A consideration of the Symptoms, Morbid Anatomy and Etiology.

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by

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Rickets

Synonyms — English, Rickets;
Latin, Morbus Anglius, Articuli Duplilati;
German, Die Englische Krankheit
Doppelflieder Zwiewuchs;
French, Rhacitome, Maladie Anglaise;
Italian, Rhacitide.

History. — The name primarily derived from the Old English verb "withen,
to twist or twist away, arose no doubt from the application of a term commonly applied in the country to inanimate objects, to a disease whose most striking characters are contortion and deformity.
The ill-favoured 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 "stiled."
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 "Rhacitis,
partly on account of the similarity of the sound, partly as a derivation from the same Greek "skēxis", a spine, on
the ground that the dorsal spine is one of the first parts attached. This, rather than Trousseau's derivation from the Norman word "riquets," appears to be the correct origin of the English name. Rickets was recognized 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 civilization, and 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 and 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 and pressure, thus giving rise to various distortions and deformities.
The evolution of the teeth is delayed, their structure is fragile, imperfect, and subject to early decay. This striking affection of the osseous skeleton has concentrated attention upon one special part of the disease, and 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 and pathology considered almost entirely in this relation. The effect of growth and nutrition is, however, by no means limited to the bony framework of the body; all the chief structures are involved.

The muscles are wasted and 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 and convulsion are readily excited. The blood is impurify
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 box-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 outward form, the sickly 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. Dr. 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 occasionally 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, subnormal, unless there be concurrent scurvy or some inflammatory complication, such as broncho-pneumonia.

The abdomen is prominent owing partly to depression of the rectum, 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 and offensive; sometimes, in the absence of biliary colouring matter, almost white; sometimes green, sometimes of darker colour, and slimy.

There is a tendency to bronchial and laryngeal catarrh, so that bronchitis and laryngitis are frequent complications. Nervous symptoms form a marked feature of the disease. Sir J. Jenner first noted the exceptional tendency to convulsion, a close dependence 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.

Dyspnoea, stridulus, tetany, and general convulsions stand in special relation to the rickety 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, and 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; or 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, it must always be regarded as a significant indication that the condition of the nervous system has become unstable. It is one in which convulsions would readily be set up.\footnote{Tetany—} is a curious state of painful muscular contraction, \footnote{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 pickety subjects are prone.

It is true that tetany occasionally arises in adult life as a sequel of exhausting diseases, as in women during lactation, \footnote{as a sequel to diarrhoea \& typhoid fever.}
Such cases are, however, comparatively rare, for tetany is especially an affection of early childhood, if of the rickets state. (In the Brit. Med. Jour., May 1, 1897, epitome (350, pp. 72)) Hassoitz, at the Wiener Medicinisher Club (Neuro. Centrabl., March 1897), showed a child with latent tetany and 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 unproven; Hassoitz has never seen tetany occur with chronic gastro-intestinal catarrh in the absence of rickets, and the seasonal variations of tetany are exactly the opposite of those of gastro-intestinal disorders, tetany being most frequent in the winter, while the gastro-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 pachitic
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 is some noxious substance entering by the respiratory tract from the foul air in the dwelling 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 accoucheur's 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 adducted, flexed, and overlapping. The forepart of the sole of the foot is hollowed and 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 episiotonus, occasionally those of the jaws, causing trismus; if this does ever occur, it is rare, beyond the experience of Dr. Cheadle (Vide Albutt, 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 AlburtVol., 1897, in
which anodal "tetanus" has been ob-
served in man. It is said this increased
nerve-muscular irritability may some-
times 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
Cyanogenia Stridulus with rickets.
I., aged 4 years 9 months, Male Sex;
present condition, pale, anaemic, feeble,
very thin, chest bulging, with enlarged
joints at the junction of the cartilages
of 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 and five months.
Cariesimus Stridulus, first came on at the age of four months, but no other attack until three years and nine months old, which I witnessed, and two more since. These, along with the unmistakable signs of rickets, suggest the case, as one commencing with that disease; tetany & cariesimus Stridulus following.

The symptoms of rickets, according to Sir William Jenner, Bart., C.C.B., in his clinical lectures (1895), are:
- A general cachexia which very rarely manifests itself before the fourth month, usually between the fourth and tenth months.

One case a boy was three years old below 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 excitement.

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 pale and steady appearance; its natural lively expression is replaced by a pensive, sad, languid aspect; its face grows broad and square; when placed upright, it sits "all of a heap," its spine bends, and 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, and 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 be accounted for by their diminished size.

The abdomen is larger than natural, because the chest is smaller & the diaphragm more depressed than in health.

The liver & spleen are often larger than natural.

Indeed, the capacity of the pelvis is diminished; the muscles of the abdomen & intestines are less powerful even than they are in their normal condition; moreover, derangement of the digestion is always present to favour the excessive formation of phlegm.

Writers state that the intellect of the sickly 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 idiocy, they resemble rather children of low intellectual capacity & power much younger than themselves, & it retrogrades as the ricketty 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. F. Judson, S. Bury, M.D. R. C. P. in the "Cyclopaedia of the diseases of Children. Heating. Vol. II." state that the earliest manifestation of rickets consists in beads at the juncture of the ribs with the costal cartilages (rosary), & 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, 4 round spots of local thinning may be detected on occipit, 9 parietals, 7 even exceptionally on the frontals, to these Elsaesser, who first discovered the condition, gave the name of Craniotales, 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 9 parietal bones, in front and behind the anterior fontanelle, these masses of soft, vascular, bony growth 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 secondary variety or type of rickettsy skull is dolico-cephalic, the frontal region, not very broad, is prominent in the middle portion, the top of the skull is flattened, upper part of occipit projects considerably. Whatever shape, the head is generally larger than a head of healthy child of same age.

Heinrichmann 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 occasional 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. Cliby, M. J. Lond., F. R. B. S. Eng. in his book on "Pneumonia," 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" if some bending of the bones may be seen at this stage, he quotes Dr. Gee, who states that a child who is not idiotic a weakened by some recent disease, who cannot walk at the age of eighteen months, is either rickety or paralysed. The complexion is of a peculiar earthy tint, the child sits in a heap. At this time, too, "beading" of the ribs and enlargement of the radial and ulnar epiphyses are found. The spleen occasionally the liver are felt to be enlarged, the abdomen is distended.
At the annual meeting of the British Medical Association held at Edinburgh, 1895, I spoke of the occurrence of a pad on the dorsum of the foot in rickets (Bid Med Jour Oct 15, 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 I 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 for 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 independently 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 epiphyseal lines. I 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 affection of the bones could be felt. Of the 86, 20 cases came under this category; the average age being 14 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, & solidly oedematous to the touch. Of the 86 cases, 49 presented this appearance; the average age was
27 months, the average duration of the attack was 11\textfrac{3}{4} 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 27 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. I 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, very 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, it is made up of thickened subcutaneous tissue and 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 and undue pallor of the skin, I leave the bony changes well-apparent to the touch.
Such are the symptoms and physical character of rickets in its full and complete expression. All these features are not, of course, present in every instance. The disease is met with at every degree, from the smallest indication 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. And 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-bends constituting the earliest signs met with in the foetal state may be found alone in the early stage.

The concurrence of head sweats, or cyanismus, 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 per-
haps be present; but the concurrence
of all in the same subject is extreme-
ly rare.

Morbid Anatomy. —
Dr. W. Jenner describes the bone changes
as exhibiting extensive preparation
for ossification & imperfect per-
formance of the process.
Dr. Cheadle (Vide Allbutt Vol. III. 1841)
observations agree in the main
with the opinion that, as shown
by Kassowitz, the pachyderma process
is not merely a defective ossification
but is accompanied by a hyperplasia.
Sir Wm. Jenner, in his Lecture on 1898, says that W. Paget, in expounding 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 excudation 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 a 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 intimate nature. But, as little as we have progressed, we certainly are in advance of those who still regard rickets to be merely a chemical abnormality of the bones by a deficiency of the earthy salts.

Dr. Gustave Smith in his second edition on "Diseases of Children" says the changes in the bones are affected in three ways, growth retarded and irregular ossification of part still remaining cartilaginous interposed with a 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 points of junction with the various cartilages.
Mr. A. H. Sulzer, in his book on "Rachitic Defaminities," says the bones in severe cases pass through three stages: (1) Stage of congestion. (2) Stage 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 follows:

1. The Cranium (Vide Albatt 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 "craniotubes" of Elhassar, are perceptible in the parietal & occipital bones; small bosses or swellings form symmetrically on the frontals, on the parietals & occipital & even on the temporal bone.

The atrophic lesions of craniotubes 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 & in some cases elonged, the parietal & occipital protuberances being well marked; a groove runs along the site of the sutures 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. I. 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, 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 and 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 i. 1893, says the ricketsy head is distinguished
(1) Anterior fontanelles remain open long time (healthy child, closed before end of 2nd)
(2) Thickening of bones, just outside sutures (sutures deep furrows)
(3) Relative length of antero-posterior diameter.
(In deceit both two types are given by Dr. S. Barlow & Juddon. S. Bury. 727 the
polyhedral, in its antero-posterior tracing, with a broad square forehead & flattened crown.
The second form 'dolicocephalic', in which the fronto region is not very broad, is prom-
inent 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
obstruction of the bones, the two last chiefly to disease of the cerebrum.
On 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 are developed from
below upwards, e.g., inferior extremities before trunk, but this is not the case.

W.锡尔 remarks that the circumference of the head is often much increased in some cases owing to hydrocephalus. (In the discussion on pachycephalus at the London Pathological Society in 1881, an opinion was expressed that psychic 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, it gives the impression of the so-called "egg-shell crackling"; this condition known as craniotabes, is met with also in congenital syphilis.

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 rickety 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 fullness of the lateral aspect of the frontal bones, obliquity of the upper wall of the orbits, prominence of the eyes, are characteristic of rickets. In the parietal regions the eminence on the central part of these bones is exaggerated, taken with the enlarged frontal eminences, the natiform appearance results. This is, however, better marked in congenital syphilis.

In the face the chief error of development is in the lower jaw. Mahins observes (vide Virchows System of Surgery, vol. i, p. 368) the development of the outer wall of this
bone being defective, interfaces with the acquisition of the proper arch formed by the body, the incisor teeth being arranged transversely, 9 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 (Heinichmann). 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 M. Jenner Lezi) is modified by the softness of the rib bones & their yielding to pressure, & the great determining cause of the thoracic deformity is atmospheric pressure, aided by the elasticity of the lungs. Politansky 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, and 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, as 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 transversely 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, and 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 "roseary" by 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, and no vestige of them remains in adult life. Posterior nodosities in severe cases jam near the angles of the ribs.
(which appear to arise from partial fractures) they are less symmetrical, vary in position on successive 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, lichety softness of bones, it is rare in children under twelve months old, except subjects of atelectasis & congenital malformations of heart. It 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 rachitic children. With displacement of the bones, there is frequently some scoliosis, so inasmuch as the subluxation is on the "convex" side of the rachitic curve, it is probable the scoliosis may be the immediate cause owing to the forward and upward tilting of the shoulder on the convex side, thus pushing the inner end of the clavicle out of place. (Vide, Tubby, "Rachitic Deformities, 1896). Gilbey (Int. Clinics, Vol. IV. 1895, p. 239) in a clinical lecture, alludes to several cases; he says "Sately we have a way of curing these that it is very good; his attention was first called to it by J. Hemson who had injected alcohol, two or three injections setting up inflammation around the articulation, then binding the part, with successful 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 & weakness of the musculature & ligaments was suggested as an explanation of the cause of rachitic torticollis. Thecau of Lille reported in the Revue d'Orthopedie 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 with- 
drawn. Later, the head was thrown back, and its backward tilting was harder to 
prevent than the lateral. Pain was not severe, but the children were febrile 
and resisting examination. The lateral deformity lasted about three months, 
and the posterior also disappeared after a time under appropriate general treat- 
ment, and the use of a rubber collar to keep the head in position.

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

Mr. Tully says, the conjugate is decreased owing to the prominence of the sacro-
vertebral angle. In many consequence, the thrust inwards of the head of the femur at the acetabulum on each side, the lateral aspect of the pelvis is 
flattened, and the pubic arch is diminished. The tubera pubis are approximate and the ventral ilium expanded by weight of viscera.
of its head 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, comprised
between the weight of the body acting
downwards from above through the spine
and the upward resistance of the thigh bone,
gives way; thus a a general result the
pelvis becomes narrowed by the pushing
forward of the sacral portion towards
the pubes, it 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 epiphysis 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 forearm become curved and twisted. The force of
gravity appears to be aided and modified by the support and traction of the muscular
attachments, which are very well marked, being drawn out during the soft stage.
In fig 158 rhachitic deformities. Subly.
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 7, 1897, two cases are
reported, which were read before the
Warren Club, Nov 3, 1896 by J. S. Stone M.D.
Boston, showing the cause of bowing
of the left forearm in rhachitic children.
Cases 1 & 7, aged 14 months, of Jewish
parentage, was brought to the out-patient
clinic at the West End Nursery on July 5, 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 roxary or bowing of the legs. The left forearm presented at the junction of the upper 3 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 11. M. E. C., aged 2½ years of Irish parentage, was brought to the out-patient clinic at the Children's 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 diarrhoea 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 roseary, 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 are 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 occasionally 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 and standing, & the femur becomes bowed in the 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 (incorvation of the neck of the femur) in the majority of cases is due to rickets infantile & adolescent. If the latter the disease is said to be "local" in its manifestation (Vide Dr. 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 and 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 Arbiton. Jane 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 Jane. Such are the general features of the bone affections and deformities in rickets.

Some of these abnormal conditions disappear with time and growth. In adult life the beads on the ribs are no longer recognizable, the enlargements of the ends of the long bones undergo more or less complete calcification, 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 bony structure 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 moebid 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 bone 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; some times 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 and destroying adjacent columns. In extreme cases this latter zone may be quite wanting, the spongy bone approaches the columnar zone of cartilage. The bone formation takes place not only around the spaces but also above and 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 and vascular. The bony lamellae are thin and friable, and it is evident that rapid absorption is going on; the medullary portion of the shaft is more vascular than normal, the inter-spaces larger. The marrow is relatively large in amount, red and gelatinous instead of yellow fatty.
The formed bone itself undergoes change; the vessels enlarge and new ones form, which press upon the septa between the medullary spaces and cause this partial absorption, so that the spaces open into each other. Thus the strength of the bone is impaired, and 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. Chedde, Vol. III, 1897) not to removal of that material as at first supposed, but according to the later observations of Harrowitz, 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.

As Dr. Jenner in his lecture on rickets says: from the experiments of Lehmann, J. Marchand, the animal matter of rickety bones, differs, in some cases, from what 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 39 parts of organic to 61 of inorganic matter; whereas of rickety children yield about 79 parts of organic to 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) it 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 shaft
of the long bones is from the flat bones, enters the blood, is thrown out of the system by the urine. The agents concerned in the nutrition of the bones not only do not take lime from the blood, but they take 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 Marchand's experiments to contain six times its 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 Dr. Barlow & J. S. Bury (vide Healing Vol ii) Gladale (vide Allbutt vol iii) Hehn & Seemann, (quoted by Godfart, 3rd ed. 1894, pp 637), have not been verified by the later observers, so that the observations of Musson, as previously stated, are most likely correct.
As the active process subsides, vascularity of the growing portion lessens, the spongy bone tissue becomes hardened and 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, and is more vascular than in health.

The outer fibrous layer is thickened, and 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 (Reinating Vol II), it varies also in density and in the degree of calcification from spongy, boneless tissue to normal osseous tissue.
The periosteal formation, which is the sole form which obtains in the flat bone, shows excessive hyperplasia of the proliferating layer and imperfect ossification of like character. But where there is a pressure, e.g., the weight of the brain upon the softened tissue, the bone does not develop, and 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 phlegmatic state more or less induration of bone—sclerosis—follows.

Microscopic examination—

According to Tully, shows the deeper or osteogenic layer of the periosteum to be chiefly affected. The layers of spongoid bone beneath to consist of calcified pellets arranged radially to the surface of the diaphysis. Beneath the pellets of the bone are large red medullary spaces.
Trexes System of Surgery, Vol. I, p. 365, says: The cartilage of the epiphyses, under the microscope, show excessive and irregular increase of the cells. In the spaces between the columns, much vascular round-celled material exists. Away from the growing line masses of calcified cartilage are seen, till further much enchondric bone is present.

J. Sims, Woodhead, in his "Practical Pathology," 3rd Edition, (after staining with Chromic acid, Osmic acid, Biernocarmine & Iodine) observes as follow (x50). In the thickened belt of cartilage, with the irregular calcareous & bone 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 and 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, \( \times \) closely following them, appear spaces similarly to those met with in ordinary bone, many of which are lined with a regular layer of pink cells or osteoblasts, \( \times \) true bone, or a structure which very closely resembles it, is formed. Even in the midst of the bone, formed in this position, masses of irregular cartilage cells may be still seen.

\((x 500)\) The above appearance when observed more closely, the great irregularity in the size of the cells, in the matrix, \( \times \) in the calcification of the matrix, the calcification of the cartilage cells at certain points, their proliferation and apparent transformation into osteoblasts. Even when true bone is formed, it appears to be laid down.
without any attempt at order, or regularity, 9 bone, calcified cartilage, 9 true cartilage are mixed up, apparently indiscriminately. Here we see then, that the chief points to be noted are the enormous 9 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) 9 partly calcified (stained green), may be seen. These trabeculae form an open network, 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 9 irregular
in structure, in some cases almost like spongy bone. In this tissue the number of vessels and 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 coating trabeculae, in the space between the osseous trabeculae. In normal bone these osteoblasts grow slowly, form around them periosteal, which gradually become calcified. In the case of rickets, however, these osteoblasts are formed in very large numbers, but any periosteal which they may form is always small in quantity and 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 and ossification go on slowly and 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, or 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 and strong from the fact that the osteoplastic tissue is present in such large quantities. The bones may be stunted and deformed, this is owing to the fact that ossification sets in so rapidly that the epiphyses unite at an early date than usual, so 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, and like the bones, yield undue traction and pressure. Thus the ligamentous structures which bind together the bones of the feet give way as the pickety child begins to bear its weight upon them, flat foot results. In like manner the ankles relax under the pressure of the feet, the feet splay out sideways; the knees ligaments give way and knock-knee results; the supporting ligaments of the vertebral column yield, it becomes concave or curves laterally; thus throughout all joints the laxness and feebleness of the binding ligaments and tendons prevail and alter shape and symmetry.

Muscles. It should be stated 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 extreme cases...
to move in bed, or even to raise the head. If the structure of the muscle be examined they are seen to be flabby and wasted. Under the microscope the striation is blurred and indistinct.

In Jenner (lect. iii) says the muscles are small pale flabby & soft; & when examined under the microscope, the fibres are found to be singularly colourless, translucent; the transverse strie very delicate, sometimes scarcely 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 anaemia 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 congenital syphilis, the fat is deficient.
The child is pyz domaciated. This emaciation & pallor (vide Jones, sect. iii), are the two great features, during life, of albuminoid infiltration. The anaemia is often most remarkable; viz., a little serosity, effused in the cellular tissue, the child has that transparent, waxy, greenish-yellow tint, which is sometimes seen in the anaemia of young women. Now when there is decided anaemia: 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, it is exemplified by the tendency to laryngismus etc. (vide Allbutt Vol. vii.)
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 more 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 tracts; with this a compensating emphysema is developed over the anterior borders, where the forward projection of the sternum removes support and promotes distension of the air vesicles.

"In 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 catarrh of the mucous lining of the bronchi's 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 pickets and 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, & the falling in of the thoracic wall (Vide Allbutt Vol. III). (due to Sternum forced forward & relative positions changed, Jenner's). White patch of thinned 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. Hodgkin's theory of the patch in ricket was due to pressure aided by movements of heart. Dr. Wilks in lectures on Pathological Anatomy termed it Attraction 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 and harder than normal. This is due to a felt diffuse fibrosis, with slight increase of cellular elements and deficiency in earthy salts, as in the bones.

The hyperplasia is probably due to the chronic hyperemia set up by obstructed circulation through the lungs, the result of contraction of the thorax combined with feeble heart power, collapse and emphysema, which retard the outflow from the portal circulation. Such hyperplasia is readily set up in childhood, a period when the somatic process is active. Dr. Cheadle has seen such fibrosis in marked degree in a child as the result of chronic bronchitis and emphysema. On the hyperplasia may be pruritue.
in origin, due to the circulation in the
blood of some peculiar material, such
as has been held to be the active cause
of hyperplasia in the bones and the
hyper-activity of the sweat glands.
In certain instances the hyperplasia
is of syphilitic origin, especially when
accompanied by great enlargement of spleen.
Dr. Jenner (in lecture iii) states that
the liver and spleen are often larger than
natural, but not the whole of the liver
infected.
Dr. Dickenson’s observations (vith Bristowe
Edition) seem to show that a similar
process, in some respects like that in the
bones, goes on in the kidneys and lymphatic
glands, especially in the liver and spleen of
rickety children, that these organs become
enlarged and indurated, the seat partly of
interstitial development of fibriloid tissue,
partly of overgrowth of the glandular elements.
The changes are transitory, are accom-
panied by a deficiency of the earthy
salts. They are quite distinct from
amyloid or carcaceous degeneration.
The Spleen & Lymphatic Glands.

In Albright, 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 cardiae 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, & firous from like interstitial & cellular change.
Sir H. Jenner in his “Clinical Lecture II on Vichets” states that the spleen, in increased in size, may be firming or 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, it 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, tolerably moist, so 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 inflamed, I am free from adhesion. There is no increase in the number of white corpuscles.

Dr. Goodhart in "Diseases of children pp. 441 states that in rickets the spleen is large, its capsule perhaps a little thick, its substance firm, pale or dark-coloured. Under the microscope the fibrous septa of the organ are thickened. He has seen hyaline thickening of the septa which might be called fibrotic in four cases which he has examined. As is well known, an albuminoid change has been described by Sir W. Jenner as peculiar to rickets, but this can only occur in the more extreme cases, it is decidedly uncommon. He has never seen it. I see it only occasionally.

On page 660, he states. The albuminoid disease of all these viscera has been described 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 fibril 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. (pp. 60)

He has only notes of forty-four cases, in twenty-four of these the phlegmatic 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 fibril nature, reveal visibly 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 136-133", says that the alterations in the liver, spleen & lymphatic glands are present only in exceptional cases.

Dr. Dickinson (quoted by Dr. Eustace Smith, Britton, 5th ed pp 951. 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 which the corpuscles are crowded together. Increase in size seems due to a chronic congestive process, which causes a large development of hyaline fibriloid material.

In his Diseases of the Kidney part ii pp 468. he says the ricketsy condition is profoundly different from the cardaceous, though the two have a superficial resemblance: it is distinguished from the cardaceous by the deficiency not of potash but of lime. The saline constituents in 100 grammes of fresh spleen are contracted in the healthy, cardaceous ricketsy, as follow.
Saline constituents in 100 grammes of fresh Spleen.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Salts of the earths</th>
<th>Salts of the alkalies</th>
<th>Composition of the salts of the alkalies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Potash</td>
</tr>
<tr>
<td>1. Healthy Spleen</td>
<td>2.74</td>
<td>1.054</td>
<td>0.322</td>
</tr>
<tr>
<td>2.</td>
<td>1.21</td>
<td>1.084</td>
<td>0.301</td>
</tr>
<tr>
<td>3.</td>
<td>1.42</td>
<td>1.092</td>
<td>0.301</td>
</tr>
<tr>
<td>Cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Extensively cardaceous. Disease of spine, with discharge.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cases</th>
<th>Salts of the earths</th>
<th>Salts of the alkalies</th>
<th>Composition of the salts of the alkalies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>121</td>
<td>772</td>
<td>194</td>
</tr>
<tr>
<td></td>
<td>141</td>
<td>982</td>
<td>124</td>
</tr>
<tr>
<td></td>
<td>258</td>
<td>658</td>
<td>218</td>
</tr>
<tr>
<td></td>
<td>201</td>
<td>886</td>
<td>245</td>
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<tr>
<td></td>
<td>354</td>
<td>699</td>
<td>203</td>
</tr>
<tr>
<td></td>
<td>Salts of the earths</td>
<td>Salts of the alkalies</td>
<td>Composition of the salts of the alkalies</td>
</tr>
<tr>
<td>----------</td>
<td>---------------------</td>
<td>-----------------------</td>
<td>-----------------------------------------</td>
</tr>
<tr>
<td>Case</td>
<td></td>
<td></td>
<td>Potash</td>
</tr>
<tr>
<td>1. Ricketsy enlargement of spleen</td>
<td>0.94</td>
<td>0.989</td>
<td>0.283</td>
</tr>
</tbody>
</table>

Ellen Smith.
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 hydrocephalus 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, or 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.
Mr. 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 or sulphuric acid,
such as are said by Virchow to be
characteristic of lardaceous infiltration.
Dr. Gee (Vide On convulsions in children
At 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 17½ lbs., the brain 59 oz.; the
average at this age being 38.4 oz.
A girl of the same age, also rickety, weighed
15 ½ lbs., the brain 42½ oz., the average being
34.97 oz. In both cases the brain appeared
to be perfectly healthy.
Dr. Mellon Yagge alludes to one case that
came under his own notice, the case was
thought to be one of advanced hydroceph-
alus, until an examination after death
showed the brain filled up the cranial cavity, and to six other cases under Dr. Fletcher Beach of the Derrynish Asylum.

Dr. Beach has found a uniform granular appearance in the white matter under the microscope, with nerve cells scattered sparsely throughout, and 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 Hutton Tagge, edited by S. H. Bye Smith, M.D. Vol. 1, p. 556, 1500.)

(Dr. Eustace Smith, in "Diseases of Children" 2nd Edition pp. 350. States that the brain is sometimes small & shrivelled, so that fluid is thrown out to fill up the space.)

Dr. Goodhart in "Diseases of Children" p. 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. I think it is rare, he has not hitherto met with such a case.
The Blood. In the Boston Medical Journal, Vol. I., p. 309, 1847, 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 forms of leucocytes are also considerably different. The limits, as given by Sandolin, are as follow: (Sahib, f. Kinderheilk. 1893, t. 7, xxxv, s. 177.)

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 mono nuclear 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 and in the amount of protoplasm. Finally, an increase in the number of eosinophilic cells, even if considerable, seems to be of less significance.
Anemia of various sorts is very common in children. It develops more easily and more frequently as the result of various morbid conditions or 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 and replaced but new ones formed. Hence any disturbance of nutrition is more serious and results in more rapid and significant changes in the blood. The classification of the anemias of children is at best a very crude one. That of Atwood 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 childhood. 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. In (more) has also noted this in the anaemia secondary to various diseases. (New York Med. Rgs., XVII, 251)

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 leui, & 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,
A number of red corpuscles, is marked with 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 leucocytes. In all but the mild anaemia without leucocytes is 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, the 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 & Al. & Weiss, is as follows: Constant diminution in number of erythrocytes; constant more or less marked diminution in haemoglobin; poikilocytosis; very many nucleated red cells mostly of abnormal form.
many showing karyokinetic figures, polychromatophilia of the nucleated red cells of many of the non-nucleated, pretty marked leucocytosis, always polymorphs. In addition there is always splenic tumour or 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, syphilis, 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. Fischel (Zeitschrift für Heilkunde. 1892, xiii, 277, 278. Heiner. Ibid. Wochenschrift. 1894, vii, 241.) has found the same type of blood in cases of rickets both with and without splenic enlargement. Moreover, cases of progressive anaemia with enlarged liver and spleen do not always show this condition of the blood. Whether the condition known as anaemia...
infantum pseudo-leukémica 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 and to a higher degree in children than in adults. While in adults the increase of white cells is almost entirely in the polymuclear 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 polymuclear neutrophiles, and 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 gastrointestinal tract, while 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, it concludes that the proportions of the leucocytes correspond to certain tissue conditions. Alterations in these are thus characteristic in 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. xlv, 401. Wiener med. Pres. 1894. xxx. 1532. Archiv. f. Kinderheilk. 1884-85, xviii, 181.) has found that in normal children there is a constant relation between the specific gravity of the blood and 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.
To 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 lepra. 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, but others without splenic enlargement showed a high grade. He concludes that pockets as such do not cause any peculiar anaemia, but that according to the severity of the
Acute process and the associated involvement of the blood-forming organs very different forms and gradations of chronic anaemia may exist.

Felsenthal (Archiv f. Kinderheilk., XV, 78.) examined the blood in 12 cases of acute between the ages of nine months to 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 and the severity of the case. The haemoglobin 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
in the severe cases. In the majority of the
white corpuscles were small and mononuclear.
Weiss (Jahrb. f. Kinderheilk., 1893, xxxv, 146).
examined the blood in six cases, he found
that all severe cases of rickets show a
typical leuco cytosis, the principal part
of which is to be attributed to an increase
of the mononuclear cells. The polynuclear
forms are also numerous, so that the
polynuclear cells appear relatively
diminished.
Gaudelius (Jahrb. f. Kinderheilk., 1893,
N. 7, 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, so
that any change in them is dependant
on concurrent involvement of the intern-
nal organs.
Luzet (Études sur les Anémias de la
Pension et sur l'Anémie Pseudo-Leu-
comique. Hèse, Paris, 1890. Archives
Générales de Médecine, 1891, p. 397.
La France Médicale, 1891, xxxviii, 771).
thinks that the blood in rickets associated with splenic enlargement may show all stages from mild anaemia to leukaemia. He thinks that the splenic tumour causes leucocytosis, increases the anaemia, and makes the prognosis more gloomy.

Notch (Pediatrics) reports the results of the examination of the blood in two cases of myelitic anaemia, one without and one with splenic tumour. The diminution of red corpuscles and of haemoglobin was more marked than in the latter. It showed no leucocytosis, however, whereas while the former did.

Hoch & Schlesinger (Centralblatt f. inn. Med., 1891, xii, 893, Hæmatologische Studien, Leipzig u. Wien, 1892), has found that the specific gravity of the blood in rickets is entirely independent of the severity of the rachitic process, but varies with the anaemia.

Pelzential & Bernhard (Archiv f. Kinderheilk., xvii, 333), have also shown that it varies with the amount of haemoglobin.
Morse (Boston Med Surg Journal, Vol. 1, 1897, p. 369) examined the blood in twenty cases of active, uncomplicated mictions in infants under the age of one. The blood was in every case taken from a lobe of the ear, and 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 and the white corpuscles in two cubic millimetres were counted.

The haemoglobin was estimated with a von Heischl instrument. Cover-slips were in all cases made at the same time, hardened with equal parts of alcohol and ether, and stained with Ehrlich's 'triple stain'. A differential count of at least 500 white corpuscles was then made. The classification recommended by Ehrlich being used. As far as possible the blood was taken about noon, and 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, and those in which there is splenic tumour. The first group comprises 9 cases; the second, four; and the third, seven. The data of the individual cases are as follows:

**Mild Cases**

**Case I. Male, negro. Eight Months.**
Breast for six weeks. Melvin's food since.

**Case II. Female Irish. Seven months.**
Breast for three months. Then Hollick's milked milk.
Head sweats. Restless at night. Tendency to diarrhea.


Case V. Female, negro, thirteen months. Breast of ton. Mellini's for eight months. Thin milk, also a little "table food". Never well. Very fussy. Head sweats a good deal. Cannot creep or stand.


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, tympany.

No variation in size or shape of red corpuscles.

Case VIII. Female, negro. Eight months.
Condensed milk one month; cow's milk, three months; malted milk, four months. Head sweats a great deal, sleeps poorly. Diarrhoea for a long time.


Three macroblasts & two normoblasts seen in counting 500 whites.

Case xii. Male, Italian. Nineteen months.


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 hypnosis, not disappearing on suspension.

Marked poikilo. Heart & lungs normal.


Cases with Splenic Tumour

Nearer pale. Parietal eminences very large.
Frontal somewhat so. Fontanelle almost closed.
No teeth. Sits straight. Slight rosy.
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. Frenchian. Twenty months.
Always cow's milk, low flour diet.
Measles at one year. Very few digestive symptoms.
Sits with marked kyphosis in lower dorsal region, which is only partially obliterated on suspension. Small chest. Marked rosy.
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. Subject to attacks of vomiting & diarrhoea. Head sweats badly. Sits alone.
7. Dr. N. Baby. Pale. Large square head. Fontanelle very widely open but not tense. No teeth.
Heart & lungs normal. Abdomen large & tense.
Liver normal. Spleen palpable. Epiphyseal 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.
Marked rosy. Heart & lungs normal.
Abdomen full. Liver normal. Spleen distinctly palpable. Large epiphyseal at wrist.
Legs held loosely & rotated outwards at hips.


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. 9 emaciated. Marked palo
with yellowish tinge.
Fontanelle not closed. Four teeth.
Nasal. Heart & lungs normal.
Abdomen very full & tense.
Umbilical hernia. Liver not enlarged.
Spleen fills left half of abdomen,
reaching nearly to median line
9 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. Occasionally
free nuclei. No abnormal forms
of white corpuscles.
The results of the blood examination
are here given in tabular form:
The figures in these columns represent percentages.

<table>
<thead>
<tr>
<th>No</th>
<th>Age (in months)</th>
<th>Type of Disease</th>
<th>Haemoglobin</th>
<th>Erythrocytes</th>
<th>Leucocytes</th>
<th>Small mono-nuclear</th>
<th>Large mono-nuclear</th>
<th>Polychromatophilic</th>
<th>Lymphocytes</th>
<th>Morphology of Erythrocytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8</td>
<td>Mild</td>
<td>60</td>
<td>4,170,000</td>
<td>9,600</td>
<td>34</td>
<td>4</td>
<td>61</td>
<td>1</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>&quot;</td>
<td>57</td>
<td>4,280,000</td>
<td>9,300</td>
<td>36</td>
<td>3</td>
<td>60</td>
<td>1</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>&quot;</td>
<td>60</td>
<td>5,048,000</td>
<td>14,770</td>
<td>61</td>
<td>4</td>
<td>33</td>
<td>2</td>
<td>Moderate variation in size of normoblasts</td>
</tr>
<tr>
<td>4</td>
<td>13</td>
<td>&quot;</td>
<td>67</td>
<td>4,956,000</td>
<td>18,800</td>
<td>24</td>
<td>14</td>
<td>35</td>
<td>4</td>
<td>Nearly normal</td>
</tr>
<tr>
<td>5</td>
<td>13</td>
<td>&quot;</td>
<td>48</td>
<td>5,024,000</td>
<td>17,900</td>
<td>47</td>
<td>8</td>
<td>44</td>
<td>1</td>
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</tr>
<tr>
<td>6</td>
<td>72</td>
<td>&quot;</td>
<td>70</td>
<td>5,144,000</td>
<td>10,700</td>
<td>52</td>
<td>4</td>
<td>42</td>
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<td>17</td>
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<td>5,102,000</td>
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<td>43</td>
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<td>4,840,000</td>
<td>10,200</td>
<td>53</td>
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<td>41</td>
<td>2</td>
<td>Slight variation in size</td>
</tr>
<tr>
<td>9</td>
<td>14</td>
<td>&quot;</td>
<td>62</td>
<td>5,538,000</td>
<td>9,000</td>
<td>32</td>
<td>16</td>
<td>31</td>
<td>2</td>
<td>Considerable variation in size. Moderate poikilocytosis</td>
</tr>
<tr>
<td>10</td>
<td>6</td>
<td>Severe</td>
<td>63</td>
<td>4,704,000</td>
<td>13,900</td>
<td>48</td>
<td>4</td>
<td>48</td>
<td>0</td>
<td>Moderate variation in size. Slight poikilocytosis.</td>
</tr>
<tr>
<td>No.</td>
<td>Age (in Months)</td>
<td>Type of Disease</td>
<td>Hæmoglobin</td>
<td>Erythrocytes</td>
<td>Leucocytes</td>
<td>Small mononuclear</td>
<td>Large mononuclear</td>
<td>Polynuclear neutrophiles</td>
<td>Eosinophiles</td>
<td>Morphology of Erythrocytes</td>
</tr>
<tr>
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</tr>
<tr>
<td>11</td>
<td>2</td>
<td>Fever</td>
<td>52</td>
<td>3.308.000</td>
<td>9.200</td>
<td>69</td>
<td>05</td>
<td>23</td>
<td>1</td>
<td>Marked variation in size of polychromatic normoblasts.</td>
</tr>
<tr>
<td>12</td>
<td>19</td>
<td>&quot;</td>
<td>67</td>
<td>3.290.000</td>
<td>7.200</td>
<td>29</td>
<td>14</td>
<td>53</td>
<td>1</td>
<td>Slight variation in size of pale polychromatophiles.</td>
</tr>
<tr>
<td>13</td>
<td>24</td>
<td>&quot;</td>
<td>64</td>
<td>4.604.000</td>
<td>8.500</td>
<td>39</td>
<td>15</td>
<td>45</td>
<td>1</td>
<td>Nearly normal.</td>
</tr>
<tr>
<td>14</td>
<td>4</td>
<td>Spleen palpable</td>
<td>77</td>
<td>4.724.000</td>
<td>22.000</td>
<td>63</td>
<td>4</td>
<td>31</td>
<td>2</td>
<td>Nearly normal.</td>
</tr>
<tr>
<td>15</td>
<td>20</td>
<td>&quot;</td>
<td>73</td>
<td>4.448.000</td>
<td>14.400</td>
<td>49</td>
<td>6</td>
<td>40</td>
<td>5</td>
<td>Moderate variation in size of pale polychromatophiles.</td>
</tr>
<tr>
<td>16</td>
<td>9</td>
<td>&quot;</td>
<td>60</td>
<td>4.068.000</td>
<td>14.500</td>
<td>34</td>
<td>3</td>
<td>56</td>
<td>Y</td>
<td>Normal.</td>
</tr>
<tr>
<td>17</td>
<td>15</td>
<td>&quot;</td>
<td>64</td>
<td>4.936.000</td>
<td>11.900</td>
<td>39</td>
<td>6</td>
<td>48</td>
<td>Y</td>
<td>Same as to 10.</td>
</tr>
<tr>
<td>18</td>
<td>5</td>
<td>Spleen considerably enlarged</td>
<td>57</td>
<td>4.519.000</td>
<td>15.900</td>
<td>44</td>
<td>4</td>
<td>47</td>
<td>5</td>
<td>Slight variation in size of &quot;pear&quot; shape. Rarely a normoblast.</td>
</tr>
<tr>
<td>19</td>
<td>20</td>
<td>&quot;</td>
<td>68</td>
<td>5.158.000</td>
<td>13.200</td>
<td>37</td>
<td>5</td>
<td>40</td>
<td>4</td>
<td>Considerable variation in size of pale polychromatophiles. occasional normoblast.</td>
</tr>
<tr>
<td>20</td>
<td>17</td>
<td>Spleen very large</td>
<td>60</td>
<td>3.556.000</td>
<td>12.400</td>
<td>28</td>
<td>10</td>
<td>5.5</td>
<td>Y</td>
<td>Marked variation in size of &quot;pear&quot;, moderate polychromatophiles. Numerous nucleated forms.</td>
</tr>
</tbody>
</table>
An analysis of these cases shows that the number of red corpuscles was in all cases normal or but slightly diminished; and always proportionately that the percentage of haemoglobin was always diminished, always proportionately more so than that of the red corpuscles; that there was a leucoctosis in a little more than half the cases; that this leucoctosis occurred more frequently in those cases with splenic tumour than in those without; that the amount of the leucoctosis was independent 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 and absolutely decreased proportion of haemoglobin agrees with the results obtained by Felsenthal. It must be remembered that condition, however, as has already been noted,
is not an uncommon one in the anaemias of childhood.

The results as regards the white corpuscles do not agree with those of Fesselthal, who found a leucocytosis in every case, but rather corroborate Monti's conclusions that leucocytosis may or may not be present and 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 Gandobin 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 .... 4-5 per cent
- Large mononuclear .... 5-6 ....
- Polynuclear neutrophiles .... 4.5 ....
- Eosinophiles .... 4-5 ....

That is almost the same as in the cases without leucocytosis. These results do not agree with those of Weiss & Selenthal, who found the increase in the mononuclear 7 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 kinds of leucocytes.

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

The association of nucleated forms 7 of variations in the size 7 shape of the red corpuscles with an almost un-diminished 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 or 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 or nearly normal, and the percentage of haemoglobin both absolutely and relatively diminished. The anaemia may or may not be accompanied by leucoctysis. Leucoctysis 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, or 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. ii, Dr. Barlow & Bury state that the anaemia is of the chlorotic variety, the red corpuscles are diminished, and there is no marked increase of leucocytes.

Dr. Challenge in Allbutt vol iii. 1894, 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, it is often out of all proportion to other changes; the red corpuscles are diminished in number, the shape and size irregular, the haemoglobin 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" 2nd Ed. 1894, p. 630, remarks that the condition of the blood in pachydermatous children has received but little attention. Chemically it has practically received none. He has made numerous microscopic observations of the blood of pachydermatous children, and 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.
<table>
<thead>
<tr>
<th>Name</th>
<th>Age</th>
<th>Case</th>
<th>Total</th>
<th>Red</th>
<th>White</th>
<th>Appearance of Corpules</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annie Vincent</td>
<td>1 yr</td>
<td>Rickets, with enlarged spleen, liver very anaemic in teeth. Under Dr. Barlow.</td>
<td>103</td>
<td>3/5</td>
<td>169</td>
<td>Blood looked watery</td>
</tr>
<tr>
<td>Boy</td>
<td>10 months</td>
<td>Rickets, large spleen, pallid, cachectic, almost chlorotic look. Under Dr. Barlow.</td>
<td>70</td>
<td>2/5</td>
<td>174</td>
<td>Ditto ditto</td>
</tr>
<tr>
<td>Samuel How</td>
<td>1 yr</td>
<td>Enlarged spleen from rickets. Glands also somewhat enlarged. Under Dr. Gee.</td>
<td>102</td>
<td>2/5</td>
<td>25.6</td>
<td>Blood looked watery. Child was troublesome. Neither of these estimations quite satisfactory.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Another examination at a week's interval</td>
<td>149</td>
<td></td>
<td></td>
<td>Ditto ditto</td>
</tr>
<tr>
<td>Fred Bryant</td>
<td>16 months</td>
<td>Rickets. Spleen greatly enlarged - as low as crest of ilium &amp; to within an inch of umbilicus</td>
<td>89</td>
<td>3/5</td>
<td>145</td>
<td>Blood watery, corpuscles normal</td>
</tr>
</tbody>
</table>

Averages: 103
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. Marchand's experiments (quoted by Jenner, Lect. iii. & Heating 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. Ludger in 1883 proved that lime was not in excess in the urine of rickets. Dr. Goodhart in "Diseases of Children, 1894, p. 386) 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 Klein & Seemann. Dr. Cheadle (Vide Albright, Vol. iii, 1897) remarks that it would appear that there is really no important difference between the urine of rickets & 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's ill.) states that so far as he knows, no microscopical record of progressive foetal rickets exists, there seems to be no doubt, from the observations of Guérin, Ripier, Tauro, and 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 and malnutrition of the mother might well cause rickets in utero. Indeed the observations of those above mentioned as well as Harrowity and Schimuy seem conclusive as to its occasional occurrence in varying degree.

Mr. Tully ("Hindu deformsities" p. 253) says as to the existence of congenital rickets, Virchow is agreed, 9 cases have been brought forward by Shattock (Path. Soc., Trans., 1881). Herrick and others.


Steiner mentions the existence of a
specimen of rickets focus 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, and there is incipient leading 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 and 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 alteration of structure. These cases 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 and wasted as when rickets concurs with general starvation; it will probably have the depressed nose and 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, a cranio-talas, first described by Elsäßer in 1843, has till lately always been held to be a sign of rickets. (Vide Goodhart p 634). Mr. Parrott & others have called this doctrine in question, I 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. Dr. Barlow & Dr. Lees (Path. Soc. Trans. vol xxxii. p. 323 et seq.) collected 100 cases of cranio-talas; 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 fibro-sis of liver, spleen & syphilitic glands (theadle).
Scurvy rickets, so called acute rickets. Rickets as a rule progresses slowly and 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 supervision of such acute symptoms as extreme tenderness of limbs, periosteal swellings, sponginess of gums, haemorrhages. The periosteal swellings are due to subperiosteal haemorrhages, & the condition has been shown by Dr. Barlow & Chedale to be in reality one of scurvy, often, no doubt, superadded to rickets, but in its nature distinct.

Dr. Barlow, in the "Medico-Therapeutical Transactions", Vol. xxvi, p. 182, 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 femur, tibia, scapula, ribs, cranium--with a tendency to fracture, & sometimes to separation of the shaft from the epiphysis, as occurs in syphilis, acute marasmus, & 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. Sulley in "Practical Deficiencies," p.234, says, with reference to scurvy rickets, the factors in the causation of this disease,
are anaemia and an intensification of these errors of feeding which produce rickets. The symptoms are swelling of the limbs, due to sub-periosteal or intermuscular extravasation of blood, purpuric spots on the skin, spongy gums, haemorrhage from the kidney, 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 characterized 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. Bristow in "The Theory & Practice of Medicine" 1st Ed. p.934 states that acute rickets seems to affect both sexes equally, to occur mainly between the ages of six & eighteen months. The periosteal haemorrhages affect the lower limbs more frequently than the upper, are occasionally met with in connection with the ribs, scapulae, corne of the skull. The swelling, in 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 connection in connection with the vena.

E. Mayer (Deutscher med. Koch. Vereins Beil. 4) stated that recently cases of acute rickets (Scarty rickets) a Barlow's disease have been observed frequently in Berlin. He related four cases which he had himself observed during 1895.

In first symptoms were observed in three infants in the six months, 9 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 Mayer'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 Reutner 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, 9 had seen Barlow's disease in a few cases only. Keith's albumose milk was superintended
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 scurvy symptoms. Potassium was replaced by sodium bicarbonate, the quantity of the latter added was so small that the infant took only 1/2 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 and insisted on the importance of early diagnosis. He thought it important not to use sterilised foods for too long a time, but 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, and that the occurrence of bone lesions ought not to be awaited.

Dr. Ashley & Wright, (Diseases of Children, 7th Ed. p. 326) thus sum up the identity of the disease; Dr. Cheadle & Harlow 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, and in their opinion there is much to favour this view.

Achondrodystrophy foetalis.

Achondroplasia, foetal cretinism.

Dr. Cheade (Vide Allbutt id. 1894) mentions other cases, again, of so-called "foetal rickets", characterized chiefly by extreme abnormal shortness of the long bones of the limbs, have likewise been found to lack the special features of Akatau bone change. The area of proliferating
cartilage is absent; where 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 rickets 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 fontal cretinism. Dr. Goodhart (Vide Diseases of Children, 3rd 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 Dr. 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 (Ibid., Rep. Med. Coll. Phys. 1892 Vol. IV, S. 235). The bones developed from membrane.
Those developed from cartilage late in intra-uterine life are normal; but those which are ossified from cartilage before birth are dwarved. 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 craniotales—the bones at the base of cartilaginous development are dwarved & 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. {Ostomalacia} There is in rickets a deposition of new bone, which is deficient in lime; when the process is extensive, 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.
Vehm, 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. Jackson 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 osteomalacia, for the specimen
of the bones from this case, & the appear-
ances closely resembled those of the
osteomalacia of adults, whilst those
of rickets were absent.
Dr. Darlow describes a case, in which other
marks of rickets such as rib-ends, 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 2nd
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 remark-
able growth of bone, in a girl of fifteen
months old. In the skull, the new growth
a consequent thickening was enormous;
a pile-like new bone gradually monopo-
lised the diploic space; in the extremit-
ies, fusiform nodules 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 generalized bone-softening, the enormous development of imperfect, 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 ("Dr. Atkinson, 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 of late rickets in 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,' 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 and tenderness of the knees, but no rise of temperature. The case was clearly not one of good rickets. Anti-rachitic diet, cod liver oil, & steel wine were given, but the patient grew worse instead of better. Soda of potash was then tried, a 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.xxii
p. 391),

Mr Robert Jones, at the Liverpool Medical
Institution (Vide Brit. Med. Jour, Feb. 8, 1896,
p. 341) exhibited a girl aged 16 with recur-
descent 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 diabetic 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. H. Streetley (Vide. Illust. Med. News. Sept. 1888, 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, a tumour. From the slow progress of the disease, and the sudden development of a superadded scoliosis. Mr. Streetley diagnosed Aegisis 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. Gauthrey 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 appointed to investigate the case, viz. Dr. Hilton Yagge, Mr. Harrington Harvard, and Dr. Drewett, came to the same conclusion, that the changes were identical with rickets. The first
ends in this case were enlarged, the bones were much distended, the child was quite helpless. He died two years later, and a post-mortal examination was made by Dr. Abercrombie, and Dr. Barlow, who found the epiphysial lines of the bones thickened and 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, and delicate, from an early age the ankles grew outwards and the knees inwards. The humerus fractured, and subsequently the femur. In this, at the age of ten was admitted in Guy's Hospital. It was then found that the long bones were very tender and flexible, and this outer shell could be pressed inwards like the skull in craniotubes.

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 phosphiatic calculi.

Often 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 microscopic 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 XIX) 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. Mammary 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.
Clinton refers also to a case under Mr. Pilb, 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 previous to coming under treatment. No history of rickets in infancy; she walked at the age of 12 months, and 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, and absence of sexual development.

Ramsay (Brit. Med. Jour, 1887, 7, 1213) records the case of a girl, aged 14, in whom the epiphyses were enlarged & genu valgum ensued.

Palin (Practitioner, vol. xxiv, 1890, pp. 275-210) refers to a girl, aged 6, who came under the care of Mr. King at Peking 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. Barratt Bury (Healing's Encyclopedia of diseases of children, vol ii, 1883) refers to two cases in which there were very active manifestations at the age of 11 & 9 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 4, 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 and more grave up to the end of the fifth year. (Lucas in Lancet 9 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. Bautley in June, 1893, 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 &
Chicken-poxt at the age of 4; an elder sister states that the disease first commenced at the age of 4, that the child had 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; prognathous; 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 laterally below the level of the nipples; marked leading, especially of the fourth to seventh costo-chondral joints; prominence of the angles of the ribs behind; Carinosis Suluus not present; no heart or lung disease detected. Abdomen large & doughy, flabby muscular, 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 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 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 "buckling"; 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 limb; feet were affected.

General condition—A small, stunted child with flabby, ill-nourished muscles, a 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 and 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 and walk. The enlargement of the epiphyses has very much diminished so that the lower ends of the forearms is hardly noticeable. The claudication of the ribs has practically disappeared. The legs are a little less bent, abdomen very large and prominent.

Lansley has called this a case of recrudescence 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 and symptoms of recrudescence consist in the marked enlargement of the epiphyses, an enlargement which has practically disappeared while under treatment; a corresponding increase and diminution in size of the heads on the ribs; the irreversibly sebile temperature, the muscular flaccidity and 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. Ainsley 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 and 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 and 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, nervous membrane, nervous system; these conditions are concurrent and constant, although they are less obvious and obstructive than in the affection of the skeleton. The production of a rickety condition of bone by the application of tourniquet bandage, as in the experiments of Kassowitzt (quoted by Dr. Goadby in Allbutt Vol. iii, p. 477; Dr. Barlow in Heating, Vol. ii)
does not produce the changes in the tissues. The bones are certainly soft so they give way under pressure; but the ligaments are also soft and lax, so give way under pressure likewise; the muscles are atrophied and enfeebled, the mucous membranes catarhal; the reflex nervous system hypersensitive unstable.

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

The explanation (according to Dr. Chedl in Allbutt Vol. III. 1897) of the earlier and 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 or 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 of malarial 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, even an attempt, nevertheless may be made to classify roughly the various factors at work in the causation of rickets.
Microbic origin of Rickets.

Hagenbach—Burdach, loc. cit. (Berl. Klin. Woch., May 27, 1895) discusses the etiology with special reference to rickets being an infective 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 cities, its infrequency in pure, atmospheric. 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 infective processes are frequently localised in the bones. There is nothing in the clinical picture of rickets against the view of its being an infective disease. Acute rickets is known. The spleen is frequently enlarged. The objections to the view are that no micro-organism has been found, and that similar changes in bone may be produced experimentally 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 scurvy rickets has been shown to be identical with the ordinary disease. He would look upon defective feeding, vitiated atmospheres, acute or chronic infective diseases, as predisposing causes only.

In the "Brit. Med. Jour., Aug. 24, 1895, 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 nervous system. 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, polyarthritis & bone pains, hydrocephalus, marasmus, & paresis of lower extremities.

Pyogenic organisms have been found in the bones & central nervous system of ricketsy 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 analogous to that of rickets, & marasmus.

In the "Brit. Med. Jour. April 23, 1898. Ppt. p.68" Mercoli (Gazz. degli Osped. e delle Clin. Jan. 30, 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 9 the mammary ducts of nursing women, 9 are swallowed. As long as the alimentary canal is healthy nothing happens; but if this is out of order from cataract, dentition, etc., if in any way these organisms find their way into the blood 9 are localized 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 9 the epiphyseal 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. See the "International Medical Magazine 1897, pp.369-370" Researches on the Pathogenesis of Rickets (Revue mensuelle des maladies de l'enfance, March 1897).

By Sannio 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 reported about found from cultures made from the pulp of rachitic bones Staphylococci
Streptococci pyogenes, concluded therefore that rachitic was an infectious staphylo-strepto-cocci osteitis.

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

**Technique**

He cauterized a large surface of the anterior extremity of a rib and penetrated the interior of the cartilaginous tissue with a sterilized pipette aspirated. He made cultures on gelatine plates and large agar tubes. In most cases by this procedure, he cultivated the marrow of the diaphysis of the same bone. He endeavored always to have the same quantity of substance. He selected ribs and clavicles because they were more readily obtained. In rachitic cases the chrose 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 six-
teen of these the inoculations remained sterile, six cases gave pure
cultures of streptococci, two gave the Bact-
erium coli communis, one case gave
few colonies of Staphylococcus pyogenes
albus. In the five cases showing strepto-
cocci, 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 case the colonies were few.
The author believed that a strepto-coecemia,
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 and 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 communis or 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.' 1898. Chamhier (Centralb. f. Kinderheilk., 1897, ii, 358), from his studies on the nature of rickets, is of the opinion that rickets is an infectious disease, which may be contagious. His opinion is based on the frequent presence of rickets in institutions for children, and 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 knew 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:

1. Rickets is a specific disease, caused by unknown microbes.
2. It is contagious, endemic in cities, and occasionally epidemic.
3. It is found in an epidemic form in young pigs, its germs are preserved in houses. (Pediatrics, June, 1878).

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, and warmth. According to Dr. Macleod (Vide Alburti, Vol. iii. 47), Dr. Briton, p. 924, Dr. Barlow, Dr. Burz in
Heating 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 & North 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 W. Murrays (Vide, Australasian Mag. Gaz., 1891. x. pp. 185-90) to be prevalent in the large towns, 9 cases have been met with even in the bush. The disease appears to be affected by season; various Continental authors (Zeitschrift Archiv. f. Kindeinheit, 1888, p. 293.) have studied this factor, & their results show an increased prevalence in the earlier summer months, a minimum in December. Harowitz (Wiener med. Woch., XXXIX, 1889.) ascribes this to the
confine it to the house during earliest months of life of children born in winter-time. Mrs. Middlet 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. Dr. Barlow & Bury (Feeding, 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 Haristingu says percentage is 80 in children under...
Dr. Garrod's paper in "Brit. Med. Jour. Sept. 21, 1895, p. 708," remarks that it is extremely difficult to obtain a satisfactory knowledge of the influence of locality. Such statistics as are forthcoming do not always bear out the general statements 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.

Vissling (Archiv.f. Kinderheilkunde, 13, 1885, 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, &c. 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 practicing in those countries.
we are led to believe that, in the rural districts and small towns at any rate, the disease is comparatively infrequent, and 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 zone than in others. The influence of density of population and the stress of life is certainly very great, and is well seen in the contrast, in this respect, between our large cities with their lower class populations and 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. Harradine Fletcher (Lancet, Med. Jour., Sept. 21, 1895, p. 707) that Jews and 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 and 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, 3rd Ed. pp. 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 of these were 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. Oreade 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 rickety; in the vast majority
of cases, indeed, rickety children are born of parents who do not exhibit the smallest trace of past rickets.

Dr. Fox, in "Diseases of Children & Ed. p 158-9, 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, or 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 & Ed. p 647 quotes the observations of Kito von Ritterhain which show that rickety children frequent ly come of mothers who still bear traces of having suffered from a similar disease.
Dr. Garrod & Hetcher (Vide Brit. Med. Jour. Sept. 21, 1893, 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 Hitter von Hitterhain, Reiffer, 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 heredity 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.

Jay in "Rheumatic Deformities" 1896, p 223, says heredity plays no part in the production of rickets.

Jenner "Lecture in" says there are no facts to prove that rickets is hereditary.

Dr. Bristowe (Vide Theory & Practice of Medicine, 4th 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 this 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 smudges, the linear scars, the pegged teeth, the heratitis is 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; 2, conversely, many children who suffers from congenital syphilis are not rickets. (Chadle Vide Allbutt loc iii. 97, I also quoted by Drs. Carroll & Fletcher in Brit. med. Jour. Sept 27, 1893, 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.

Baginhev found evidences of syphilis in 6 per cent of all rickets children. Dr. Eustace Smith (Vide Diseases of Children, 2 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 subjects, & 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 rickets; & 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 and insanitary conditions must come into operation before the disease can occur. Dr. Garrard & 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 Parrott's is untenable, although prepared to rank syphilis among the predisposing causes, as a disease tending the nutrition both of mother & child.

Mr. Sully 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 and wasted, 0 it presents some of the distinctive signs of syphilis; 0 to these cases especially belong the boss-like pro-
fications of the frontal & occipital bones in their most extreme form, 0 the craniotubes, first described by 
Elsässer in 1843, although they are not absolutely limited to the syphilitic 
variety. Dr. Thomas Barlow & Dr. Lees 
(Pat. Soc. Trans., Vol. xxxii, p. 323) collect-
ed 100 cases of craniotubes, 0 from them 
they concluded that forty-seven per 
cent of the total are almost certainly 
syphilitic; 0 to this may be added 
the observation of Dr. Barter (Pat. 
Soc. Trans., Vol. xxxii, p. 361) that of the 
twenty-three per cent, of craniotubes in 
rachitic children, seventy-five per 
cent, were syphilitic. 
Possibly the enlargement of lymph-
atic 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 and amongst the poorer classes there, and 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 and sunshine, afford evidence of this. The want of sunlight and warmth appears to tell especially upon children of southern race when reared in cold and 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 xxv. pp. 316-320.) to suffer from rickets to such an extent that even those brought up at the breast do not escape.

Dr. Garro de Hotchler (Vide Med. Jour, Sept. 25, 1895. p. 708) state that children brought up in sunless, airless, and 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, and the production of rickets by degrading nutrition. Although, however, these influences of defective hygiene are frequently concerned in the production of rickets, Dr. Merckel states, they are not constantly present, and 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, and cleanliness and warmth are concerned; a child may enjoy these in perfection and yet become rickety; or again a child may not become rickety although brought up under the most unhealthy external conditions.
The Tubercular Diathesis - In lecture iii, p. 14, Dr. 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, 1859, 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 crisis chief 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 and very quickly bring his life to a close. Fully in his book on "Athanitic Deformities. p 253, states that the tubercular diathesis may be considered a factor in so far as it is a cause of general weakness, and predisposes to digestive disturbances and diarrhoea. Ritter von Ritterhain, Senator (Vide Liebscher'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 & Hatcher (Vide Brit. Med. Jour, Sept 27, 1893, p 708) lead them to doubt the frequency of their association. Dr. Goodhart (Vide Diseases of Children, 5 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 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, 1898, p. 707. state that in confronting such a problem as that of the maternal element 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 William Jenner (Lect. iii. p. 34. & Eustace Smith, Diseases of Children, p. 137; Drain'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 bowing of the ribs, may be detected if looked for at about the completion of the first six months of life, and the experiences of Dr. Garnod & Fletcher agree with that of Lusthing (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:

1st. 7. 8. 9. 10. 11. 12. 13. 14. 15. 16. 17. 18. Total 141
3. 4. 5. 6. 11 36. 36. 19. 13. 2. 6. 5. 3

Sixty-eight were boys, seventy-three girls.

Dr. Gee "On Rickets" St. Bartholomew's Hospital Reports, vol. iv. p. 89, 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, 92 in the fourth year.
Breifner (Jahrbuch S. 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 newborn 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 and osteomalacia & scurvy.

Schwarz (Medizinische Jahrbuch, viii, 1884) 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. Carrod & Hetcher.

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. In the book "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 lactation 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. Dr. Garrod & Fletcher (vide Brit. Med. Jour, Sept 27, 1893) have met with numerous examples of rickets even 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, and delayed dentition. The infant had been fed on good cow's milk and 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, and 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 and water, and no farinaceous food 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 and a half months before the patient, was 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, and 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, and at the seventh month of pregnancy was confined to bed for four weeks, and 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, and their children have developed rickets apart from any recognized dietary cause.
Dr. Garrett & 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 ricketty, 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, & even 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, a previous & subsequent children have been quite free from ricketsy manifestations.

The numerous & rapid pregnancies —

Dr. Jenner (Sect. 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 ricketsy. The explanation of this fact is that among the poor the parents are generally worse fed, worse clothed, worse lodged, the larger the number of their children — the mean wages remain stationary — 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.

Dr. Garrod & Hatcher (Proc Brit. Med. Jour. Sept. 21, 1895, p. 705.) 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, I had been given no Saracenous food. It was the fourteenth child.

The mother had been married twenty-five years, and the previous child was twenty months old at the time of the patient's 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 and after pregnancy, had been married fifteen years. Her milk was abundant. I had joined the sole diet of the child. Hygienic conditions were good.

2. Multiple Pregnancy.

The following case illustrates the influence of twin pregnancies, & it may be supposed that just as during lactation when 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.

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 confine-
ments, 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. So 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,
I had had good ever since. It was a
second child after an interval of eighteen
years without any intermediate concep-
tions. 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
fetus, 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 and often overlooked factor in the causation of rickets. A male child, aged 3 months, showed distinct beading of ribs. The mother, aged 22, had been married four years and had two children. The first child was being suckled throughout the first five months of the second pregnancy. The labour and puerperal period were natural. The child was entirely breast-fed, and the supply of milk was abundant. A second patient, a male, aged 4 months, with definitely beaded ribs, was a ninth child, and 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 cows' milk and 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 mother in favouring the development of rickets does not cease with the birth of the child, but lasts throughout the period of lactation.

Dr. Bradle in his book on "Artificial Feeding of Infants and 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 when 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 rickety during the time of suckling. Even with congenital syphilis at birth, the suckling does not become rickety.

Dr. Barlow & Dr. Lees (Vath. Path. Trans. vol. xxxii. p. 330.) found that in eleven children with craniotubes who were brought up entirely at the breast, not one showed the smallest signs of rickets.
Dr Barrod & Fletcher (dict Brit. Med. Jour, Sept. 21, 1893, 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 these elements which are necessary for the proper nutrition of the child. Gumpolowitz (Prager med, Wochenschrift, xiv, p 50, 1889) at Prague, found signs of rickets in 28 per cent, of breast-fed children attending the 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 to 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:
   1. Abnormalities of mother, for example,
      (a) Retracted nipples; single available breast,
      owing to abscess, new growth, etc.,
   2. Abnormalities of child, for example,
      hare lip, cleft palate, etc.
   (c) Sharing of supply by twins.
   (d) Deficient activity of gland.

II. Deficient quality of milk supply, due to:
   1. Mother's health during lactation.
   2. Menstruation during lactation.
   3. 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
rachitis can only be ascribed to the diffi-
culty experienced in obtaining the
natural supply. In the case of
Twins are 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.

11. Mother's Health during Lactation:
The influence of the mother's health upon the constitution and quantity of the milk is well recognised; concurrent illness, overwork or worry and 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, and after that had good cow's milk, a pint and half per diem. During lactation the mother had an attack of either gout or rheumatism, lasting for two months, and 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 uncommon occurrence during lactation, at any rate among the lower orders. Schlichter (Wiener klin. Wochenchrift, ii, 1889, 51952; iii, 1890, 475. Summary, Schmidt's Jahrbücher, vol. 226, p. 53) found that among 424 suckling women at a lying-in hospital no fewer than 50 menstruated during the first two months and a half.

Jilbury Fox (Trans, Obstet, Soc., London, 1863, iv, p. 260.) considered this one of the most important of all the causes of rickets, and 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, and 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, Bequerel & 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 32 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 Dr. Friedel & others, however, tends to support the view
that this has an important influence in the causation of rickets, I 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. In the months it had had fresh cows' 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, and 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. Her 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, and was afterwards given fresh cow's milk in sufficient quantity, and later other suitable articles of diet.

Over-lactation,—Dr. Eustace Smith in "Diseases in Children" 2nd Ed, 1887, p. 137, says the long continued suckling may induce rickets, for the breast milk after a time ceases to satisfy the infant's wants, and too little additional nourishment is supplied. Dr. Goodhart ("Diseases of Children," 2nd Ed p. 443) believes that prolonged suckling causes rickets. Dr. Garrod & Fletcher (Brit. Med. Jour., Sept. 21, 1896, 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 a few disturbing factors as possible. Syphilis was excluded in every case which has been quoted, and 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 hygiene causes, for example: A mother affected with exophthalmic goitre had borne seven children within 12 years, all were suckled for about 16 months, 9 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 Gracie's disease, dating from twelve months previous to the birth of the child.
Anatomical predisposing causes of rickets: — Sulky (Virtue Rickets and Deformities, 1864, p. 253) states that Benecke has studied the predisposing causes of rickets from an anatomical standpoint. 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 Benecke's observations. The large size of the arteries explains the existence of hyperaemic condition of the bones, especially at the epiphyseal junction, of the increase of development and 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 the bone and
muscle, & the latter becomes flabby 
& jelly-like in consequence.
Regarded in these lights, it would seem that Beneke's observations are very important in elucidating the predisposing causes of rickets.

Disorders of Digestion:

Dr. Lucadle (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; I 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 or excessive, or when it coincides with particular faults of diet. It probably acts by removal of certain special elements which are the least quickly or rapidly digested or absorbed. Digestive disturbance is not, therefore, an invariable essential factor in the production of rickets.

Dr. R. Etze 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 commonly present in rickets is 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 protein food. The blood is
rendered poor in proteid bodies, the glands suffer thereby, and 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, and lymphatic glands.

Faults of Diet.—Errors of diet play so conspicuous and obvious a part in this connection that they concentrated upon themselves most of the attention that has been given to this subject. Dr. Garrod and 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 and potent cause, but
Sometimes it is the only cause.

Rickets is produced as certainly by rachitic diet as is scurvy by scorbutic diet.

And it is seen in these by no means uncommon cases where children can healthy or healthy, well-to-do parents, brought up under perfect hygienic conditions so far as air, light, cleanliness, and warmth are concerned, yet become rickety when brought up on artificial food. The only fault we can discover is the dietetic fault; and such cases are cured by a correction of the diet, without any other change of hygienic conditions; they are cured in fact, by an antirachitic diet as certainly as scurvy is cured by antiscurvy diet.

The effect of diet was shown in the most striking manner by Dr. 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; on the contrary, it may be over-fed, fat and gross, yet extremely rickety. Dr. Carroll & Nettleship in "Brit. Med. Jour., Sept. 21, 1893", 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 and 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 fetus 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 and 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, confection, 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" 3rd, 1894.
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
Mémoires sur les Differenôts duc systême osseux, Paris, 1859; also quoted in the "Cyclopedia of the diseases of children" (Heating, vol ii by Dr. Darlow, Bury) who kept puppies on a meat diet for 3 or 6 months, which showed all the signs of rachets, although imprisoned by the later experiments of Rapier & (Mem, et compl., rend., soc. de sci., med., de Lyon, 1863, iii. pp. 199-208; Arch., de Physiol., norm., et path., Par, 1874, 2. X. pp. 108-125. also Heating, loc. cit.) on cats, dogs, & chickens & failed to produce rickets, has been confirmed by experience at the Zoological Gardens. In many years the lion whelpes have been weaned early, & put upon a diet of raw flesh only; they have invariably become rachytic 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, coarseness of sinew & bending of bones; & the identity of the metiotic 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 could take flesh meat. Four weeks after being weaned, it was noticed to be very quiet, constantly lying down; it 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 not flabby. After a time, it began to take milk, now it seems quite strong, healthy, but dwarfed. 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 hinders 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. So, Jenner, in lect iii, says this view is altogether erroneous. It 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.
Crossart & Milne Edwards (quoted by Cheadle & also by Dr. Jackson & Bury, Healing 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 (Saurb. f. Kindeheilkunde, 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. Sitztber, d. Gesellbuch f. Morphol. u. Physiol, in München, 1889-1890, v. 101. & Baginsky, Lehrbuch d. Kindekrankh. (Frieden's Sammlung). B. vi, p. 270 et seq. (1883), claim to have established the existence of true rickety change by such treatment. Dr. Barlow & Bury (Vide Healing Vol ii p 246) state that other considerations show the untenability of such hypothesis.

Thus (1) slight but characteristic rickety changes, such as increased vascularization, proliferation of cartilage, etc,
are met with when the new-formed bone, is quite normally calcified.

(2) Krukenberg 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 minor degree of rickets the calcification of cartilage covers a much greater area than normal.

(4) Cow milk is much richer in lime salts than human milk; yet is more prolific in rickets; or, whatever the food, it would be impossible sufficiently to decrease the supply of lime to produce the great poverty found in bones severely 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. Neale (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 rickety 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 rickety, 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 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.

Hitzmann, Sitzberger, d. Hais, &c. Acad. d. Wiss. in Wien, 1873, No. 17.) I also quoted by Jenner, Smith, Godber, &c. put forward another view of its action. It irritates the osseous 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, Gorus) but Newbauer failed to find it in the urine of a case of extreme rickety. & Luelzer, in 1883, proved that neither lime nor phosphoric acid is present in excess in the urine of rickets. Hitzmann claims by its administration
to have produced the condition directly. This formation of lactic acid, however, has not been confirmed; and the hypothesis is rendered improbable by the fact that rickets arises in children in whom there is no apparent disorder of digestion to cause lactic acid fermentation, who digest the starch or maltose thoroughly, and even wax unduly fat upon it. Moreover, according to repeated observations of my own, writes Dr. Chadde, the rickety state disappears, 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 neutralized 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 of preparations of milk. (See "Artificial Feeding of Infants" 4th Edition, 1896, Mudge)

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<thead>
<tr>
<th>Elements</th>
<th>Human</th>
<th>Cows' Milk</th>
<th>Cows' Milk</th>
<th>Cows' Milk</th>
<th>Akens' Goals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Luf)</td>
<td>Fresh</td>
<td>Condensed</td>
<td>Pure</td>
<td>Pure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Average from analyses by Payen, Goupy-Sarang, König, Aitch, Whitelegg)</td>
<td>(Former Holder)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrogenous elements or proteins</td>
<td>2.35</td>
<td>4.374</td>
<td>2.187</td>
<td>2.916</td>
<td>2.37</td>
</tr>
<tr>
<td>(Casein albinum)</td>
<td></td>
<td>1.458</td>
<td>2.187</td>
<td>2.916</td>
<td>2.37</td>
</tr>
<tr>
<td>Hydrocarbons or fats</td>
<td>2.41</td>
<td>3.493</td>
<td>1.166</td>
<td>2.332</td>
<td>4.46</td>
</tr>
<tr>
<td>Carbo- &amp; Lactic hydrates (Lactose)</td>
<td>6.39</td>
<td>4.403</td>
<td>1.467</td>
<td>2.954</td>
<td>5.02</td>
</tr>
<tr>
<td>Lime</td>
<td>1.019</td>
<td>1.71</td>
<td>1.467</td>
<td>1.60</td>
<td>2.0</td>
</tr>
<tr>
<td>Phosphoric anhydride</td>
<td>1.026</td>
<td>1.234</td>
<td>1.468</td>
<td>4.03</td>
<td>0.5</td>
</tr>
<tr>
<td>Other constituents of ash</td>
<td>0.295</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water</td>
<td>88.51</td>
<td>87.053</td>
<td>93.512</td>
<td>91.530</td>
<td>87.38</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>


Table II

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

<table>
<thead>
<tr>
<th>Elements of Food</th>
<th>Standard of Human Milk</th>
<th>Bread Jelly</th>
<th>Malted Food partially denitrified</th>
<th>Malted Food more highly denitrified</th>
<th>Malted Food with denitrified milk</th>
<th>Prepared entire Wheat-hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrogenous Proteins</td>
<td>2.35</td>
<td>2.73</td>
<td>2.705</td>
<td>2.711</td>
<td>3.35</td>
<td>2.723</td>
</tr>
<tr>
<td>Hydrocarbons x fats</td>
<td>2.41</td>
<td>0.50</td>
<td>1.695</td>
<td>3.632</td>
<td>1.08</td>
<td>2.009</td>
</tr>
<tr>
<td>Carbohydrates {</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactic acid from milk</td>
<td>6.39</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grape Sugar</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Various Dehydrine Forms</td>
<td>13.24</td>
<td>5.618</td>
<td></td>
<td>3.953</td>
<td>6.211</td>
<td>87.12</td>
</tr>
<tr>
<td>Total Carbohydrates</td>
<td>16.62</td>
<td>6.211</td>
<td></td>
<td>77.28</td>
<td>6.211</td>
<td>87.12</td>
</tr>
<tr>
<td>Lime</td>
<td>1.01</td>
<td></td>
<td>0.08</td>
<td>1.01</td>
<td></td>
<td>0.10</td>
</tr>
<tr>
<td>Phosphoric Anhydride</td>
<td>0.26</td>
<td></td>
<td>0.11</td>
<td>1.43</td>
<td></td>
<td>1.16</td>
</tr>
<tr>
<td>Other ash</td>
<td>1.62</td>
<td></td>
<td>1.17</td>
<td>0.14</td>
<td></td>
<td>1.40</td>
</tr>
<tr>
<td>Water</td>
<td>8.01</td>
<td>8.01</td>
<td>89.643</td>
<td>44.54</td>
<td>4.37</td>
<td>85.619</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>
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, throw light upon this point, certain defects appear to be constant. An analysis of the foods on which rickets is most frequent ly & 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 ii & iii) show invariably deficiency in two of the chief elements so plentiful in the standard food of young animals, namely, animal fat & protein. 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 rickety—namely, biscuits, rice, & fruits—is markedly deficient in proteid, & fat is practically absent. The food of the lion cub, which became rickety on a diet restricted to raw flesh, was almost destitute of fat, & was poor almost also in earthy salts, although rich in proteid. The meat was that of old hoves almost destitute of fat, & once a thick 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.
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; they 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 & cure of rickets. That rickets frequently follow the prolonged food vomiting & diarrhoea provoked by cow's milk, is consistent with this estimate of the effect of deficiency of fat & proteid in its production. In as the fat must be emulsified or 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 caseine & salts, which are in solution & ready for immediate passage into the circulation. The result would be a great privation of fat & proteid.

Dr. Sheardle (vide Allbutt Vol iii. 1897.) 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 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 follow. The curative power of cod-liver oil is evidence in the same direction, and it is interesting to find in this connection, as recorded by M. Keno, 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, for rickets is found to be deficient in the bones of vivica.

Proteid, again, is essential to the vitality of protoplasm, for indeed to the activity of all vital processes, it 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 and earthy salts—one or both—would explain not only the faultiness of bone, but also the feebleness of muscle, the anaemia, the catarhal 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 and 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 rickets bone, is suggestive of a sub-acute or chronic inflammatory condition; this view is supported by the experiment of Rassowitz, 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 and cell proliferation observed in the liver and 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 ricketsy 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 peculiar 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 and cell proliferation are the results of the impetus of the formative process left unsatisfied in its normal direction in want of materials for the building of the permanent structure.
With regard to the microbic origin, Hakenbach believes it to be an infective process, and looks upon other causes as predisposing only. Harowitz under certain conditions is disposed to admit various microorganisms as the cause of rickets. Miroldi pleads for the microbic origin of rickets, believing that the disease is caused by the effect of streptococci and staphylococci. Ettore also holds this view, and concluded that rickets is an infectious staphylo-streptococcic osteitis. Chaumier is of opinion that it is of an infective 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 (Bilbury too 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 Britton, Barlow, Bury, Smith, Gee, Jenner, Garrod, Fletcher, Hanowitc, Ketter, Ritchie & others, to favour the development of the disease, to aggravate it in degree, but are not absolutely essential factors.
Disorders of digestion. He considers this to be the actual cause, others look upon it, as predisposing.

Faults of diet. Dr. Barlow & Fletcher state that this is the greatest factor of all. Dr. Headley 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 protein 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, qua rickets, invari-
ably 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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Old Trafford,
Manchester.