (quoted by Jenner, Lect iii & Keating Vol ii,) were said to show by analysis six times the normal quantity of lime salts. Newbauer failed to find it in the urine of a case of extreme rickets. Luelzer in 1883 proved that lime was not in excess in the urine of rickets. Dr Goodhart in ("Diseases of Children, 1894 pb536) states, "The urine is said to contain too little urea & uric acid, & an increase of the earthy phosphates; though this statement has been called in question by Rehn & Seemann. Dr Cheadle (Vide Allbutt. Vol iii, 1897) remarks that it would appear that there is really no important difference between the urine of rickety & healthy children in this respect; nor can the presence of that lactic acid be detected which has been anticipated in accordance with a theory of the pathology of the disease which is no longer tenable.

Forms & Varieties of Rickets, & Conditions Resembling it.

Foetal rickets Dr Cheadle (Allbutt Vol iii) states that so far as he knows, no micros-copical record of progressive foetal rickets exists, there seems to be no doubt, from the observations of Guérin, Tripier, Lauro, & others, that in certain rare cases rickets begins in intra-uterine life, In his book on "Artificial feeding etc 4th Ed, 1896. he says the feebleness of health & malnutrition of the mother might well cause rickets in utero, & indeed the observations of those above mentioned as well as Narsovitz & Schwarz seem conclusive as to its occasional occurrence in varying degree.

Mr Tilly (Rickitic deformities p 253) says "as to the existence of congenital rickets, Virchow is agreed, & cases have been brought forward by Shattock (Path. Soc, Trans, 1881.), Herrock & others. T. C. Railton reports & figures a case in the Brit, Med. Journ, 16th June 94. p1299. Steiner mentions the existence of a

specimen of rickety foetus in the museum of the Hospital for Sick Children in Prague, 9 other cases are on record (Goodhart). The long bones in this disease are bent, fractures are found, & there is incipient beading on the ribs which on microscopic examination exhibit all the peculiar characteristics of rickety bone; the ossification of the cranial bones is also delayed.

There are other cases in which bone changes, such as softening & partial fractures of the long bones, are found in the womb, but the microscope discloses no proliferating cartilage, no imperfectly calcified spongy bone, no true rickety aberration of structure, These case have been traced to congenital Syphilis.

Syphilitic Rickets –

When rickets arise in conjunction with congenital Syphilis certain modifications follow. The child is as small, puny & wasted as when rickets concurs with general starvation; & it will probably have the depressed nose & linear scars

associated with congenital Syphilis; further distinctive features are projections on the frontal & occipital bones, the so-called Syphilitic bosses; these appear to be especially well marked in Syphilitic cases, & although met with in other instances in which no history or sign of Syphilis exists, it is usually in minor degree. The thinning of the flat bones, or craniotales, first described by Elsässer in 1843, & has till lately always been held to be as sign of rickets. (Vide Goodhart p 654). M. Parrott & others have called this doctrine in question, & consider the complaint a sign, not of rickets, but of congenital Syphilis. It is said to occur in thirty to forty per cent. of all cases of rickets. D. J. Barlow & Dr Lees (Path. Soc. Trans. vol xxxii. p. 323 et seq.). collected 100 cases of craniotales; they conclude that forty-seven per cent, of the total are Syphilitic. Dr Baxter (Path Soc Trans. vol xxxii p 361) gives 75 per cent. In some cases there is hypertrophic fibrosis of liver. Spleen & Lymphatic glands (Cheadle).

Scurvy rickets, So called acute rickets.

Rickets as a rule progresses slowly & insidiously; in a few cases it begins more abruptly, but the disease never sets in suddenly. In all cases in which this comparatively sudden onset has been alleged, there have been signs of bone changes & muscle weakness of longer standing which had attracted little notice until the process became more active. There are, however, certain cases which have been called acute rickets from the rapid supervention of such acute symptoms as extreme tenderness of limbs, periosteal swellings, Sponginess of gums, haemorrhages.

The periosteal swellings are due to subperiosteal haemorrhages, & the conction has been shown by Drs Barlow & Cheadle to be in reality one of scurvy, often, no doubt, superadded to rickets, but in its nature distinct.

Dr Barlow, in the "Medico-Chirurgical Transactions", Vol. LXVI, p 159. gives eleven cases, two of which are of the greatest

possible value, for he was able, by a post-mortem examination, to demonstrate the actual nature of the lesion that existed, from these two cases, & another published in the 'Transactions of the Pathological Society of London' by Mr Thomas Smith, it is shown that the clinical features of acute rickets are associated, it is true, with moderate rachitic changes, but much more with extensive subperiosteal haemorrhage - chiefly of the femora & tibiae, scapula, ribs, & cranium - & with a tendency to fracture, & sometimes to separation of the shaft from the epiphysis, as occurs in Syphilis, acute necrosis, & perhaps other conditions also. (Vide Goodhart p 641) Dr Gee in ("St Bartholomew's Hospital Reports", Vol. XVII p. 9) has published cases evidently of the same kind under the name of "osteal or periosteal cachexia". Mr Tubby in "Rachitic deformities p 254. says, with reference to Scury rickets, the factors in the causation of this disease,

are anaemia & an intensification of those errors of feeding which produce rickets. The symptoms are swelling of the limbs, due to sub-periosteal or intermuscular extravasations of blood. purpuric spots on the skin, spongy gums, haemorrhage from the kidneys, & spontaneous fracture of the bones.
Dr G. Sims. Woodhead in ("Practical Pathology" 3rd Ed, p 471) states that this acute form of rickets, which is supposed to be due to constitutional disturbance, associated more or less directly with the administration of unsuitable food, is characterised by sudden swelling of the bones, especially of the femur (over which there is tenderness or even great pain), oedema, a spongy condition of the gums & petechial haemorrhages in the skin,
After death similar haemorrhages may be found in most of the soft tissues & under the periosteum, especially at the junction of the epiphyseal cartilages with the long bones.

These haemorrhages are sometimes very extensive, though it is somewhat remarkable that necrosis seldom or never seems to follow this condition. We have the same cellular proliferation both in cartilage & in the bone, rapid absorption, & irregular & imperfect calcification. Dr Bristowe in "The Theory & Practice of Medicine" 7th Ed, p 934. States that acute rickets seems to affect both sexes equally, & to occur mainly between the ages of six & eighteen months.

The sub-periosteal haemorrhages affect the lower limbs more frequently than the upper, & are occassionally met with in connection with the ribs, scapulae, & bones of the skull. The swelling, for the most part, is not symmetrical or generally distributed. The lower extremity suffers more than the arm, the thigh more than the leg, & the neighbourhood of the tibia more than that of the fibula.

Petechial extravasations have been found in connections ~~in connection~~ with the viscera.

In the Brit. Med. Jour., Feb 29th, 1896-2pt. p 33.
E. Mayer (Deutsche med. Woch. Vereins Beil., 4) stated that recently cases of acute rickets (Scurvy rickets) a Barlow's disease have been observed frequently in Berlin, & related four cases which he had himself observed during 1895.
In first symptoms were observed in three infants in the six months, & in the fourth infant in the seventh month.
Three of the infants died. All belonged to families in good circumstances & all had been fed with "Keith's albumose milk." Hamburg, in the discussion which followed Meyer's paper at the Berlin Medical Society, said that cases of this disorder had been described ever since the end of the eighteenth century, & that Heubner had collected 260 cases recorded before the introduction of albumose milk. Moreover he had himself used this milk in the rearing of from 400 to 500 children, & had seen Barlow's disease in a few cases only.
Keith's albumose milk was superheated

milk to which albumen was added, & a little potassium bicarbonate. This salt was added in such proportions that the infant took a little over 6 grs a day. If taken regularly potassium bicarbonate was itself capable of producing scorbutic symptoms. Potassium was replaced by sodium bicarbonate, & the quantity of the latter added was so small that the infant took only 1½ gr daily. During the nine months since this change was made no case of Barlow's disease had been observed among the children fed on the albumose milk. Cassel mentioned four cases which he had observed & insisted on the importance of early diagnosis. He thought it important not to use ~~steril~~ sterilised foods for too long a time, & upon the first signs of scurvy to change the diet at once to fresh milk, or to milk which had been cooked for at most not more than ten minutes. Baginsky expressed the opinion that in making an early diagnosis

much importance was to be attached to the appearance of anaemia, & that the occurrence of bone lesions ought not to be awaited.

Drs Ashby & Wright, (Diseases of Children, 1st Ed. p. 326) thus sum up the identity of the disease; Drs Cheadle & Barlow both incline to the view that they are really examples of scurvy brought on by improper food, more especially by the absence of fresh milk for the dietary .... Others incline to the opinion that the condition is rather an exaggerated or excessive form of the anaemia, which is usually present in severe rickets, & in their opinion there is much to favour this view.

Chondrodystrophia foetalis, Achondroplasia, Foetal cretinism.

Dr Cheade (Vide Allbutt vol iii 1897) mentions other cases, again, of so-called "foetal rickets, chacterised chiefly by extreme & abnormal shortness of the long bones of the limbs, have likewise been found to lack the special features of rhachitic bone change. The area of proliferating

cartilage is absent, & there is little or no columnar arrangement of cells, ossification taking place, in fact, almost entirely from periosteum; while the thickening of the ends of the ribs is not a cartilaginous overgrowth, as in the true rickety head, but an osseous ring. From the peculiar fusing together of the bones forming the base of skull, the condition was regarded as one of foetal cretinism. Dr Goodhart (Vide Diseases of Children. 5th Ed. 1894. p 672) states that neither the bone nor cartilage lesions are those of rickets, but they are those of cretinism.

Mr. Shattock, however, considers that some of the cases illustrate a condition of rickets which has begun & ended in utero.

Later investigations, especially by Drs Symington & A. Thompson, appear to render it probable that the condition is distinct both from rickets & cretinism, of which the chief features are arrested or defective enchondral ossification in ~~enchondral~~ intra-uterine life — chondrodystrophia foetalis (Lab. rep. Roy. Coll. Phys. 1892 Vol. iv (p. 238).

The bones developed from membrane

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& those developed from cartilage late in intra-uterine life are normal; but those which are ossified from cartilage before birth are dwarfed. Thus, whilst the flat-bones of the skull, formed from membrane, are normally grown — in contrast to the semi-membranous condition in rickets, with its hypertrophic bosses & atrophic craniotabes — the bones at the base of cartilaginous development are dwarfed & prematurely united. The ribs & pelvis are also defective, & all the long bones of the limbs shorten to half their usual length. The remainder of the cartilage-formed bones, which are developed chiefly after birth, are of normal size. In the majority of these cases, the foetus perishes in the womb or soon after birth; a few only survive.

Osteomalacia There is in rickets a deposition of new bone, which is deficient in lime, & when the process is extreme, as in certain cases where the general atrophy of other tissues is marked, the body is wasted & bones small; this condition

has been called "rickety osteomalacia."
Behn, of Frankfort, has described a condition, which he calls "Infantile Osteo Malacia", which differs in some points from ordinary rickets.
There is in the "British Medical Journal 1884, vol. i. p. 213", by Dr. Judson Bury of Manchester, a case recorded of a female infant of eight months, which, in the absence of any rachitic changes, in the appearances in the medulla, in the thinning & easy fracture of the bones, is not unlikely to have been an example of true osteo-malacia, for the specimens of the bones from this case, & the appearances closely resembled those of the osteo-malacia of adults, whilst those of rickets were absent.
Dr Barlow describes a case, in which other marks of rickets such as rib-beads, grooves & cartilage proliferation were entirely wanting, & the morbid condition seemed to be analogous to the osteomalacia of adults (Vide art "Osteomalacia" p. 147 Cheadle)

Dr Goodhart (Diseases of Children 5th Ed 1894, pp 670-71) states that the bones in this disease, become thin, soft & porous, & their medullary canals disappear before an advancing growth of soft porous bone. The bones so affected are quite readily cut with a knife; but in the only two that have been examined after death, there were distinct rachitic changes in the cartilage zone, though but moderate in degree. This state of things occurs in young children. He has met with an instance which, in respect of softness, resembled this one, but which was characterised by a remarkable growth of bone, in a girl of fifteen months old. In the skull, the new growth & consequent thickening were enormous; a pile-like new bone gradually monopolised the diploic space; in the extremities, fusiform nodes were produced, in which more or less of the entire thickness of the shaft was converted into the same soft material. These changes were associated with pronounced rachitic

changes in the ends of the bones, & some have contended the entire process a rachitic one; but the marked degree of generalised bone-softening, & the enormous development of imperfect bone, are conditions which form no part of common rickets in the human subject. Bone changes, in many respects resembling these, have been found in unquestionably syphilitic infants "But," borrowing the words of the committee that examined the specimens ("Drs. Hilton Fagge. Barlow. Warrington Haward. Goodhart."—Trans. Path. Soc. Lond., Vol. XXXIV. p. 201.) "that such are necessarily & solely syphilitic appears to them in their present state of knowledge not proven. The apportionment of the effects produced severally by rickets & syphilis in this & other cases cannot as yet be determined." Very much the same must be said of late rickets & its relation to osteo-malacia. Some cases more resemble rickets, others osteo-malacia; but whether the two diseases are the same, or distinct diseases resembling each other is uncertain.

Recrudescent or Late Rickets.—

Rickets is a disease of the first two years of life, & in the majority of cases it makes its appearance before the end of the first year: in some instances, however, the disease does not set in until late. Dr Cheadle, in his book on 'Artificial Feeding of Infants & the diseases which arise from fault of diet in early life' 4th Ed, 1896. records a case of a boy nearly 10 years of age. The disease had only commenced to show itself nine months before, yet it was already so advanced that the patient had become unable to stand. The ribs were beaded, the chest walls driven in, the ends of the Tibiae much enlarged. There was pain & tenderness of the knees, but no rise of temperature. The case was clearly not one of food rickets, Antirachitic diet, cod liver oil, & steel wine were given, but the patient grew worse instead of better. Iodide of potassium was then tried, & remarkable improvement followed for a time, & the condition was thought to be Syphilitic, although there

was no positive evidence of this.
Eventually the child went out, & subsequently died of bronchitis. A cast of his distorted limbs & body is now in the museum of the hospital.
That this case was one of the true rachitic changes in the bones was subsequently almost certainly proved on post mortem examination (Vide Path Trans vol xxxii p. 391), &
Mr Robert Jones, at the 'Liverpool Medical Institution' (Vide Brit. Med. Jour, Feb 8th, 1896, p. 341) exhibited a girl aged 16 with recrudescent rickets. She could walk perfectly until two years ago, when considerable epiphyseal disturbances occurred.
Changes were apparent in the skull, Jaw, upper limbs, sacrum, & lower extremities.
The forearms presented the deformities usually ascribed to crawling, although during infancy & childhood they were perfectly straight. The family history was good, & no dietetic cause could be found for the deformities, which were extreme. Mr Jones did not consider such cases to be as rare as described, having seen between 20 & 30 of them.

C. B. Keetley (Vide. Illust Med News. Sept 1888, & Ann Surgery Vol ii, p. 308) reports a case in a woman aged 20 years.
She had noticed for some time a marked swelling of the right hip. This was variously diagnosed, as dislocations, periostitis, & tumour. From the slow progress of the disease, & the sudden development of a superadded scoliosis. Mr. Keetley diagnosed rhachitis adolescentium.
A wedge of bone was removed from the convexity of the femur, the bone snapped across, the adductor longus divided, & the limb straightened. Microscopical examination of the bone removed showed the changes characteristic of rickets.
Dr Dawtrey Drewett (Vide. Trans. Path. Soc. Lond. Vol. xxxii. p. 386.) showed at a meeting of the Society, in 1880. a boy, aged 10. in whom the disease was well-marked.
The ~~Committee~~ committee appointed to investigate the case. viz Dr Hilton Fagge, Mr. Warrington Harward, & Dr. Drewett. came to the same conclusion, that the changes were identical with rickets. The wrist

ends in this case were enlarged, the bones were much distorted, & the child was quite helpless. He died two years later, & a post-mortem examination was made by Dr. Abercrombie, & Dr. Barlow, who found the epiphysial lines of the bones thickened & irregular, as in common rickets.

Dr Davies-Colley in the Trans. Path. Soc. Lond, Vol, XXXV, mentions a case of late rickets in a girl of ten. She had always been pale, thin, & delicate, & from an early age the ankles grew outwards & the knees inwards. The humerus fractured, & subsequently the femur, & for this, at the age of ten was admitted in Guy's Hospital. It was then found that the long bones were very tender & flexible, & this outer shell could be pressed inwards like the shell in craniotabes.

The urine was much deficient in phosphoric acid, only one-third the normal amount being present; the calcium was in excess.

She died, at the age of thirteen, from a

suppurative pyelitis, due to the formation of phosphatic calculi.
After death several of the bones were found much distorted — some were hypertrophied & dense, others light & thin, & in some were tumour-like expansions of a light porous bone, with fibrous looking tissue intersecting them.
The microscopical examination by Mr Symonds showed a complete absence of compact tissue & of Haversian systems, a porous bone being filled with fibrous tissue.
Clinton (Vide St Thomas Hospital Reports, 1884, vol xiv) gives details of a case in a girl, aged 12, in whom the symptoms had been present for twelve to eighteen months, no deformity having been previously noted.
She was backward as a child, dentition delayed, & did not walk until aged 3½ years.
Mammae were well developed, & catamenia had been present for four months.
The chief characteristics were great enlargements of the epiphyses, extreme bowing of the legs & exaggeration of the clavicular curves.

Clifton refers also to a case under Mr Pitt; a girl, aged 14, who had been treated as an out patient four years before for flat-foot. At that time no rachitic changes were noted. The disease came on gradually at the age of 12, & more rapidly during the six months previously to coming under treatment. No history of rickets in infancy; she walked at the age of 12 months, & could run 6 months later.
The chief characteristics were general epiphyseal enlargement, extreme distortion of the legs, genu valgum on the right side and genu varum on the left, poor nutrition, stunted growth, & absence of sexual development.
Ransford (Brit. Med. Jour, 1887, I, 1213.) records the case of a girl, aged 14, in whom the epiphyses were enlarged & genu valgum ensued.
Palin. (Practitioner, Vol. XLV, 1890, pp 275-320) refers to a girl, aged 6, who came under the care of Mr. King at Pekong for well-marked rickets. The bones became

straight, & a lateral curvature disappeared in the course of four months' hospital treatment.

Duplay (Gaz. des Hôpitaux, Paris, 1891, p. 1397) mentions a girl, aged 17, with the radius & ulna of each side affected.

Barlow & Bury (Keatings Cyclopedia of diseases of Children, vol ii, 255) refer to two cases in which there were very active manifestations at the age of 11, & which proved post-mortem to be undoubted rickets. In one case certainly, in the other probably, there had been previous infantile rickets. The authors regard the cases as remarkable recrudescences of the disease.

Sir W. Jenner in his 'Lectures on Rickets', refers to the case of a boy in whom the symptoms did not manifest themselves until he was a little over 3 years of age, & the case of a girl, aged 9, just beginning to suffer.

Dr Edmund Cautley (Brit. Med. Journal Jan 4th, 1896, p 13.) states that it is generally assumed that rickets is a disease of early life only, developing during the

period of the first dentition. Even such a great authority as Charles West states that he has never seen the disease commence later than 3 years of age, although he has known the symptoms become more & more grave up to the end of the fifth year. (Lucas in Lancet 9th of June 1893, says that late rickets comes on about puberty, & is associated with albuminuria).

The following case is closely allied in character to the cases of Barlow & Bury, & the photographs of their patients resemble remarkably the photograph of the present patient.

The following are the notes of the case;

M. J., female, aged 11, came under the care of Dr Cautley in June, 1895, for anorexia, vomiting, & feverishness of one week's duration.

Family History.- Older & younger children are strong & healthy.

Past History.- This is somewhat imperfect on account of the mother having recently deserted the family. Measles &,

XIII

Chicken-pox at the age of 4; an elder sister states that the disease first commenced at the age of 4, that the child has never been able to walk well, & latterly has been unable even to stand.

Present Condition – Head dolichocephalic; receding small forehead, a little prominent in the middle line; Skull asymmetrical, the right parieto-occipital region being somewhat flattened; flattened vertex; Sutures & fontanelles all closed; eyes wide apart; bridge of nose flattened; dilated nostrils; prognathos; upper jaw a little beak-like in the middle line, being elongated antero-posteriorly; palate not highly arched; teeth up to date, gums receding, & lower central incisors loose; earthy complexion anaemia. Chest flattened latterally below the level of the nipples; marked beading, especially of the fourth to seventh costo-chondral joints, prominence of the angles of the ribs behind; Harrison's Sulcus not present; no heart or lung disease detected. Abdomen large & doughy; flabby muscles, flatulent distension;

enlarged liver & a little enlargement of the spleen. Back arched. Upper limbs; exaggeration of clavicular curves; scapulae normal; humeri small, epiphyses enlarged; great enlargement of radial epiphyses on both sides; old greenstick fractures of the radius & ulna on each side, most marked in the ulna, especially on the left side at the junction of the upper with the middle third; on the right side of the fracture is situated about the middle of the shaft; much radial deflection of both forearms; marked enlargement of the distal ends of the metacarpal bones of the thumb; phalanges normal. Lower limbs; Pelvis distorted; shafts of the femora curved antero-posteriorly; the the lower epiphyses of the femora & the upper ones of the tibiae & fibulae are much enlarged, while the lower epiphyses of the two latter bones are even more enlarged proportionately to their size; Shafts of the tibiae are flattened, much "buttressing"; marked genu valgum, especially on the left side; the deformity of the two legs differs somewhat; the antero-posterior curvature is most marked in the

lower third; feet were affected.
General condition – A small, stunted child with flaby, ill-nourished muscles, & considerable anaemia; mental condition a little backward for her age.
Course, – For sixteen days the temperature was irregularly raised, sometimes reaching 102° to 103° F., although no cause could be found except the rickets. Subsequently the child steadily improved & gained weight.
Condition four Months after being first seen. – The child is a much better colour, stronger, & has gained flesh considerably; is able to stand & walk. The enlargement of the epiphyses has very much diminshed, & at the lower ends of the forearms is hardly noticeable. The beading of the ribs has practically disappeared. The legs are a little less bent, abdomen very large & prominent.
Dr Cautley has called this a case of recrudescent rickets for the reason that the disease was present at an early age, although apparently it did not.

commence as soon as is usually the case. From the age of 4 to 11 the deformity has been present and increased, & is still present in a very marked degree. The signs & symptoms of recrudescence consist in the marked enlargement of the epiphyses, an enlargement which has practically disappeared while under treatment; a corresponding increase & diminution in size of the beads on the ribs; the irregularly febrile temperature, & the muscular flacidity & wasting. It differs from some of the cases recorded as cases of late rickets in that the disease does not appear to have at any time subsided since the date of onset, although no doubt it has remained quiescent. Of the cases referred to by other writers it is a curious fact that nearly all are of the female sex. Judging from an isolated case like this one, Dr Cautley is inclined to regard cases of late rickets as recrudescences of the disease rather than as the development of the disease in a subject previously unaffected.

Etiology

Rickets is the result of imperfect & perverted nutrition; so far pathologists are agreed; there is, however, some divergence of opinion as to the exact causes of this defect. Most of the hypotheses have been based solely upon a consideration of the morbid changes met with in the bones, as if this comprised the whole pathology of the disease. To trace the nature & source of these faults of ossification may supply the key to the nature of the general affection, but no pathological doctrine can be regarded as satisfactory which does not also explain the morbid conditions of muscle & tendon, mucous membrane, & nervous system; these conditions are concurrent & constant, although they are less obvious & obtrusive than in the affection of the skeleton. The production of a rickety condition of bone by the application of Esmarch's bandage, as in the experiments of Kassowitz (quoted by Dr Cheadle in Allbutt Vol iii. 994 & Drs Barlow & Bury in Keating. Vol ii)

does not produce the changes in the tissues. The bones are certainly soft & they give way under pressure; but the ligaments are also soft & lax, & give way under pressure likewise; the muscles are atrophic & enfeebled; the mucous membranes catarrhal; the reflex nervous system hyperexcitable & unstable.

It is difficult to understand how the bone lesions & the other concurrent tissue changes can stand to each other in the relation of cause & effect, as has been suggested; or how morbid ~~morbid~~ conditions, regularly associated with the bone lesion & in proportion to it, can be regarded as accidental.

The explanation (according to Dr Cheadle in Allbutt Vol iii. 1897) of the earlier & greater prominence of the bone changes lies in this, that the bones, being in the most active stage of their growth, exhibit the departure from the normal most clearly at a time when the coexisting changes in other organs & tissues are smaller or less visible.

Many are the causes to which the production of rickets has been attributed, Faults of diet, setting up gastro-intestinal catarrh,

impaired digestion, vomiting & diarrhoea; want of light & fresh air; deficient clothing, dirt, & general bad hygiene; Syphilis; inherited tendency & microbic in origin, have all been credited with more or less reason as concerned in the genesis of the disease. The various factors, enumerated above, which are concerned in the causation of rickets present a very complex & difficult problem for solution. Whereas in some cases a single cause seems sufficient to account for the whole of the observed effects, in the majority of instances investigation shows that several different & often independent factors are simultaneously at work. These, however, are not all in action in every instance, & they are not, therefore all essential, nor equal in constancy & potency. Bearing in mind the fact that in comparatively few cases can a single cause be assigned, ~~an~~ an attempt, nevertheless may be made to classify roughly the various factors at work in the causation of rickets.

Microbic origin of Rickets.—

In the Brit. Med. Journ. July 6, 1895: epit i. Hagenbach-Burckhardt (Berl. Klin. Woch. May 27th, 1895) discusses the etiology with special reference to rickets being an infectie process. Theories attributing the disease to deficiency of lime salts, to lactic acid, are no longer tenable. Kassowitz, under certain circumstances, is disposed to admit various micro-organisms as the cause of rickets.

Poisons due to micro-organisms can readily be supposed to set up the lesions found in the disease. The temperate zone is the one in which rickets abounds. The cases of rickets increase at the beginning of the cold season, when children are kept in the house. The greater the altitude, the less frequent is rickets.

The infective theory would explain the prevalence of the disease in citiated, & its infrequency in pure, atmospheres. Both rickets & Tuberculosis are most developed in large towns & in notoriously unhealthy streets. Enfeeblement of the

individual by acute or chronic disease predisposes to both diseases. Measles also predisposes to both. In early age chronic infectie processeses are frequently localised in the bones. There is nothing in the clinical picture of rickets against the view of its being an infectie disease. Acute rickets is known. The spleen is frequently enlarged. The objections to the view are that no micro-organism has been found, & that similar changes in bone may be produced experiment-ally in animals by withholding lime salts. The author thinks that the disease set up in this way is not identical with rickets, nor does he think that foet-al rickets has been shown to be identical with the ordinary disease. He would look upon defectie feeding, vitiated atmospheres, acute & chronic infectie diseases, as predisposing causes only. In the "Brit. Med. Jour, Aug. 24, 1895. Epit. p 32." Mercoli (Gaz. Med. di. Torino, Nov 11. 1895) pleads for the microbic origin of rickets, believing that the disease is caused by the effect of

ordinary pyogenic organisms upon the osseous & nervous systems. Clinically he finds support for this theory in the fact that rickets develops independently of social condition; frequently begins with eczema, boils, or intestinal catarrh; occasionally occurs epidemically; & is accompanied by fever, polyarthritic & bone pains, hydrocephalus, marasmus, & paresis of lower extremities. Pyogenic organisms have been found in the bones & central nervous system of ricketty children. Experimental injection of pyogens into the bones & epiphyseal cartilages of young rabbits produced common osteomyelitis, but in other cases an osteomyelitis without trace of suppuration, with hypertrophy of the ends of the bones, hypertrophy of cartilages analagous to that of rickets, & marasmus.

In the ("Brit. Med. Jour. April 23. 1898. Epit. p68") Mercoli (Gazz. degli Osped. e delle Clin., Jan. 30th. 1898) again draws attention to his experiments & observations on this subject. He believes that rickets is due to infection from the presence of streptococci & staphylococci. These organisms are constantly

found in the mouths of children & the mammary ducts of nursing women, & are swallowed. As long as the alimentary canal is healthy nothing happens; but if this is out of order from catarrh, dentition, etc, & if in any way these organisms find their way into the blood & are localised in all the organs, but especially in the parts of greatest functional activity, the disease is produced. In the infant these are the nervous system & the epiphysial ends of the bones; hence the symptoms in rickets. The bone lesions are considered by the author to be examples of chronic osteomyelitis of a special type. In the "International Medical Magazine" 1897. pp. 369-370. "Researches on the Pathogenesis of Rachitis (Revue mensuelle des maladies de l'enfance, March 1897). By Smariotte Ettore. M.D. After reviewing the more important work done in this line, the author gives the results of his own experiments. Ettore repeated the work done by Micoli, who has stated also found from cultures made from the pulp of rachitic bones staphylococci

& Strepto coeci pyogenes, & concluded therefrom that rachitis was an infectious staphylo-strepto-coccic osteitis.

Ettore made bacteriological examinations of the epiphyses of rachitic & non-rachitic bones during the months of Dec., Jan, Feb, & March, beside one case in September & one in October. He also studied during May, June & September twelve cases of non-rachitic bones.

Technique He cauterized a large surface of the anterior extremity of a rib & penetrated the interior of the cartilaginous tissue with a sterilized pipette & aspirated. He made cultures on gelatine plates & large agar tubes. In most cases by the same procedure, he cultivated the marrow of the diaphysis of the same bone. He endeavoured always to have the same quantity of substance. He selected ribs & clavicles because they were more readily obtained. In the rachitic cases he chose bones in which the lesions were evident to the eyes. In the sound

bones examined there was no trace of rachitis. He made twenty-five examinations in the bones of non-rachitic children. In sixteen of these the inoculations remained sterile, six cases gave pure cultures of streptococci, two gave the Bacterium Coli Communis, & one case gave few colonies of Staphylococcus pyogenes albus. In the six cases showing streptococci, four were from children dead of diphtheritic angina associated with the streptococcus, the fifth was in a child from a cystic scarlatina, & the sixth from a fatal case of broncho-pneumonia; in the last cases the colonies were few. The author believed that a strepto-coccemia, in which one of the localizations had been the bones, especially the epiphyses, had existed during life. There were no other appreciable lesions of the bones. The two cases in which he found the Bacterium coli communis had died of very grave measles complicated by broncho-pneumonia & acute enteritis,

& from this he infers that the bacilli were present as the result of an infection by this organism during life & were not the result of invasion after death. He concludes from these observations that in septic conditions in children the microbes readily invade the bones, that they are found in the long bones & especially in the epiphyses, & that in a child the Bacterium coli commu-nis & other bacteria inhabiting the intestine very rarely, if ever, invade the bones in the first 30 or 35 hours after death.
In the "Clinical Journal. T. 1898." Chaumier (Centralb. f. Kinderheilk. 1894. II. 358). from his studies on the nature of rickets, is of the opinion that rickets is an infectious disease, which may be contagious. This opinion is based on the frequent presence of rickets in institutions for children, & also on the fact that in case a child suffers from rickets in a house occupied by other tenants the rickets will also make its appearance in other children.
He further observes that wet-nurses, whose

own children were rickety, communicated the disease to the children they nursed. Casati has mentioned that he knows of families whose children born in certain houses were rickety, but those raised in another house remained free from the disease.

The author became aware of epidemics among young pigs, in whose bones he later found the characteristics of rickets.

He arrives at the following conclusions;

i^st Rickets, is a specific disease, caused by unknown microbes.

ii It is contagious, endemic in cities, & occasionally epidemic.

iii It is found in an epidemic form in young pigs, & its germs are preserved in houses (Pediatrics. June, 1898).

Climate Season & Locality :-

Although rickets seem to exist in all parts of the world its occurrence is chiefly influenced by climate, & chiefly in respect of dryness, sunlight, & warmth. According to Dr Cheadle (Vide Allbutt Vol iii. 94). Dr Bristowe 4 Ed p 924. & Dr Barlow & Buz in

Keating Vol ii; it is most common in the temperate zone, especially in cold, damp, cloudy regions such as England. Holland, & certain portions of Germany. Italy. Austria. France & South America. It is rare in Greece, & least common where malaria exists, & rare in Tropics. It diminishes in frequency as high northern latitudes are approached, &, on the other hand, declines towards the South until, as stated, almost disappears in the Tropics. In Australia, rickets was believed practically not to exist; but in 1891 it was shown by Mr Muskett (Vide, Australasian Med. Gaz., 1891, X. pp285-91) to be prevalent in the large Towns, & cases have been met with even in the bush. The disease appears to be affected by Season; various Continental authors (Quisling Archiv. f. Kinderheilkunde, IX, 1888, p293.) have studied this factor, & their results show an increased prevalence in the earlier Summer months, & a minimum in December. Barowitz (Wiener med. Woch., XXXIX, 1889.) ascribes this to the

confinement to the house during earliest months of life of children born in winter-time. Dr Cheadle states that cases are more frequent & more severe during the dull, cold winter months when the children lie chiefly indoors, & lack light, fresh air & warmth; that is, when vitality is lowest.

With regard to locality the chief point made out is, that the disease is, generally speaking one of great cities of London, Manchester, Liverpool, Glasgow, Vienna, New York; & that in this country it is especially prevalent in the towns of the great manufacturing districts of Lancashire & Yorkshire, the Black Country, Scotland & Wales; in these regions of smoke & darkness the mothers are largely workers engaged away from home, & their children are chiefly brought up by hand.

Drs Barlow & Bury (Keating. Vol ii) state that it is most common in Prague London & Manchester, an average of thirty per cent attending the hospitals has been given by Ritter. Gee & Ritchie. In Vienna Kassowitz says percentage is 80, in children under 3.

Drs Garrod & Fletcher in "Brit, Med. Jour. Sept 21, 1895. p 708", remark that it is extremely difficult to obtain a satisfactory knowledge on the influence of locality & such statistics as are forthcoming do not always bear out the general statements ~~as are~~ which are made as to the comparative immunity of certain parts of the earth's surface. Nevertheless, it seems to be established that in colder regions rickets is more common than in the warmer zones.

Quisling (Archiv. f. Kinderheilkunde, IX, 1888. p. 293.) in a statistical paper shows that the prevalence of the disease does not widely differ in various European capitals. Even in Philadelphia the percentage of children seen at the hospitals that are rickety is approximately equal to that at Manchester, London, & Frankfort; & this is especially interesting, as it is opposed to the view often held in England that rickets is a comparatively rare disease in the United States & in Canada. As the result of enquiries from a number of medical men practising in those countries

we are led to believe that, in the rural districts & small towns at any rate, the disease is comparatively infrequent, & there seem grounds for thinking that, except in so far as its occurrence is modified by the struggle for existence in densely populated regions it will not be found to be conspicuously more common in one part of ~~the Temperate &~~ the Temperate zones than in others. The influence of density of population & the stress of life is certainly very great, & is well seen in the contrast, in this respect, between our large cities with their lower class populations & our rural districts.

Influence of Race. A difficulty is met with in estimating the liability of different races, but it has not seemed to Dr Garrod & Fletcher (vide Brit. Med. Jour. Sept 21. 1895. p 707.) that Jews & members of the Latin races resident in London show any less tendency than English children. The suggestion that their liability is even greater may arise from the fact that as a rule their circumstances are often more adverse than those of our own poorer compatriots.

Influence of Sex – Statistics as to the relative frequency of Rickets in boys & girls vary; but the general outcome of these seems to be that Sex exerts no influence, that the disease is distributed evenly between the two sexes.
Dr Goodhart (Vide Diseases of Children 5th Ed. p 646.) collected 141 cases, 68 of these were boys, & 73 were girls.
Dr Gee (Vide, St Bartholomew's Hospital Reports, vol iv. p. 69) gives 635 cases, 365 boys, & 270 girls.
Inherited Tendency – No satisfactory evidence has been produced to show that rickets is ever transmitted from the parents to the children. According to Dr Cheadle rickets dies out with childhood, & is not likely to be handed down to the offspring of mature persons.
The influence of heredity is probably limited to the transmission of a weakly constitution, or to some factor of imperfect nutrition of the foetus in the womb. That heredity is not an essential or constant factor is shown by the fact that the children of perfectly healthy persons become richety; in the vast majority

of cases, indeed, rickety children are born of parents who do not exhibit the smallest of past rickets.

Dr Eustace Smith (Diseases of Children 2nd Ed. p 138-9) remarks that hereditary tendency is considered by some observers to be an element in the etiology of the disease. In the case of so common an affection it must, no doubt, often happen that the father or mother of the patient had once suffered in a similar way; but that a parent who had been rickety in childhood should give birth to a weakly infant, & that this infant, brought up in violation of all the rules of health, should develop rickets, is surely but slender evidence in favour of the hereditary transmission of the disease. Supporters of this theory usually point to the cases of so called 'congenital rickets' as instances of the inherited form of the disease. Dr Goodhart in "Diseases of Children 5 Ed p 647 quotes the observations of Ritter von Rittershain which show that rickety children frequently come of mothers who still bear traces of having suffered from a similar disease.

Drs Garrod & Fletcher (Vide. Brit. Med. Jour. Sept. 21. 1895. p 707.) state that heredity has long been looked upon as having a share in the causation of rickets, & its influence has been recognised by Ritter von Rittershain. Pfeiffer, Henoch, & others. They are not in a position to make any very definite statement about it, but they point out that any estimation of the influence of heridity in this connection is rendered extremely difficult by the fact that parent & child may alike suffer from rickets, not because of any inherited tendency, but because both were exposed in infancy to similar unfavourable conditions of diet or hygiene.
Tuby in "Rhachitic Deformities" 1896. p 253. says heredity plays no part in the production of rickets.
Jenner "Lecture III" says there are no facts to prove that rickets is heredity.
Dr Bristowe (Vide Theory & Practice of Medicine. 7th Ed. p 927.) states that it does not appear to be hereditary in the sense that rickety children are the offspring of rickety parents.

Congenital Syphilis Most Authors quote, but do not agree with M. Parrot, who laboured to show that rickets is always the consequence of an hereditary syphilitic taint. The arguments of this observer in favour of his view are derived chiefly from morbid anatomy. He points in particular to the anatomical changes observable in the epiphyseal ends of the long bones in the two diseases as evidence of the specific nature of rickets. But the latter is not only a disease of the bones; & although the epiphyses in the two cases may present a certain similarity of lesion, there are other alterations of structure in rickets which are different from those of syphilis. In the majority of cases the children bear about them none of the well established signs of congenital syphilis; the eruption, the snuffles, the linear scars, the pegged teeth, the keratitis are wanting. The peculiar tendency to functional nervous disorders, have no counterpart in the specific disease. In many cases, moreover, the history seems absolutely

beyond suspicion; & conversely, many children who suffers from congenital syphilis are not rickety. (Theadle Vide Allbutt Vol iii. 97, & also quoted by Drs Garrod & Fletcher in Brit. Med. Jour, Sept 21st, 1895, p 707). But in opposition to this view they quote Monti who states that he has never seen a case of hereditary syphilis in which rickets was not present.
Baginsky found evidences of syphilis in 6 per cent of all rickety children.
Dr Eustace Smith (Vide Diseases of Children, 2nd Ed. p 138) says that rickets is common in localities where congenital syphilis is rare, & rare in places where the latter is common, It is met with in animals as well as the human subject, & is produced in them by faulty hygiene & bad feeding as it is in the child.
Still, although it cannot be allowed that rickets is caused by syphilis. Syphilitic infants may become rickety: & it is probable that a parent weakened

by a former syphilis may, without transmitting the taint to his offspring, beget a child of feeble constitution in whom rickets can be easily induced. But in both these cases injudicious feeding & insanitary conditions must come into operation before the disease can occur.

Drs Garrod & Fletcher state that in common with most of those who have written upon the subject, including such authorities as Henoch, they are quite convinced that such a view as Parrot's is untenable, although prepared to rank syphilis among the predisposing causes, as a disease tending the nutrition both of mother & child.

Mr Tilly in 'Rhachitic Deformities' states that syphilis plays no part in the production of rickets

Dr Cheadle (loc cit.) thus sums up. It is clear, then, that syphilis is not a constant, invariable, essential factor, Congenital syphilis modifies rickets, it does not create it. The cases in which it does

play a part have special features of their own. The child is puny & wasted, & it presents some of the distinctive signs of syphilis; & to these cases especially belong the boss like projections of the frontal & occipital bones, in their most extreme form, & the craniotabes, first described by Elsässer in 1843; although they are not absolutely limited to the syphilitic variety. Dr Thomas Barlow & Dr Lees (Path. Soc. Trans., Vol, xxxii, p. 323.) collected 100 cases of craniotabes, & from them they concluded that forty-seven per cent, of the total are almost certainly syphilitic; & to this may be added the observation of Dr Baxter (Path. Soc. Trans., Vol. xxxii. p 361) that of the twenty-three per cent, of craniotabes in rachitic children, seventy-five per cent, were syphilitic.

Possibly the enlargement of lymphatic glands, liver, & spleen, met with in some cases, may prove to be rather a syphilitic than a rachitic change.

Bad hygiene - Defective hygienic conditions are largely concerned in the production of rickets. The great incidence of the disease upon the population of large cities & amongst the poorer classes there, & the fact that rickets is comparatively rare in the bright, sunny climates of the South, where life is spent largely out of doors in fresh air & sunshine, afford evidence of this. The want of sunlight & warmth appears to tell especially upon children of southern race when reared in cold & uncongenial climates.
The child of Neapolitan parents, for example, brought up in the great cities of America, are stated by Dr Snow of Buffalo (Vide Med. News (Phila), 1894 L. XV. pp. 316-320.) to suffer from rickets to such an extent that even those brought up at the breast do not escape
Drs Garrod & Fletcher (Vide Brit. Med. Jour, Sept 21st. 1895. p 708) state that children brought up in sunless, airless, & often overcrowded rooms & seldom taken out into the fresh air, often develop rickets, even when receiving a

diet beyond reproach. A practitioner who had lived for nine years in Auckland, New Zealand, informed them that the only case of rickets that he had seen during that period occurred in a child aged 2 years that was practically never in the open air.
Under the same heading may be included want of personal cleanliness, lowered bodily warmth from scanty clothing favour the production of rickets by degrading nutrition. Although, however, these influences of defective hygiene are frequently concerned in the production of rickets, Dr Cheadle states, they are not constantly present, & are not therefore invariable or essential factors. but in extreme cases, they are generally at work. Many cases of rickets, however, arise in patients who live under excellent sanitary conditions, so far as air & light & cleanliness & warmth are concerned; a child may enjoy these in perfection & yet become rickety; & again a child may not become rickety, although brought up under the most unhealthy external conditions.

The Tubercular Diathesis – In lecture iii., p 34. Sir Wm Jenner states that phthisical parents are no more likely to have rickety children than are non-phthisical parents. The facts contained in a table made by Dr Edwards, Physician to the Consumption Hospital at the East of London, renders it probable that they are even less likely.

Dr Eustace Smith (Vide Disease in Children. 2nd Ed, 1889. p. 138) says a pronounced tubercular disposition appears to have a protective power against rickets; for although weakly, phthisical parents may give birth to feeble infants who readily fall victims to rickets, it is rare to find the latter disease in a family where other members have died of tubercular meningitis or other form of pure tuberculosis – unless, indeed, the tubercular mischief has occurred secondarily to rickets. The reason of this immunity seems to be that the causes which are

capable of setting up rickets will induce Tuberculosis in a child predisposed to this form of illness & very quickly bring his life to a close. Tully in his book on "Rhacitic Deformities. p 253, states that the tubercular diathesis may be considered a factor in so far as it is a cause of general weakness, & predisposes to digestive disturbances & diarrhoea. Ritter von Ritterhain, Senator (Vide Ziemssen's Handbuch, XIII, erste Halfte, 1879, p. 178), & others, consider that phthisis in the father is an important predisposing cause of rickets in the child. The experiences of Dr Garrod & Fletcher (Vide Brit. Med. Jour, Sept 21st. 1895; p 708) leads them to doubt the frequency of their association.

Dr Goodhart (Vide Diseases of Children, 5th Ed, 1894, p 647) states that Trousseau held that the two were mutually exclusive. But there can be no doubt that Tuberculosis is not uncommon as a sequel to rickets. Hillier says, the two conditions seldom go on actively at one time.

Maternal Causes of Rickets

Dr. A. E. Garrod & Dr. H. M. Fletcher in the "Brit. Med. Jour.", Sept. 21. 1895; p 707. state that in confronting such a problem as that of the maternal elements in the causation of rickets, the statistical method cannot be relied upon. Trustworthy results are only to be obtained by careful observation of numerous individual cases, in which, to all appearances, other factors may be excluded, & in which this element stands out predominant. Accordingly in place of statistics examples illustrating the various points raised, are quoted.

This maternal element has received comparatively little attention from authors either here or elsewhere, & although almost all authors, & especially Sir Wm Jenner (Lect. III., p 34. Eustace Smith, Diseases of Children, p. 137; Quain's Dictionary of Medicine, 1894; article, Rickets, Pepper, Text-book of Medicine) in our own country have laid some stress upon the state of the mother's health, few have dealt with the individual factors in detail.

In this connection it is necessary to discuss the usual period of onset of rickets. In a large proportion of cases the earliest traces of the disease, such as beading of the ribs, may be detected if looked for at about the completion of the first six months of life, & the experiences of Drs Garrod & Fletcher agrees with that of Luisling, (loc. cit.), & most other Continental authors, that the maximum number of cases commence between the 6th & 18th months.

Dr Goodhart (Vide Diseases of Children 5th Ed. 1894, p 646) analysed 141 of his own cases, to show the time of life the disease is met with

5 m.	7 m.	8 m.	9 m.	10 m.	11 m.	12 m.	18 m.	2 years	2½	3	3½	4	5	6	Total
3	1	3	2	5	6	11	36	26	19	13	2	6	5	3	141

Sixty-eight were boys, seventy-three girls.

Dr Gee "On Rickets" St Bartholomew's Hospital Reports, vol, iv, p. 69." gives much larger number than these, Of 635 cases (365 boys, 270 girls) 32 were under six months, 144 from six to twelve months, 183 from twelve to eighteen months, 133 between eighteen months & two years, 116 in the third year, & 27 in the fourth year.

Pfeiffer (Jahrbuch f. Kinderheilkunde, XXIV, p. 248) points out, most cases only come under medical treatment at a later period, when deformities of bones or late dentition excite the anxiety of the parents.

As regards the occurrence of rickets in newly-born children, its frequency has certainly been exaggerated owing to the inclusion in this category of cases of other forms of bone disease, such as syphilitic affections & osteomalacia & cretinism.

Schwarz (Medicinische Jahrbuch, VIII. 1887) even states that among 500 newborn children in Vienna 75.8 per cent, showed distinct signs of rickets - figures which are entirely at variance with the experience of Drs Garrod & Fletcher.

Even Henoch (New Sydenham Soc., II, p. 408) looks upon the question of such occurrence as still an open one, although he considers that it may occur. & Cheadle (Artificial Feeding & Food Disorders of Infants, 1889) enunciates similar views.

On the other hand there can be little doubt that there are most important

causes at work prior to the birth of the child which may lead to the development of rickets at an early period of life, & these form the first group of maternal influences, the ante-partum causes. The second group in which the maternal influence is exerted through the milk, & which may be called post-partum or lact-ation causes.

The ante-partum causes are as follow—

(1) Ill health, malnutrition, or disease of the mother during pregnancy.

One of the most important causes of malnutrition on the part of the mother is phthisis concurrent with pregnancy. Drs Garrod & Fletcher (Vide Brit. Med. Jour, Sept 21, 1895.) have met with numerous examples of rickets born under such circumstances. From amongst these they select the following examples.

A female child, aged 11 months, was brought to the out-patient department at the Hospital for Sick Children, Great Ormond Street, exhibiting marked

signs of rickets in the skull, ribs, & in delayed dentition. The infant had been fed on good cow's milk & water since birth; no farinaceous food had been given. The mother had had six confinements, but three children were said to have died of "consumption" in infancy. She herself had had haemoptysis, was found to have tubercular laryngitis, & was much wasted.

A male infant, aged 17 months, exhibited well marked rickets. It was suckled for five weeks, & was then given cow's milk & water, & no farinaceous before eight months. The mother died of consumption thirteen months after the birth of the child. She was known to have had the disease for two years before her death. Her only previous child, born ten & a-half months before the patient, & only suckled for five weeks, died at the age of 15 months. It will be noticed that in both the above cases although the children were brought up by hand, their diet had been as good as possible under the circumstances.

In the following case the nature of the mother's illness was uncertain, & the child was breast-fed; A female infant, aged sixteen months, with well-marked rickets, was suckled for eleven months, & had had in addition cow's milk in a bottle. When seen it was having two pints of good milk daily.
The mother had poor health, & at the seventh month of pregnancy was confined to bed for four weeks, & also for the fortnight before delivery with what was described as "abdominal inflammation". In this case the influence of numerous pregnancies cannot be excluded, as the mother had borne seven children in the course of fourteen years.
They have also collected other examples in which the mothers have suffered from want of food, excessive vomiting, as well as from various more or less ill-defined illnesses during pregnancy, & their children have developed rickets apart from any recognised dietary cause.

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Drs Garrod & Fletcher are convinced of the truth of the following statement of Sir William Jenner: "The health of the mother has a decided influence upon the development of rickets in the offspring; of this much they are sure, that when the mother is in delicate health, in a state of which anaemia & general want of power form the prominent features, without being the subject of disease usually so called, there the children are often in a very decided degree rickety, although the father is in robust health, & the hygienic conditions in which children are placed most favourable".

(2) Want of Fresh Air & Exercise during Pregnancy:- Cases belonging to this group probably occur more frequently in private than in hospital practice, but examples are seen in which signs of rickets have appeared in the child when the mother had been obliged to maintain a recumbent attitude, & was therefore confined to the house during some months of pregnancy,

although there had been no concurrent disease, the feeding of the infants was above reproach, & previous & subsequent children have been quite free from rickety manifestations.

III Numerous & Rapid Pregnancies. Sir Wm Jenner (Lect III), states that it is very common for the first, or the two or three first born children to be free from any signs of rickets, & yet for every subsequent child to become rickety. The explanation of this fact is that among the poor the parents are generally ill fed, ill clothed, & ill lodged, the larger the number of their children - the means wages remain stationery - the calls on his means are increased. And among the rich & poor the larger the number of children the more has the mother's constitutional strength been taxed, & the more likely is she to have lost in general power.

Drs Garrod & Fletcher (Vide Brit. Med. Jour. Sept, 21, 1895. p. 708.) state that they imagine that no one will be disposed

To question the liability of the youngest children of large families to rickets, or that such liability is increased if the successive pregnancies have followed rapidly upon each other. Out of a series of such examples they quote the following typical cases;
A male child, aged thirteen months, exhibited marked rickets. It had been brought up upon good cow's milk from birth, & had been given no farinaceous food. It was the fourteenth child. The mother had been married twenty five years, & the previous child was twenty months old at the time of the patients birth. No other child was known to be rickety. Both parents were healthy, & there had been no miscarriages.
A male child, aged 10 months, with marked rickets was the youngest of eight children all born at full term & suckled. Enquiry failed to elicit any account of rickets in the other children. The mother, who was in

good health during & after pregnancy, had been married fifteen years. Her milk was abundant & had formed the sole diet of the child. Hygienic conditions were good.

(iv) Multiple Pregnancy.—

The following case illustrates the influence of twin pregnancies, & it may be supposed that just as during lactation the one twin may get more than its fair share of milk from its mother, so in their intrauterine existence the nutrition of the more weakly one may suffer in comparison with that of the other. A female twin, aged 19 months, exhibited marked rickets. Both children had been breast-fed for three months & afterwards given a good supply of cow's milk & no farinaceous food under the age of 12 months. The other child was very slightly rickety. There had been one previous confinement a year before the birth of the twins.

v Age of Mother at Birth of the Child.—

It is a recognised fact that a mother advanced in life, even though she has

not had numerous previous confinements, is more apt to produce rickety children than a younger woman. The following are examples of this:-

A male child, age 10 months, was decidedly rickety. It had been suckled entirely. The supply of milk was plentiful. The mother aged 42, had been married twenty-two years, & had had eight children, of whom the eldest was 21. No previous child had been rickety. The previous child was aged 4 years, & the mother was in good health.

A male child, aged 2½ years, had been brought up on good fresh milk to the age of 1 year, & had had good ever since. It was a second child after an interval of eighteen years without any intermediate conceptions. The signs of rickets were conspicuous.

It is possible that pregnancy at an unduly early age has a similar influence upon the nutrition of the foetus, but we are not able to bring forward examples illustrating this point.

vi. Lactation during Pregnancy-
It is believed lactation during pregnancy to be a very important & often overlooked factor in the causation of rickets. A male child, aged 8 months, showed distinct beading of ribs. The mother, aged 22, had been married four years & had two children. The first child was being suckled throughout the first five months of the second pregnancy. The labour & puerperal period were natural. The child was entirely breast-fed, & the supply of milk was abundant. A second patient, a male, aged 4 months, with definitely beaded ribs, was the ninth child, & none of the others had been rickety. The previous child had been suckled at least the first three months of the last pregnancy. The mother was healthy, but having little milk had supplemented the breast with good cow's milk & barley water. Here again the influence of numerous pregnancies cannot be excluded. Such examples might be multiplied almost indefinitely.

Post-Partum or Lactation Factors.

The influence of the condition of the mothers in favouring the development of rickets does not cease with the birth of the child, but lasts throughout the period of lactation.

Dr Cheadle in his book on "Artificial feeding of Infants & the Diseases which arise from Faults of diet in early Life" 4th. Ed. 1896, says it does not appear in children at the breast, except in special instances wher the milk is insufficient or defective, as from special debility of the mother or from prolonged lactation, he thinks it may be affirmed broadly that children feeding well on a full supply of good breast-milk up to the age of 8 or 10 months do not become richety during the time of Suckling. Even with congenital Syphilis at work, the Suckling does not become richety. Dr Barlow & Dr Lees (Path. Soc. Trans, vol xxxii. p. 330.) found that in eleven children with craniotabes who were brought up entirely at the breast, not one showed the smallest signs of rickets.

Drs Garrod & Fletcher (Vide Brit. Med. Jour, Sept. 21st, 1895, p 710.) state that breast-fed children are not, as is so often held, by any means immune from rickets, & whilst their liability is in part due to the causes which have already been mentioned, there can be no doubt that it is also in part due to a deficiency in the mother's milk of those elements which are necessary for the proper nutrition of the child. Gumplowitz (Prager med, Wochenschrift, xiv, p. 50, 1889.) at Prague, found signs of rickets in 28 per cent, of breast-fed children attending th Klinik, whilst 50 per cent, of the hand reared children suffered in the same way. Moreover the hygienic factors affect breast-fed as much as bottle fed children. The recognition of this liability is of extreme importance & one to which little attention seems to have been paid, since lactation errors are easily controlled, whereas the ante-partum causes admit of much less regulation.

The lactation causes may be classified as follows,

I Deficiency of milk supply, due to:

(α) Abnormalities of mother, for example, retracted nipples; single available breast, owing to abscess, new growth, etc.

(β) Abnormalities of child, for example, hare lip, cleft palate, etc.

(c) Sharing of supply by twins.

(δ) Deficient activity of gland.

II Deficient quality of milk supply, due to:

(α) Mothers health during lactation.

(β) Menstruation during lactation.

(c) Pregnancy during lactation.

III Excessive duration of lactation.

I Deficient Milk Supply.—This group hardly requires further discussion, since the enumerations of the conditions referred to will almost suffice. The cases in which the abnormality is confined to the child, although falling into this group, can hardly be referred to as a maternal cause. Cases are seen in which the occurrence of rickets can only be ascribed to the difficulty experienced in obtaining the natural supply. In the case of

twins the conditions are complicated by other causes also, & it appears to be especially the weakly twin that tends to develop the disease, partly because it obtains less nourishment, & partly from inferior nutrition in utero.

II. Mother's Health during Lactation:- The influence of the mother's health upon the constitution & quantity of the milk is well recognised; concurrent illness, over-work or worry & confinement to the house, are all active factors in this connection. The case of a male child, aged 14 months, the youngest of seven children, who exhibited well-marked rickets. The infant was suckled only for six months, & after that had good cow's milk, a pint & a half per diem. During lactation the mother had an attack of either gout or rheumatism, lasting for two months, & felt very ill during that period. The quantity of milk was diminished. In this case the influence of numerous pregnancies & of the age of the mother cannot be excluded.

Menstruation during Lactation.

Menstruation appears to be no ~~less~~ uncommon occurrence during lactation, at any rate among the lower orders. Schlichter (Wiener klin. Wochenschrift, ii, 1889, 51 & 52; iii, 1890, 4 & 5. Summary, Schmidt's Jahrbucher, vol. 226, p. 55) found that among 424 suckling women at a lying-in hospital no fewer than 50 menstruated during the first two months & a half.

Tilbury Fox (Trans. Obstet. Soc., London, 1863, iv, p. 260.) considered this one of the most important of all the causes of rickets, & gives as his experience that, with very few exceptions which can be otherwise explained, "wherever the rachitic child is entirely dependent upon the mother's milk, the mother will be found to have menstruated during lactation, regularly for several months, & the degree of rachitis to be in direct ratio to the frequency, duration, & amount of the menstrual flow. If the child is partly fed upon food

other than the nurse's milk, the disease is lessened in degree or altogether prevented." He quotes a series of 39 cases to illustrate these points.

As regards the changes taking place in the milk during menstruation, Bouchut, Beguerel & others found an excess of fat & a diminution of lime salts, & especially of phosphates, but there has been considerable disagreement in the matter, & the most recent careful analyses of Schlichter show that the changes are very slight, & he looks upon them as insufficient to cause rickets. He also, unlike earlier observers, was unable to detect any marked change in the general condition of the child during the occurrence of the menstrual flow, although he carefully watched 52 infants under these circumstances. He concludes that the occurrence of menstruation after the sixth week is neither prejudicial to the child or mother. The clinical experience of Drs Gorod & Fletcher, however, tends to support the view

that this has an important influence in the causation of rickets, & from amongst a series of cases bearing upon this question the following are selected;

A male child, aged 12 months, with marked rickets, was suckled entirely for ten months, & had no farinaceous food. For two months it had had fresh cow's milk. It was the first child. The mother, aged 25, but looking ten years older, was evidently not a person of robust health, had never had any serious illness, & stated that she was quite well during pregnancy. The supply of milk was abundant during the whole lactation period, but the menses returned three months after the birth of the child, & continued regularly but somewhat more copiously than usual from that time. The child was not in any way upset during the periods.

A male child, 13 months old, with marked rickets, had been suckled entirely to 12 months. The mother, who was

weakly-looking, had borne five children in ten years, the previous child being 4 years old at the time of the patient's birth. One at least of the previous children had rickets. She always menstruated during lactation, generally from the fourth month after delivery. The loss was much as usual in amount, & no change was noticed in the children during the periods.

Pregnancy during Lactation:- It may easily be supposed that when pregnancy occurs during lactation, this event will not be without influence upon the child at the breast by affecting the quality of the milk. It is not believed to be a very uncommon factor in the causation of rickets, especially among the lower orders, in which lactation is so often prolonged to an undue extent, this has already been given as a cause of future rickets in the unborn child. The following is a case to illustrate this;

A female child, aged 2 years, with conspicuous rickets, was the second of a family of three. The mother, aged 28, had been married five years. Neither of the other children were known to have rickets. She was pregnant with the third child six months whilst she was suckling the patient. The child had the breast for nine months, & was afterwards given fresh cow's milk in sufficient quantity, & later other suitable articles of diet.

Over-Lactation, — Dr Eustace Smith in "Diseases in Children" 2nd. Ed, 1889, p 137. says the long continued suckling may induce rickets. for the breast-milk after a time ceases to satisfy the infants wants, & too little additional nourishment is supplied. Dr Goodhart (Diseases of Children, 5th Ed p 645) believes that prolonged suckling causes rickets. Drs Garrod & Fletcher (Brit. Med. Jour, Sept 21st, 1895, p 710.) state that too prolonged lactation is a well recognised cause of rickets. The cases which they have quoted have been carefully selected to illustrate

particular points, with as few disturbing factors as possible. Syphilis was excluded in every case which has been quoted, & in all the evidences of rickets were conclusive. Nevertheless, where so many interacting influences are at work, it is extremely difficult to bring forward scientific proof that any single one of them is responsible in any particular case. In some instances an intricate tangle of maternal factors, apart from any obvious dietetic or hygienic causes, for example:- A mother affected with exopthalmic goître had borne seven children within 12 years, all were suckled for about 16 months, & during her last pregnancy was suckling the previous child for some months. Here we have illness of the mother, rapid child-bearing, over-lactation & pregnancy during lactation combined. The seventh child, aged 13 months, had beaded ribs, curved spine, enlargement of the epiphyses, & rickety skull. The mother exhibited marked signs of Grave's disease, dating from twelve months previous to the birth of the child.

Anatomical predisposing causes of Rickets:— Tully (Vide Rhacitic Deformities, 1896, p 253) states that Beneche has studied the predisposing causes of rickets from an anatomical stand point. He found that in rachitis the heart is of average size, but the arteries are abnormally large. Jacobi aptly says "As it is not probable that a chronic disorder in its slow progress should work a rapid change in the blood-vessels, the inference is a sound one, that if the disorder cannot have altered the blood-vessels, these must have given rise to or be connected with the nature of the disorder". This, by the way, is in support of Beneke's observations.
The large size of the arteries explains the existence of hyperaemic condition of the bones, especially at the epiphysial junction, & of the increase of development & thickening of the bones after the morbid process ceases.
The large arteries induce a low blood pressure, & therefore there is retardation of the circulation both in ~~in~~ bone &

muscle, & the latter becomes flabby & feeble in consequence.

Regarded in these lights, it would seem that Beneke's observations are very important in elucidating the predisposing causes of rickets. (Abstract from a paper by C. N. D. Jones on Rhachitic Deformity, Annals Surg. Vol. IX. pp 241–271).

Disorders of Digestion:–

Dr Cheadle (Vide. Allbutt Vol III. 1897) states that disorders of digestion appear to play a part in a number of cases; at any rate symptoms of gastro-enteric disorder, flatulence, vomiting, diarrhoea, & offensive stools – not infrequently precede the distinctive signs of rachitic change. They are not, however, constant accompaniments; numbers of children become rickety who have no such antecedent gastro-intestinal disturbance, & numbers who do suffer from it do not become rickety. Further when the disturbance is extreme, & vomiting & diarrhoea are severe & prolonged, the

the result is not rickets, but general atrophy; & examination after death in fatal cases shows little or no evidence of the characteristic changes in the bones. It appears, then, that digestive disturbance of this kind is only effective when it is not extremely prolonged & excessive, & when it coincides with particular faults of diet. It probaly acts by removal of certain special elements which are the least quickly & rapidly digested & absorbed. Digestive disturbance is not, therefore, an invariable & essential factor in the production of rickets.

Dr. K. Elze. of Berlin in his book on "The nature of Rickets", 1897. (Vide, Review, Brit. Med. Jour, Sept. 18, 1897). advances the view that the dyspepsia so comonly present in rickets & manifested by the large abdomen is not merely a concomitant symptom, but the actual cause of the disease, the other manifestations being secondary to it. The dyspepsia he regards as due to an insufficient quantity of proteid food. The blood is

rendered poor in proteid bodies, the glands suffer thereby, & deficient power of digestion results, with a still further diminished absorption of proteids. The deficiency of proteids in the blood, by affecting the haematopoietic tissues, produces the various changes which he describes in the blood, bone marrow, & lymphatic glands.

Faults of Diet — Errors of diet play so conspicuous & obvious a part in this connection that they concentrated upon themselves most of the attention that has been given to this subject. Drs Garrod & Fletcher state that undoubtedly this is the greatest factor of all.

Dr Cheadle, in introducing the discussion on rickets at the meeting of the Brit. Med. Assoc, in August, 1888, stated that the food factor is the only factor which is anything like constant. In Allbutt, Vol iii, 1897. He states that the vast majority of cases of rickets arise in connection with errors of feeding. The fault of diet is not only the most common & potent cause, but

Sometimes it is the only cause.
Rickets is produced as certainly by rachitic diet as is scurvy by scorbutic diet.
This is seen in those by no means uncommon cases where children born healthy & of healthy, well-to-do parents, brought up under perfect hygienic conditions so far as air, light, cleanliness, & warmth are concerned, yet become rickety when brought up on artificial food. The only fault we can discover is the dietetic fault; & such cases are cured by a correction of the diet, without any other change of hygienic conditions; they are cured in fact, by anti-rachitic diet as certainly as scurvy is cured by anti-scorbutic diet.
The effect of diet was shown in the most striking manner by Mr. Bland Sutton in the case of rickety animals at the Zoological Gardens; these animals got rapidly well on a change of one condition only, namely, of food.
Directly or indirectly, food is probably an invariable factor. The fault, moreover,

is one of quality rather than of quantity. A child may be reduced by starvation to the last stage of atrophy, & yet not be rickety; & conversely, it may be over-fed, fat & gross, & yet extremely rickety. Drs Garrod & Fletcher in "Brit, Med, Jour, Sept 21, 1895, state "We are inclined to believe that under certain circumstances the excessive administration of good cow's milk, beyond the amount which the child is able to assimilate, may produce rickets even in the absence of obvious digestive disturbance.

There is a special fault of diet, one which produces a special defect of nutrition, & not necessarily general malnutrition. In this respect, it has already been noted that, in this country at any rate rickets is rare amongst sucklings. The only instance within Dr Cheadle's experience, of rickets arising in a child while at the breast during the first ten months of life was one in which the mother became pregnant during lactation; the suckled infant became rickety, the foetus unborn escaped; rickets, then,

may arise if the mother's milk be insufficient or otherwise defective. Such cases are, however, undoubtedly rare, Even with congenital Syphilis at work, as before mentioned, the child at the breast does not become rickety. If breast-fed children become rickety it is after weaning; the disease occurs almost entirely amongst children brought up by Hand.

The exact nature of the diet fault which lies at the root of the rickety condition has been the subject of many hypotheses & much controversy.

Certain broad facts, however, have been established with regard to it, which throw great light upon the matter.

In the first place, children fed almost entirely upon farinaceous preparations — oatmeal, corn flour, bread, patent foods, with little or no milk, even if such diet produce no digestive disturbance — certainly become rickety.

Dr Buchanan Baxter (Quoted by Dr Goodhart, in "Diseases of Children" 5th Ed, 1894,

p 646, & Dr Eustace Smith in "Disease of Children" 2nd Ed, p 137) made some most careful enquiries in the hand-feeding of infants, amongst the out-patients at the Evelina Hospital; he tabulated one hundred & twenty consecutive cases of rickets, & the result was not less than ninety-two per cent, of the whole number had been given farinaceous food before the age of twelve months & in many of them the disease dated from the time it was first given. Similarly in the case of animals Mr. Bland Sutton observed that the young monkeys at the Zoological Gardens in London, if deprived of their mothers milk & fed entirely upon vegetable food, chiefly, fruits become rickety. Two young bears fed exclusively upon rice, biscuits, & raw meat, of which latter they hardly ate, died of extreme rickets. It is not a diet limited to vegetable food only which is associated with rickets. The artificial production of rickets in young animals by Guérin (Vide

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Mémoires sur les Difformités due Système osseux, Paris, 1859; also quoted in the "Cyclopædia of the diseases of children" Keating, Vol ii by Drs Barlow, Bury) who kept puppies on a meat diet for 4 or 5 months, which showed all the signs of rickets, although impugned by the later experiments of Tripier L. (Mem, et compt, rend, Soc, de Sc, méd, de Lyon, 1863, iii, pp, 199-208; Arch, de, Physiol, norm, et path, Par, 1874, 2, S, i. pp, 108-125. also Keating, loc cit.) on cats, dogs, & chickens & failed to produce rickets, has been confirmed by experience at the Zoological Gardens. For many years the lion whelps have been weaned early, & put upon a diet of raw flesh only; they have invariably become rickety to so extreme a degree that it has been found impossible to rear them. The condition is a true rachitis; there is the same feebleness of muscle, the same debility, laxness of sinew & bending of bones; & the identity of the morbid changes has been fully established.

I have had recently under observation, a kitten which has developed the signs of rickets, History, after being taken from its mother, it could not be induced to take milk, but would take flesh meat, four weeks after being weaned, it was noticed to be very quiet, constantly lying down, & seemed afraid to jump off a chair, then it began to walk with a peculiar gait; on examination, I found the epiphyses enlarged especially those of the front legs, which were quite bowed, the muscles were soft & flabby, After a time, it began to take milk, & now it seems quite strong, & healthy, but dwarfed, & the legs deformed.
The potency of such diets in the production of rickets has led to many hypothetical explanations of the exact nature of the defect in them; whether this be negative or positive, the want of some necessary ingredient, or the presence of some noxious ingredient which perverts nutrition, especially that of growing bones.

The explanation which at first suggested itself was that as the bones are soft & deficient in mineral matter, & especially in lime salts, a want of lime salts in food is the cause of the deficiency in the bones. Sir Wm Jenner, in lect iii, says this view is altogether erroneous, & seems to be proved by the fact that not only is there an insufficient deposition of the lime salts in the growing extremities of the long bones, but there is an error in position of the small amount deposited there. The earthy salts are found in the cells instead of the matrix. And yet further, not only is there an insufficient quantity of the lime salts & error in position of those present, but there is absorption of those deposited ere the disease begins; for bones previously hard, soften. The lime is taken up from the well-constituted shafts of the long bones, & from the flat bones, enters the blood, & is thrown out of the system in the urine. The agents concerned in the nutrition of bones not only take the lime from the blood, but they take the lime from the bones.

Chossart & Milne Edwards (quoted by Cheadle & also by Drs Judson & Bury, Keating Vol ii) in 1842 experimented with pigeons & animals by giving food deficient in lime & produced curvature of the bones & obtained easily breakable bones, but Friedleben (Jahrb. f. Kinderheilkunde, B. iii, H. ii, pp 61-137, pp. 147-148. also quoted by Eustace Smith, Barlow & Bury) repeated the experiments & proved that while fragility of the bones resulted, the characteristic changes of rickets were found wanting. Others, however, as Voit, E. Sitzgber, d. Gesellsch. f. Morphol. u. Physiol, in München, 1889-1890, v, 101. & Baginsky. Lehrbuch d. Kinderkrank. (Vreden's Sammlung). B. vi, p. 270 et seq. (1883), claim to have established the existence of true rickety change by such treatment. Drs Barlow & Bury (Vide Keating Vol ii p 246) state that other considerations show the untenability of such hypothesis. Thus (1) Slight but characteristic rickety changes, such as increased vascularisation, proliferation of cartilage, etc,

are met with when the new-formed bone, is quite normally calcified.

(2) Kruchenberg has shown that the ashes of uncalcified cartilage consist principally of lime; hence marked proliferation of cartilage would be quite impossible with a due supply of lime.

(3) In the mino degree of rickets the calcification of cartilage, covers a much greater area than normal.

(4) Cows milk is much richer in lime salts than human milk, & yet is more prolific in rickets; & whatever the food. it would be impossible sufficiently to decrease the supply of lime to produce the great poverty found in bones Severally affected by rickets.

(5) Finally, the therapeutic administration of lime preparations is not curative, while without any treatment Spontaneous cure may take place although the child continues to eat the same food.

Dr Cheadle (Vide Allbutt, Vol. iii, 1897) States that, while admitting that rickets

may be produced artificially in animals by absolute privation of lime, yet that the want of lime, at any rate in the form of hydrate or carbonate, is not in itself the essential cause of rickets, as we see it in children, is proved by conclusive evidence. First there is the fact that rickets is extremely common in the limestone districts where the drinking water is so heavily charged with lime that the children must necessarily take abundance of it; moreover numbers of children become richety who have lime water regularly added to their food. Secondly, there is the fact, according to Dr Luff's analysis, that foods upon which children are especially liable to become richety, such as the farinaceous food for ~~analysis~~ instance, are rich in lime & also in phosphoric acid; & cows milk is richer in these than is human milk, So that not only will abundance of lime salts in the food not prevent the development of rickets, but as a matter of fact the disease is usually associated with a full supply of these materials.

The lactic acid Theory of rickets.

The close association of rickets with a farinaceous diet suggested the idea that lactic acid might be the evil agent; Starch imperfectly digested, ferments & lactic acid is formed in excess, which, by uniting with the lime about to be deposited in the bones, is supposed to carry it off in soluble form.
Heitzmann, Sitzgsber, d, kais, östr, Acad, d, Wiss, in Wien, 1873, No. 17.) & also quoted by Jenner, Smith, Goodhart & Cheadle.
Put forward another view of its action is, it irritates the ossifying tissue & stimulates growth when the material necessary to complete the structure is wanting.
Lactic acid is said to have been found in the tissues of rickety animals & in the urine (Marchand, Lehmann, Gorup) but Newbauer failed to find it in the urine of a case of extreme rickets, & Luelzer, in 1883, proved that neither lime nor phosphoric acid is present in excess in the urine of rickets
Heitzmann claims by its administration

to have produced the condition directly. This formation of lactic acid, however, has not been confirmed; & the hypothesis is rendered improbable by the fact that rickets arises in children in whom there is no apparent dis-order of digestion to form lactic acid fermentation, who digest the starch or maltose thoroughly, & even were unduly fat upon it.

Moreover, according to repeated observations of my own, writes Dr Cheadle, the rickety states dis-appears, & health restored whilst the farinaceous diet is continued unchanged except by the addition to it of certain nutritive elements in which it is deficient. These clinical experiments show conclusively that the starch cannot of itself be actively harmful. Again, rickets arises in animals fed on a diet - such as lean meat alone - which is not productive of lactic acid; & lastly, if lactic acid did exist in the blood it would be at once neutralised by alkali there. The fault in diet which is the chief factor in the production of rickets is clearly, neither deficiency of lime nor an excess of starch nor lactic acid generated from it.

Table I

Showing the proportion per cent. of the different elements in various kinds & preparations of milk. (Vide "Artificial Feeding of Infants" 4th Edition, 1896, Cheadle

Elements	Human	Cows Milk							Asses	Goats
	(Luff)	Fresh (Average from analyses by Payen, Goup-Basanez, König, Blyth, Attfield, & Whitelegge					Condensed (Werner & Rohler)			
		Pure	Diluted with two parts water	Diluted with equal quantity of water	Diluted with 1 part water & 2 parts milk	Artificial human milk	Pure	Diluted with 4 parts water		
Nitrogenous elements & proteicls (casein albumin	2.35	4.374	1.458	2.187	2.916	2.37	26.1	3.26	1.7	4.5
Hydrocarbons or fats	2.41	3.499	1.166	1.749	2.332	4.46	12.8	1.16	1.4	4.1
Carbo-hydrates { Lactine	6.39	4.403	1.467	2.201	2.934	5.02	16.0	2.0	6.4	5.8
Carbo-hydrates { Cane Sugar	—	—	—	—	—	—	27.0	3.37	—	—
Lime Phosphoric anhydride Other constituents of ash	.019 .026 .295	.671	.234	.351	.468	.57	4.03	0.5	—	—
Water	88.51	87.053	95.675	93.512	91.350	87.38	14.07	89.27	90.05	85.6
Total	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0

Table II

Showing the percentage per cent. of the different constituents in certain artificial farinaceous foods & preparations.

Elements of Food	Standard of Human Milk	Bread Jelly: Pure Jelly	Bread Jelly: One tablespoonful mixed with 4g of cow's milk & 4g of water Dr Luff's analysis	Bread Jelly: 4½ parts of mixture with water only, & 1½ parts of raw meat juice & ½ part cream Dr Luff's Analysis	Malted Food partially dextrinised: Pure Food	Malted Food partially dextrinised: Mixed with 7½ parts water & 7½ parts cow's milk to one part of Food Dr Luff's Analysis	Malted Food more highly dextrinised: Pure Food	Malted Food more highly dextrinised: ½ a tablespoonful of the food with ¼ of a pint of water & ¼ pint of cow's milk Dr Luff's Analysis	Malted Food with dessicated milk: Pure Food Dr Luff's Analysis	Prepared entire Wheat-Flour: Pure Flour
Nitrogenous Proteids	2.35	2.73	2.705	2.711	15.35	2.723	5.43	2.187	9.62	13.15
Hydrocarbons & fats	2.41	0.50	1.695	3.632	1.08	2.009	0.16	2.101	4.75	—
Carbohydrates of various forms: Lactine or Sugar of milk	6.39				1.02					
Grape Sugar	—	Total Carbohydrates	Total carbohydrates	Total carbohydrates	—	Total Carbohydrates				
Maltose	—				10.78					
Dextrine	—				64.12					
Starch	—	15.24	5.618	2.953	1.36	77.28	6.211	87.12	3.946	80.02
Cellulose	—									Starch 50
Lime	.019	.05	.08	.01	.10	.11	.14	.11	.30	.10
Phosphoric Anhydride	.026	.29	.11	.13	.142	.16	.71	.14	.38	.59
Other ash	.295	.17	.149	.11	.40	.168	2.68	.175	.71	.33
Water	88.51	81.02	89.643	90.454	4.37	88.619	3.76	91.341	4.22	—
Total	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	= 100.0	

An examination on the one hand of the foods on which children grow rickety, & on the other of the additions to diet by which the condition is cured, throws light upon this point, certain defects appear to be constant. An analysis of the foods on which rickets is most frequently & certainly produced – such as various farinaceous foods, domestic or patent, with a small amount of milk, skim milk, condensed milk, artificial foods with desiccated milk, & the like – (Vide Tables I & II) show invariably deficiency in two of the chief elements so plentiful in the standard food of young animals, namely, animal fat & proteid. The only exceptions to this conclusion appear to be the cases in which the foods that do contain a sufficient quantity of these elements – as cows' milk, for example – produce digestive disturbance, vomiting & diarrhoea, which disturbances lead to the loss of much of the material ingested. In such cases, moreover, the elements most slowly digested, those,

that is, most slowly brought into a fit state for absorption - namely, the fat & caseine - would be most largely drained away. Not only so, but to substitute a food deficient in these more hardly digested fats, & proteids, which, to ease the digestive faculty, is almost always always done, is to reach the same pathological result by a different route. At the Zoological Gardens the food on which young bears & monkeys become richety - namely, biscuits, rice, & fruits - is markedly deficient in proteid, & fat is practically absent. The food of the lion cubs, which became richety on a diet restricted to raw flesh, was ~~absent~~ almost destitute of fat, & was poor ~~almost~~ also in earthy salts, although rich in proteid. The meat was that of old horses almost destitute of fat, & once a week lean goats flesh. The bones were found to be proof even against the teeth of the adult lions, & those of the cubs were powerless against them, so that the cubs got from them neither fat, marrow, nor earthy phosphate. In this case the diet was not deficient in proteid,

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but in fat & earthy phosphates. The history of these lion cubs is very significant: with the exception of a single litter, suckled by the dam ten years before, the cubs brought up on horse-flesh in this way invariably died – the cause of death being, as invariably, extreme rickets. More than twenty litters had been lost in this way. The feeding of the last litter of lion cubs was begun in the usual fashion. The dam had very little milk, & at the end of two weeks the cubs were weaned entirely, & were then put on horse-flesh as usual. They quickly became rickety, & when seen at this juncture the muscular weakness, as well as bone deformity, were extreme. The malady advanced rapidly & one cub died. Then, by the advice of Mr Bland Sutton, milk, pounded bones, & cod-liver oil were added to the raw meat, which was continued exactly as before; they were kept in the same dens with the same amount of warmth & light & air, &, with the single exception of the addition to the diet, no change of any kind was made in the regimen.

The change in nutrition which followed was immediate & remarkable; in three months all signs of rickets had disappeared, & the animals grew up strong & healthy — a unique event in the history of the Society. The experiment seems a crucial one, & decisive as to the part played by fat & bone salts, with some caseine & lactine, in the production & ~~cause~~ cure of rickets. That rickets frequently follows the prolonged food vomiting & diarrhoea provoked by cows milk, is consistent with this estimate of the effect of deficiency of fat & proteid in its production. For as the fat must be emulsified & saponified before it can be absorbed, & as the proteid in like manner must be converted into peptone, these elements would be drained off rather than the lactine & salts, which are in solution & ready for immediate passage into the circulation. The result would be a great privation of fat & proteid.

Dr Cheadle (Vide Allbutt Vol iii. 1894.) states that of the three elements of food

the imperfect supply of which is found to be associated with rickets, fat is probably the most frequently, if not invariably deficient, The abundance of fat in milk, of the whole solids of which it forms one-fourth, points to the extreme importance of it in the nutrition of growing animals. If fat be removed from the milk, as in "Skim" milk, rickets follows. The curative power of cod-liver oil is evidence in the same direction; & it is interesting to find in this connection, as recorded by, M. Remy, that in Japan, where oils of fish enter largely into food, & children are kept partly at the breast up to five years old, rickets appears to be unknown, Animal fat probably serves some special purpose in the nutrition of the growing structures of a young organism. It is found in all cells, it is probably essential to all cell life & growth, & it is further evident that fat formed in the body out of the carbo-hydrates cannot, for structural purposes, replace the

animal fat supplied in food.
Phosphate of lime, again, is essential to every tissue, & in rickets is found to be deficient in the bones & viscera.
Proteid, again, is essential to the vitality of protoplasm, & indeed to the activity of all vital processes, & is therefore essential to the proper use of the other elements; but if it be in excess while the other elements are deficient, it may actually intensify the progress of rickets by stimulating the process of tissue development which, in the want of other materials, cannot be duly carried out. Therefore a diet deficient in the elements of animal fat, in some cases also, deficient in proteid & earthy salts - one or both - would explain not only the fattiness of bone, but also the feebleness of muscle, the anaemia, the catarrhal tendency of the mucous membranes, & the nervous irritability. All tissues - not bone tissues alone - are ill-nourished if the above structural elements are deficient.

Summary

The general pathology of rickets may, perhaps, be summed up as follows:- Rickets is a disease in which all the leading structures of the body suffer both from defective & perverted nutrition, so far Pathologists are agreed; there is, however, some divergence of opinion as to the exact causes of this defect by the different Authors mentioned. The increased vascularity of the ossifying cartilage, & the excessive proliferation of cells observed in rickety bone, is suggestive of a sub-acute or chronic inflammatory condition; & this view is supported by the experiment of Kassowitz, who produced a state of bone corresponding to that of rickets by inducing hyperaemia in the limb of a growing animal by means of repeated application of an Esmarch's bandage. The increased fibrosis & cell proliferation observed in the liver & spleen in certain cases is consistent with this observation, although

It is to be noted that these changes have not been found in all instances or in other tissues undoubtedly affected by the rickety condition. The agent in human rickets has been referred to some irritant derivative of food circulating in the blood. It has been shown that lactic acid derived from carbohydrate food cannot be ~~the~~ regarded as the peccant matter; but it may possibly be something formed from altered materials present in excess, because unused in the disorganised condition of the formative process. The profuse sweatings are suggestive of a like cause; while on the other hand the absence of pyrexia seems opposed to this view, although not conclusive against it.

It is possible that the abnormal vascularity & cell proliferation are the results of the impetus of the formative process left unsatisfied in its normal direction for want of materials for the building of the permanent structure.

With regard to the microbic origin, Hazenbach believes it to be an infectie process, & looks upon other causes as predisposing only.
Karowitz under certain conditions is disposed to admit various micro-organisms as the cause of rickets
Mircoli pleads for the microbic origin of rickets, believing that the disease is caused by the effect of Streptococci & Staphlyococci.
Ettore also holds this view, & concluded that rachitis is an infectious Staphylo-strepto-coccic osteitis.
Chaumier is of opinion that it is of an infectie process.
Congenital Syphilis, with the exception of Parrot, who believed that rickets was always the consequence of an hereditary syphilitic taint, authors do not look upon it as a direct cause, but consider the disease is modified in character by the concurrent existence of congenital Syphilis.
The same may be said of scurvy.

The tubercular diathesis does not seem to have any predisposing effect. in fact, Dr Eustace Smith states that a pronounced tubercular disposition appears to have a protective power against rickets.

Inherited tendency, there is no satisfactory evidence, on this point, but the general opinion seems to be that it does not play any important part in the disease.

Other factors mentioned, such as, Climate, Season, Locality, Race, Sex, Maternal (Tilbury Fox considered that menstruation during lactation to be one of the most important of all the causes of rickets)

Hygienic conditions, such as want of light, warmth & pure air etc, are considered by Bristowe, Barlow, Bury, Smith, Gee, Jenner, Garrod, Fletcher, Rasowitz, Ritter, Ritchie & others, to favour the development of the disease, & aggravate it in degree, but are not absolutely essential factors.

Disorders of digestion. Elze considers this to be the actual cause, others look upon it, as predisposing.
Faults of diet. Drs Garrod & Fletcher state that this is the greatest factor of all. Dr Cheadle says it is the only factor which is anything like constant, & consists in a deficiency of certain elements in food, the chief & constant defect appears to be an insufficient supply of animal fat, & therewith also, in certain cases, a deficiency of earthy salts in the form of phosphates; at the same time, if animal proteid be deficient the disease is intensified, In some cases where the fault of diet is not sufficiently great to cause it unaided & alone, the appearance of the disease is determined by the other factors.
Lastly there are facts in the disease which are remarkably suggestive in attempting to frame a pathological conception of the conditions which determine it; & chief of these is the most remarkable

fact, that rickets, quâ rickets, invariably recovers if treated properly – that is, essentially, if the child be put upon a proper diet. There is, perhaps, no other argument of equal force in favour of the disease being due to something which is withheld – in favour, that is, of the disease being dietetic.

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