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T h e s i s

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R I C K E T S

By

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PROLOGUE.

The subject of rickets is one to which I have given much thought during several years of an intimate experience in the management of the disease. I have been particularly impressed by its great prevalence amongst the more crowded districts and lower classes of our city population as compared with the less populous, but not necessarily less fortunately circumstanced country localities. I have likewise observed marked differences in physique between the parents of the city and those of the country; also the apparent contempt of the citizen parent for the health-giving influences of fresh air. Even more so have I noticed the crass ignorance exhibited in the choice of proper articles of diet for the rearing of children of tender years.

Another striking factor is the appalling death-rate amongst young children from this disease and its protean consequences, - a death-rate which, in this so-called enlightened age, is not showing the diminution it should do: largely, in my opinion, owing to the invincible ignorance and obstinacy of the lower-class mother. She, in not a few instances, insists in bringing up her infant in the same faulty way as she herself was reared. In these cases the fussy grandmother is the bugbear of every family practitioner. "Children are not reared nowadays as they were in my time," she says. Granted; and it is well for the present-day pediatric prospects that usually they are not. The grandmother-empiric, when inclined to practice her domestic medicine with its nonsensical basis, is any everyday evil, and should be fought at every turn. There is too great a tendency on the part of some practitioners to agree with her for the sake of peace and popularity and recommendation: this fact, particularly in slum experience, I have from time to time observed. "Look at me", she commands the anxious mother, "I was reared on such and such a food or in this or that way, and what's good enough for your mother should be good enough for your bairn!" Authority has spoken and nothing more can be said.

SYNONOMY.

Various names have during the past been applied to rickets, the following being the best known:

Rachitis; Articuli Duplicati; Morbus Anglicus; Rachitismus; Rachitisme; Neuures; Zweiwuchs; Doppelte Gleider; Englische Krankheit.

The nomenclature of the affection is interesting and may therefore be briefly considered. The word "rickets" is derived from the old English word "wrikken", to wrest or twist awry - it being used in much the same sense as when we speak of a "rickety table" or a "rickety chair," - so that, by analogy it has come by common consent to be applied to a disease whose predominant characteristics are deformities and osseous anomalies.

The French word is synonymous with prison; the subjects of the affection being regarded as imprisoned by the ailment and bereft of liberty of movement. "Neuure des jointures" is another name for the malady, these afflicted with it being considered as tied up - "neuues". The older writers considered the term "rickets" as descriptive of the principal symptoms of the disease, with the additional advantage that it is easy to pronounce and not difficult to bear in mind.

The historians of the affection tell us that the word is a very old one, dating back to Anglo-Saxon times, when it meant a rick or hilleck; and that when Glisson studied the affection in the seventeenth century he called it "rachitis." "The most recent and ordinary name of the disease," he says (1650), "is 'The Rickets', but who baptised it and upon what occasion and for what reason, or whether by chance or advice it was so named, is very uncertain." He looked upon it as "absolutely a new Disease and never described by any of the Ancient or Modern Writers." According to him, "it first became known about thirty years since in the counties of Dorset and Somerset lying in the Western part of England." He suggested the term "rachitis" or "rachites" as synonyms for the reason that "they that are expert in the Greek ~~language~~ in tongues may peradventure expect a name from us whereof some reason may be given." He does not think that "rickets" is a corruption of the Greek term *πάξις*, the spine, but selected the word because the vertebral column was "the first and principal among the parts affected in this evil."

The Germans speak of the malady as "die Englische Krankheit", intending to indicate the source of the original description of the disease, and not its greater prevalence in Britain than elsewhere.

Finally, it may be mentioned that various distinct affections have been included in the past under the term rickets. Thus, Barlow's disease, or infantile scurvy, used to be known as acute rickets, scurvy rickets, or haemorrhagic rickets; and achondroplasia was described by the older writers as foetal rickets.

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DEFINITION.

Rickets is a chronic affection of children which commences insidiously, and is not infrequently accompanied by repeated attacks of gastrointestinal catarrh, which are seen followed by signs of malnutrition in all the tissues of the body. Included in the symptom-complex of the disease are such phenomena as instability of the nervous system and liability to convulsions, well-marked loss of muscular power, a tendency to catarrhal affections of the respiratory mucosa, and by certain striking osseous deformities which sooner or later end in spontaneous cure.

The nature of the affection is best understood when it is regarded as a general disease of all parts of the system. It is true that it is usually recognised by alterations in the bones, but in many cases it is not even the osseous system which shows the first departure from health. The general condition of the body, the muscles, the viscera, to a certain extent the blood also, is suffering, not equally it is true, not always simultaneously, not to the same degree in all cases; but there are very few which do not show alterations beyond those in the bones. This is why so many different theories have been advanced with a view of explaining the nature of the disease, and why for an explanation of all its protean manifestations investigations have been made in the fields of pathological physiology, chemistry, neurology, and even bacteriology.

H I S T O R Y.

The middle of the seventeenth century saw the dawn of our knowledge of rickets as an independent disease, when its prevalence attracted the notice of physicians, and led to its being thoroughly investigated by a committee, specially appointed for the purpose by the Royal College of Physicians of London and including Glisson and Bate and Regemerter, the result of whose investigations were published by Glisson in the year 1650. On the subject of rickets this seems to be the earliest known publication; but there can be no doubt that the affection had been in existence long before this time, but that it had not attracted attention. Stiebel says that, according to deformities depicted on Aesop's statue, he must have been rachitic. Hippocrates is said to refer to rickets in his graphic account of certain deformities (*De articulis*); and Lusitanus, Galen, and Celsus make mention of a similar disease. Physicians in Switzerland, France, Holland, and Germany, in the sixteenth century, according to Reusner, Fermius, and Schenck, treated numerous rachitic children.

The term "morbus anglicus" or "morbus Anglorum", has for a long time on the Continent been synonymous with rickets. In this country and America the popular name of the disease is rickets, derived from the Norman term "riquets", applied to persons suffering from kyphosis and other deformities. The Greek term, rachitis, proposed by Glisson and adopted in France (*rachitisme*), is a less happy one, for it suggests a rather rare localisation of the disease in the spine, vertebral gibbosity. It is very possible, however, that in Glisson's time Pett's disease of the spine was not always differentiated from rickets, for the diagnosis sometimes presents considerable difficulty. The word "chartres" (*carcer, castrum*) was formerly quite extensively applied in France, suggesting the idea of an interference with, or deprivation of, liberty caused by the disease. At the present time the laity in France speak of "nouures" (*knettings*) and of "enfants noués", being struck especially by the epiphyseal swelling. The terms "double joints", "doppelte Gleider", "articuli duplicati", etc., have been in various quarters applied to express the same symptom.

One important characteristic of the disease, namely, softening of the bones, was first described by Mayow, of Oxford, in 1660. In 1841, Petit emphasised the evil consequences of premature weaning. Duverney (*Maladies des Os*, 1751) described the pathological anatomy of the disease, and showed that the bones in rickets are softened, rarefied, lighter than normal, more tender, more fragile, more liable to suffer green-stick fracture. He showed that the bones were rough and uneven on their surface, that they were covered in places by a new layer, that they were riddled with holes, etc. In 1897 Pertal, led by certain morbid coincidences, divided the disease, which had hitherto been regarded as a morbid entity, into seven varieties, namely, syphilitic,

scrofulous, scorbutis, rheumatic, following intestinal lesions, and following the acute exanthematous fevers.

The nineteenth century saw the symptoms, course, etiology, pathology, etc., of rickets better understood than hitherto. Ruz (Gaz. Méd. de Paris, 1834) demonstrated that the spongoid tissue of Guérin (1837-39) consisted of a reddish, elastic, reticular material. He recognised three stages of the disease: First, a period of incubation or of effusion, the blood in this becoming distributed throughout the interstices of the bones. Second, a stage of deformity, characterised by the development of spongoid tissue in the epiphyses, diaphyses, and the subperiosteal space, and also a softening and bending of the affected bones. Third, a resorption, consolidation or ossification period, characterised by the conversion of the spongoid tissue into very hard bone. An extra and sometimes absent period is, according to him, one in which the bones remain fragile and rarefied; this is his stage of consumption. Amongst the numerous experiments he performed we may note that he made puppies rachitic by dieting.

The resemblance of rickets to osteomalacia was pointed out by Trousseau and Lasègue (Arch de Méd., 1849), and Baylard (Thèse de Paris, 1852); and the microscopical changes in rickety bone were first described by Broca (Sec. Anat. de Paris, 1852), he also showing that rickets, as regards its bone pathology, is due to a deviation, an arrest, a suspension of normal bone-formation. In 1853 Virchow taught that rickets is analogous to parenchymatous osteitis, and in this he was supported by both Kassewitz and Baginsky. In 1881 Parrot (Internat. Med. Congr. of Lond., 1881) claimed that, both as regards pathology and symptomatology, rickets is always an expression of syphilis. It is unnecessary to dwell on the fact that his theory was soon disproved.

More recent writers endeavoured to show that the disease is due to a previously existing anomaly of the alimentary canal, . . . gastrointestinal dyspepsia or gastric ectasia; and after that bacteriologists endeavoured to discover a specific germ.

In this disease numerous important changes occur in the BONES generally. Under conditions of health a bone grows in length and thickness. The former depends on the formation of new tissue in the so-called zone of proliferation between the epiphysis and diaphysis, the latter on the same process on the inside of the periosteum; absorption takes place in the interior with a tendency to widen the medullary canal. In the newly deposited tissue ossification takes place readily as long as there is no disease. In the rachitical bone the formation of some new tissue and the absorption of the old take place normally; it is ossification that is defective. Excessive absorption, which at one time was considered sufficient for an explanation of the rachitical process, is not so any longer. The anomalous process is mostly taken to be a parenchymatous inflammation; some, like Pemmer (Researches on Osteitis and Rickets, Leipzig, 1885) and Tedeschi, look upon it as a nutritive disorder lit up by neurotic influences originating outside the skeleton, the cartilage proliferation near the joints and the periosteal thickenings are but secondary consequences of pressure, muscular traction, and other external or traumatic factors.

The theory of a parenchymatous inflammation has been advanced by such well-known observers as Guérin, Virchow, Baginsky, and others. The two last-mentioned pathologists insisted upon the necessity of assuming the presence of disturbances in the circulating blood, whose effect would be mostly noticed wherever the physiological functions of blood and tissues were most active - i.e., where growth happened to be most intense. Such parts are, besides the foetal and infant brain, the bones, and mainly the zones of proliferation between the epiphyses and the shafts, and under the periosteum. It is here that intense hyperaemia will set in, with the result of rendering the tissues red, soft, and succulent, and of giving rise to irregular cell proliferation and deficient, - in the later periods of impeded circulation superabundant, - calcium deposit. The increased cell proliferation extends into and beyond the ossification line, which is straight and narrow in the normal and becomes diffuse and broad in the rachitical bone. At the same time the medullary spaces filled with medullary cells extend into the conglomerates of cartilage cells. In all of these, after a while, calcium is deposited in a short time and irregularly, and the newly-formed cells are not, as is normally required, mostly first transformed into cartilage before becoming bone, but the transformation of cartilage cells into bone corpuscles is a direct one. Normal bones show this so-called metaplastic ossification to a limited degree, but all the rickety osseous structures manifest it. Numerous observers have inquired into the causes of this parenchymatous inflammation, or rather into the

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sources of the circulating irritation. Wegner managed to produce it by feeding animals on minute doses of phosphorus. But bedside observation cannot be so positive as the experimental evidence. I shall later on show to what extent bad air, improper feed, infectious diseases, chemical changes in the blood, microorganisms, etc., may prove operative.

Rickety bones show both physical and chemical alterations. Friedleben found an increase of water and of fat, the latter mainly in cases in which the rarefaction of the tissue persisted. The proportion of organic and inorganic parts is reversed: while the latter are 65% in the normal, the former are 65% in the rickety bone. Still, in places there may be found a good deal of calcium in the rachitic bones, mainly in the epiphyses. It is rather an adaptation for normal ossification than the deposit which is at fault. The pathogenesis of the disease must take into consideration what it is that results in the diminution of phosphate of lime in rachitical bones; if its relative absence from the system due either to insufficient ingestion or insufficient absorption; if there is a local disorder in the growing bone which prevents deposit of calcium, or are both at fault; and if proliferation and dilatation of blood-vessels, which is so manifest about the affected parts, have anything to do with defective ossification, and if so are they primary or secondary in nature. It is unquestionably true that feeds are not at fault unless we have to do with downright cases of starvation, and these do not become rachitical. Woman's milk, and still more cow's milk, also farinacea, contain a great deal of calcium, the milk of mothers of rachitical children as much as those of others, says Seemann; and, according to Voit, the ingested lime exceeds always that which is deposited on or required by the bones. The experimental findings of Guérin, Chessat, and Reloff, who deprived animals of phosphate of lime in their feed, do not prove much, for what they accomplished was mere fragility of bones, but not rickets. Still, it has been suggested that this and forced abstinence may be able to create, though not rickets, a disposition to the latter. But as starving infants do not generally become rickety, this cannot be the rule, though I am willing to admit that in some cases the fact may obtain. I am also of the opinion that deficiency of absorption is not at fault. When infants, healthy or rickety, are overfed on phosphate of lime a single day, there is at once more elimination of it, both through the kidneys and the intestines. In the latter it is not merely an overflow, for more is found in the lower than in the upper part of the alimentary tract, exactly like iron, which behaves in the same way; that is, it is first absorbed, and then eliminated again. To digest milk very little hydrochloric acid is required. If the latter, or chloride of sodium be present in fair quantities, a goodly amount of the salt of the food is easily dissolved and absorbed in the upper part of the intestines, and just as quickly eliminated in the lower. The circulating blood does not carry overflow material any length of time. If it did, it would soon become decomposed, - indeed, would be destroyed sooner than this floating strange material could be expected to exert a lasting influence on solid tissues. It has been

claimed that the acid condition of the blood explained the absence of calcium from the tissues. It was principally Heitzmann, in 1873, and Baginsky, in 1879, who claimed lactic acid to be the solvent of lime in the bones, and thus to become the cause of rickets. Circulating acids would be immediately counteracted by circulating alkalies. Indeed, Siedamgretzki found less of salts, but no rickets, under such circumstances, and if tolerated at all, any quantity of lactic acid flowing in the blood sufficiently to wash out lime from the tissues would first destroy life. It was suggested that possibly from a cause hitherto unknown some nascent acid of local origin would ~~exercise a local~~ effect, but no proof has been forthcoming. It is not impossible that the many causes actually known to produce rickets act primarily on some organ with internal secretion, which may still be found the anatomical and physiological source of rickets. The relation of osteomalacia to the ovaries is rather suggestive in this direction. The syphilis theory of Parrot and the nerve theory of Pommmer are undoubtedly fallacious. The microbic theory of Mircoli and others is certainly far from being proved.

The general osseous changes of rickets consist of a softening with increased bone formation at certain parts. The latter condition is most marked at the ends of the long bones, and gives rise to characteristic deformities in the ribs, tibia, radius, etc.

There are striking naked-eye appearances to be noted in connection with rachitic bones. The bones are red, vascular, though not uniformly so; their peri-epiphyseal (ossification) cartilage is bluish and very copious and broad, with irregular and indefinite outlines; their consistency is altered; they are soft; the periosteum is thickened, red and oedematous, closely attached to the loosened bone, so that when pulled off it may tear pieces of bone with it; periosteal blood-vessels are seen to enter the bone directly; the epiphyses are thick, soft and compressible; the frontal and parietal bones are thickened, still more so the tibia. The new deposits, besides the periosteum, are quite thick, from one to ten millimetres and more, and so soft as not to resist the knife. The bones when cut bleed. In the parietal and occipital bones there are open spaces of a half to one and a half or more centimetres in diameter, sometimes in, sometimes adjoining, the soft deposits; transparent when held to the light. The medullary canals of the long bones are deformed, compressed in places, and narrow. The long bones are deformed in different ways - by deposits on their surface, by curvatures, by subperiosteal infarctions, and by fractures. After recovery the bones are short, hard, sclerotic, thick, and more or less curved. In a few cases in which the process of softening is very intense and fat is deposited in the dilated areoli, porosis takes the place of sclerosis. In later life, such an unsubstantial and light bone, contrary to the general condition of recovered rickety bone, is very apt to fracture from comparatively causes.

Normally, a section of one of these growing bones shows, when cut through at the epiphyseal portion, a couple of perfectly straight but narrow lines. These lines are respectively a line of cell proliferation and a line of ossification. If, however, the bone under

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observation is from a rickety subject, it will show typical changes in these lines. They are now both irregular instead of straight. The line of proliferation has become much more vascular, thickened, softer, and of a bluish colour from hyperaemia. The line of ossification is also softer and paler.

Microscopically the cells are seen to have proliferated freely, but not uniformly, being of a patchy nature. The calcification has taken place rather earlier than usual, but here also in an irregular manner. The formation of bone from periosteum shows similar irregularities. There is a similar thickened and vascular layer, which shows the same irregularity in proliferation and calcification. The bone generally shows an increased vascularity compared with what is usually observed in the case of growing bone.

The bones of the cranium, owing to this irregular ossification and therefore slow calcification, are soft, and the fontanelles remain open longer than usual. The bones of the skull can in some cases be indented by the pressure of the finger. The frontal and parietal bones take a much more active part in rickets than the other bones of the cranium, increasing out of proportion to the others. This gives rise to the square-shaped head. The skull bones are more vascular than normal.

The Thorax.- The ribs suffer to a greater extent in rickets than in any other disease. The active changes take place at the anterior ends of the ribs at their junction with the cartilages. Here we see the lines of proliferation and ossification, giving rise to a node at the end of the bone - the so-called "rickety rosary". While this is going on, the rib itself is also suffering from the change under the periosteum, giving rise to softening of the bone itself and causing it to be easily bent.

The Spine.- The bodies of the vertebrae participate in the general softening proceeding, with the result that they yield to pressure.

The Long Bones.- These show marked changes. At the junction of the epiphysis and diaphysis there is a line of proliferation and ossification, giving rise to the thickening of the ends of the affected bones so characteristic of the disease. These changes take place especially at the lower ends of the radius, ulna and tibia, and sometimes the lower end of the femur. This occasions the thickening so often here observed.

The brain and nerves are no doubt affected in rickets, but the pathological changes have not as yet been made out. Some authors seem to think that there is an increase of neuroglia; others that there is an increase in the actual brain itself. The nerves are much more irritable and sensitive than under normal conditions.

The lungs show very important changes. There is a predisposition to congestive changes in the bronchial mucous membrane. There may be congestion of the air cells or smaller capillaries, giving rise to a pneumonic process. In this disease the lung tissue shows a great tendency to collapse.

The circulatory system is sluggish, due to some extent no doubt to the cardiac muscle being affected by the general muscular debility existing in this disease.

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Some authorities hold that there is a relative enlargement of the arteries as compared to the size of the heart, and this would also tend to a lowering of the blood-pressure.

The Blood.- Most authorities hold that there is a certain amount of anaemia in rickets; the haemoglobin has been fixed at 75%.

Morse (Bost. Med. & Surg. Jour., April 22, 1897) found that most of his cases of rickets presented a more or less degree of anaemia, which often varied with the severity of the disease. He also discovered nucleated blood-corpuscles in some of his cases. On all occasions when these were found enlargement of the spleen was also present.

A more recent writer Esser (Münchener Med. Woch., 1907) says that in almost every instance there is a marked leucocytosis.

L. Findlay (Brit. Med. Jour., May 15, 1907) gives the result of a series of eight experiments with puppies, each of the animals being four months of age. After weaning, he kept it in confinement and fed it on porridge and milk. They all developed rickets; and it would seem that in only two cases was there any sign of anaemia observed. One of the animals developed a bronchopneumonia which proved fatal. Instead of there being anaemia, four of the puppies showed a rise in the amount of haemoglobin, and also in that of the red-corpuscle count. The remaining two showed no alterations in either the haemoglobin or corpuscles. His experiments go rather to disprove a condition of leucocytosis than its presence, there being increase of the white cells in only four of the animals. In two of these a bronchopneumonia was found at the autopsy, while in the other two the leucocytosis only made its appearance at the final stage of the disease. The results of our author's investigations are, in his own words, these: "Experimental rickets is not necessarily accompanied by any anaemia, and when anaemia does appear it is, as a rule, of a minor degree, and may be accounted for by some complication. There may or may not be a leucocytosis, and this also when it occurs may be the result of some complication. It can at least be truly said that in blood of animals suffering from rickets there is no marked pathological change, and absolutely nothing characteristic!"

The alimentary tract very commonly shows a tendency to catarrhal changes occasioning such disturbances as vomiting and diarrhoea. There is, however, in certain cases a tendency to constipation. The abdomen is tumid, due to a certain extent to flatulent distension from impairment of the muscular tone of the intestinal walls.

This distension may be aided by enlargement of the liver and spleen, and in some cases the mesenteric glands. This enlargement of the spleen and liver is said by some to be due to interstitial changes. The mouth shows extreme delay in dentition, a tendency to stomatitis, and the other changes in due course to be described.

The muscles are weak and flabby, being badly developed. Likewise the various ligaments are lax, and undergo stretching with greater facility than normal. The skin is pale and unhealthy-looking.

A few remarks on the PATHOGENESIS of the affection come in appositely here. The pathological changes which take place in the muscles and bones in rickets are not definitely understood - that is, whether they are purely functional or otherwise.

Some authorities are of the opinion that, especially in the nervous system, the changes are more of a destructive process similar to those going on in the osseous system.

As to the theories regarding what gives rise to the changes in the bones, these are many and varied. One theory is that the heart being normal in size, the blood-vessels are relatively larger, causing a local hyperaemia accompanied by a lowering of the blood-pressure; this gives rise to an irritative effect on the cells - hence proliferation.

Another theory is that the lungs being insufficiently aerated there is a chronic carbonic acid poisoning, giving rise to all the phenomena of rickets.

Some consider that there is a toxin formed in the intestines, which, entering the blood, occasions the various symptoms.

These theories do not find much favour now, but some of the more important will presently be considered in detail.

ETIOLOGY.

BACTERIOLOGY.

The present state of our knowledge regarding the alleged bacterial excitation of rickets is anything but complete or profound. Several observers are inclined to think that there must be some microorganism at work; in support of the hypothesis they point to the acuteness of the changes occurring in the bones and tissues of the rachitical individual. Be that as it may, it is nevertheless a fact that all attempts, so far, have failed to isolate an etiological germ.

The bacterial theory of rickets seems to have originated in 1860, then Friedleben came across an intestinal ferment, which, he said, must have something to do with the disease; he received the support of both Oppenheimer and Kassowitz, but nothing seems to have come of their researches. Later, Volland raised a suspicion of a specific virus; and in 1894 (Chaumier (Med. Infant., 1894) said that rickets might be due to some sort of contagious and epidemic microorganism which lurked in certain dwellings. Magenbach-Burckhardt (Berl. klin. Woch., 1895) accepted the bacterial theory, impressed as he was by the analogy with which such constitutional affections as syphilis and tuberculosis found regional expression in the bones and spleen; and Mircoli (Gaz. Osped., 1891) in four cases specially examined found in two cases of one series and in four cases of another the streptococcus pyogenes and contingent lesions in the bones; to these, he said, the affection could undoubtedly be referred. Proof, however, was lacking. Marfan and Marot (Rev. Mens., 1893) examined the blood of a rachitic during a gastrointestinal attack and discovered therein the bacterium coli commune and the streptococcus. Under identical circumstances Czerny and Moser (Jahr. f. Kinderh., xxxviii.) came across the staphylococcus pyogenes aureus and albus, the bacterium coli commune, and the bacillus lactis aerogenes. Ettore (Rev. Mens., Mar., 1897) gave etiological interpretation to the last-mentioned germ and others which he found in septicaemic cases and osseous lesions; but he lost sight of the fact that the severe hyperaemia of the bones and their cartilages and epiphyses would account for the accidental findings. Charrin and Gley also failed to prove the point (Soc. Biol. de Paris, Feb. 22, 1896). They managed to produce rickets in rabbits whose parents had been dosed with the toxins of diphtheria and the bacillus pyocyaneus, enteritis being a marked feature of the attack. Lange (Verh. d. 12 Vers. der Ges. f. Kinderh., etc., 1896, p. 144) advanced the theory that a specific virus may infest certain regions and localities and climates, for the reason that rickets is rare in Iceland, Siberia, Finland, China, Japan, and certain other parts.

SYPHILIS.

Congenital syphilis is held by some authorities to be greatly concerned in the production of a disposition to rickets. The former affection, which Parrot regarded as the cause of the latter appears earlier than rickets, being often intrauterine. Parrot's arguments for this causation are that the osteophytes and the spongy osseous tissue are common to congenital syphilis and common to rickets; but there is nothing specific in these lesions. As to the pretended stimata, the cicatrices of the buttocks, the lingual desquamation, the erosions of the teeth, and the natiform cranium, they are encountered as well in non-syphilitic as in syphilitic children. More than this, the statistics of all observers of experience in children's diseases the world over bear witness against this theory of Parrot's. Syphilis is at least as common amongst the rich as among the poor, but rickets is a disease of the poor, and is very seldom seen among the more affluent members of society. Syphilitic parents have been seen to have healthy children, and the children of parents who were certainly free from syphilitic taint have been rachitic. In many cases, moreover, in which syphilis in the parents has been seen to coexist with rickets in the children, inquiry has shown that the disease of the parents was acquired subsequent to the birth of the children and the appearance in them of rickets. Doubtless rickets is not rare in hereditary syphilitics, as it is in all children who are debilitated from any cause; but the influence of syphilis is not specific or direct. It is essentially dystrophic. Cheadle modifies the Parrot's statements by affirming that syphilis only modifies rickets but does not produce it. In short, the majority of modern writers seem to be of the opinion that anything which predisposes the infant to delicacy of constitution, or to premature birth, tends to the appearance of rickets in usual course.

CLIMATE AND SEASON.

Some of the earlier writers affirmed that infants born in winter are more liable to rickets than those who commence life during the summer months. This may be due in some cases to the winter child not getting out into the fresh air as often or as soon as the summer-born infant.

Ruata (Brit. Med. Jour., Aug. 22, 1908) maintains, from a mass of statistical evidence that cold has not any influence upon the causation of rickets, as in the three coldest regions of Italy, namely Piedmont and Venice and Lombardy, the death-rates were respectively, during the five years 1901 - 05, from rickets 1.8, 15.5, and 9.1 per 100,000. In Calabria, a very hot locality, it was 10.8. The highest mortality was 40.8, in Marches, with a climate much milder than the three first mentioned. According to the same author, dampness seems to exercise a very remote effect. In seven of the dampest Italian provinces, these being flat and very wet, the death-rate varied from 2.2 to as much as 111.1; and yet at Ferrara, which is quite close to Rorigo where the death-rate is so high, the mortality figure was only 2.2. Yet all the conditions of life are the same. Contrasting the hilly districts, the same variations are found under apparently the same conditions

of life, the death-rate varying from 2.2 to as high as 49.2.

After studying the foregoing statistics it is not difficult to come to the conclusion that, so far as Italy is concerned, ~~neither~~ climate or locality has much influence on the death-rate in rickets. Nevertheless, it is a well-known fact that a great many cases of rickets improve, and not infrequently markedly so, from a sojourn at the seaside. So much is this the case that it often forms part of the treatment. In these cases, however, it is not the climate alone that is aimed at; it is the health-giving out-door existence which is the object of treatment. In some of our smaller seaside villages rickets is far from being extinct, and for the very sufficient reason that these principles are not carried into effect. The ignorant parent has been brought up to regard fresh air in the house as a death-dealer instead of a life-preserver. The child in these cases is wrapped up, put into its old-fashioned cradle, and relegated to the most remote corner of the gloomy apartment, where perhaps it secures no more fresh air than it inadvertently receives through some unobserved crack.

GEOGRAPHICAL DISTRIBUTION.

There is a general consensus of opinion that rickets is pre-eminently a disease of the zone of Central Europe, and also, at present, of the United States of America and Canada. In Europe, Holland, France, Germany, Hungary, Russia, and Northern Italy it is very common. In Switzerland it is found everywhere, more in the industrial than in the rural districts, and, parallel to the density of the population in general, mainly in the valleys. But the highest Alps have no immunity. In the higher altitudes the children of immigrants from the valleys appear to suffer more than those whose parents were native to the soil of the district. The affection is very seldom seen in the far North. In Norway, Iceland, Finland, it is said to be rare, though in Riga we are told that it is universal. In the cold climates more oxygen is used, more carbonic acid exhaled, the blood contains more cells and haemoglobin. At an altitude of six thousand feet Egger found seven million cells in the c mm. of blood. In high altitudes and on frozen soil the direct influence of the sun is stronger. On Mount Blanc the atmosphere absorbs 6% of the solar warmth only; ~~an~~ on the plains 30% or 40%. Moreover, there are more sunny days above than below. The affection is conspicuous by its absence in tropical countries. It is, I understand, unknown in Africa, Central America, Central Asia, Australia, China, and Japan. Physicians travelling in Naples and Greece have often expressed astonishment at the absence of curvatures and rickets in **general** amongst the half-naked, dirty, and neglected children. It appears that their constant exposure to air and sunlight protects them. Negroes in Africa have no rickets, in the United States of America, a great deal.

FRESH AIR AND SUNLIGHT.

The importance of these two factors have been demonstrated to my satisfaction, and very strikingly too, on innumerable every-day occasions. During the years 1896-99, I had considerable experience amongst the colliers ~~both~~ in Scotland and in the North of England. Whilst in the latter locality I had medical charge of

one of the largest ~~colleries~~ collieries in England, and in the miners' families I found that rickets was a comparatively rare disease. Now, these miners live in small houses of two or three rooms. They very often have large families; in fact, I might say - very large families, - for I attended in one case a pitman's wife in her twentieth accouchment; yet there, as I have said, rickets is rare, and at first I wondered why. The conclusion I arrived at was simply that the people did not, metaphorically, know what a closed door was. Seldom the door closed from morn till eve; and very often for the greater part of the night the door stood wide open - with the result that the people practically lived in the open air. It is interesting and instructive to contrast this with the conditions which obtain amongst the working people of our cities and towns, amongst whom it is the rule to have both the door and window closed week in and week out, except when absolutely necessary on unavoidable occasions temporarily. The natural outcome of this is that the atmosphere of the apartment, or apartments, becomes heavy and malodorous, and inimical to the enjoyment of good health.

The contrast is even more striking when sunshine is taken into consideration. The miner's house stands in a block of low buildings set sufficiently apart to allow of the sun shining freely into the rooms. Further, all clothes are aired outside in a garden some distance away from the home.

In the city I have entered sick-rooms in which the very rickety patient was lying in a heavy atmosphere, pungent with odours, and stifling. There would perhaps be a rug, or some other contrivance, laid down at the bottom of door and over the windowsill to keep out any draught. In some instances there would be a line or two of baby-clothes hanging along the roof to dry, and in the worst cases the window would be darkened by the neighbour above hanging out her washing over it. Not infrequently, whilst visiting patients in the low quarters of this city, I have had to fumble about in the dark for the door, and sometimes have been unable to discover it without a light of some sort. The passages and landings are in many cases absolutely dark, and the staircase itself dirty and badly ventilated.

Findlay (Brit. Med. Jour., July, 1908), in an excellent article giving the results of his experiments on puppies, and already referred to, tries to make out a case for want of exercise as a great factor in the production of the disease. He would, I think, have made his results much more convincing had he shown that his experiments were carried out on animals living in properly ventilated kennels with plenty of light. His "kept in confinement" and "kept in moderate confinement" are much too vague terms to be completely and thoroughly understood, especially where these two important questions are concerned.

In short, I consider bad air and darkness as amongst the most important causes of rickets. Moleschott proved an increase in the elimination of carbonic acid with the amount of light striking the skin and retina. Deep-sea animals, though they belong to the bone-fish varieties, have a cartilaginous skeleton. After respiratory disorders rickets is often observed. It is true,

however, that impeded respiration may be accompanied or followed by all sorts of constitutional anomalies. Animals imprisoned in badly-ventilated stables become rachitical. Soldiers' children locked up in their barracks in the East Indies are, according to Spencer Watson, rachitical, though those of the poor outside the barracks are not. I have shown that large industrial towns and cities have more rickets than the country communities. Still, J. P. West (Univ. Med. Mag., Oct., 1895) denies the absence of rickets from the country districts, and refers to the fact that mild cases are but rarely mentioned at all. In every village of his neighbourhood he found signs of rickets in evidence. In nine counties surrounding his residence there were, to his knowledge, eight rachitical dwarfs, three of whom were members of well-to-do Scotch families. The factory population is mainly affected. Nor are the well-to-do immune. In riga the latter class suffer like the poor; 86% of the infants are rachitical because of the long duration of the winter, namely eight months, during which the infants are kept at home. Most of my cases have been observed in the spring, the babies having been housed all winter. I have seen many less, even after the prevalence of summer diarrhoea, in the autumn or towards the end of the year. I have already shown that in Naples and Greece those badly fed will thrive when constantly out of doors in the fresh air.

SEX.

Rickets, according to my observations, attacks both male and female children in about the same proportion, though at one time I was inclined to the opinion that the latter class were more rachitical than the former.

MULTIPLE PREGNANCY.

I have always regarded twin children as liable to rickets because of insufficiency of food and incompetent attention to cleanliness, air, and so forth.

AGE.

Rickets is essentially a disease of early childhood. The symptoms of the affection may be observed as early as the second or third months of infancy, or the malady may delay its appearance until as late as the second year. The commonest age, however, is between the fourth and fifth month. Cases of late rickets have been reported as occurring in the teens, though then quite an exceptional phenomenon. The affection, it is said, is sometimes developed in utero; it may then be found at birth to have already run its course (foetal rickets), or it may progress after the child is born (the congenital rickets of Winkler). Winkler also includes in these congenital cases infants who show rachitical osseous changes during the first few weeks after their birth; and says that in such instances both the tendency to the disease and its origination are of intra-uterine reference. Boerhaave, Van Swieten, Zamboni, and others deny the existence of foetal and congenital rickets altogether; and it would seem that Glisson, Storch, Morel, Klein, and not a few others have erroneously included certain cases in this category, e.g., various deformities of the osseous system seen in the foetus and the child at birth of non-rachitical origination. Several of the

earlier writers describe cases of rickets after puberty prior to the full development of the skeleton - to 18 or 20 in the female and to 22 or 25 in the male. Glisson, for instance, had two cases between 16 and 17; Portal saw five cases from 15 to 18. Ollier regards spinal curvatures in previously non-rachitical individuals as the result of a rickety disposition - rachitisme tardif; and Tripier saw five cases of rickets in young men. Bearing upon the point under consideration, the following statistics may be quoted:

Guérin's Statistics.

Before birth	3
During 1st year	98
" 2nd "	176
" 3rd "	35
" 4th "	19
" 5th "	10
From 6th to 12th year	5
Total	<u>346</u>

Von Rittershain's Statistics.

During first six months	91
" second " "	175
From 1 to 2 years	154
" 2 " 3 "	62
" 3 " 4 "	15
" 4 " 5 "	7
" 5 " 9 "	17
Total	<u>521</u>

Ritchie's Statistics.

During 1st half-year	7
" 2nd " "	65
From 1 to 2 years	109
" 2 " 3 "	25
" 3 " 4 "	9
" 4 " 5 "	4
Total	<u>219</u>

Bruenniche's Statistics.

During 1st half-year	1
" 2nd " "	19
From 1 to 2 years	79
" 2 " 3 "	47
" 3 " 4 "	7
" 4 " 5 "	6
" 5 " 8 "	<u>14</u>
Total	<u>163</u>

HEREDITY.

According to Osler, heredity has nothing to do with the production of rickets. On the other hand, others insist that the contrary obtains. Parrot even goes the length of saying that the symptoms are due to syphilis in the parent, whereas Cheadle holds that syphilis only modifies rickets, but in no way produces it. Boerhaave also claimed rickets to be almost exclusively the result of hereditary syphilis. This view was strongly opposed by Van Swieten. Later Kassowitz took up Boerhaave's position, and as we have seen, Parrot still more so. The identity does not exist. Cazin and Icovesco (Arch. Gén. de Méd., Sept. & Oct., 1887) studied carefully one hundred and nine rachitic infants. In them syphilis was not more frequent than in the average infants observed. The influence of any and every constitutional disease of the parents or general condition

of the offspring is liable to cause a disposition to rickets. Syphilis is very common in Africa, China and Japan, all of which countries are notorious for the almost entire absence of rickets. The theory of the heredity of rickets received plausibility from the number of children rachitical in the same family and the occurrence of congenital rickets. But if we investigate the hygienic conditions of these cases of apparently inherited rickets we shall find that in many instances it is not the germ of the disease which is inherited, but rather the unhygienic habits that are transmitted from parents to child. It is, indeed, not uncommon to see a woman who has been rachitic give birth to children entirely free from any evidences of the disease and who will surely escape it if they are well fed. Rickets is heredity only in the restricted sense that children who are born of weak, delicate parents, and of those who are too old or syphilitic or tuberculous, etc., will be disposed to the disease. On the whole, the majority of modern writers seem to be of the opinion that anything which leads to a delicacy of infantile constitution or to premature birth tends to the appearance of rickets in such children. I have seldom seen children of healthy parents living a healthy life develop rickets; while, on the other hand, I have often noticed the disease make its appearance in the children of delicate parents who at the same time were living under unhealthy conditions.

ANTENATAL INFLUENCES.

If the mother is suffering from some debilitating condition during her pregnancy, such as phthisis or starvation, or ~~er~~ has too often conceived, it is quite natural to suppose that the infant, when born, will inherit a delicate constitution. The same factor, in my opinion, may obtain when either the father or the mother of the child is syphilitic. Ritter ~~u~~. Rittershain in particular showed that various constitutional maladies in the parents may have something to do with the appearance of rickets in the offspring, especially tuberculosis. He found seven tuberculous fathers and four tuberculous mothers among the parents of seventy-six rickety children whose family history he was able to investigate. To sum up, debility from whatever cause, anaemia, chronic discharges, enfeebled nutrition, etc., in one or both parents, advanced ~~at~~ ~~the~~ at procreation, may all promote the development of dyscratic affections in general, and of rickets in particular, in the child.

RACE.

According to Osler, rickets is very prevalent amongst the negroes and Italians in the slum quarters of New York City. I have already advanced statistical observations to establish its frequency in Italy.

HYGIENIC DEFECTS.

These have already received some consideration in a previous section (Climate and Sunshine). Fresh air and a maximum of sunshine are essential to the hearing of a healthy infant.

Quite recently I was called in to see an infant of a family consisting of three children aged respectively five years, three years, and four months. They lived in a small room, the doors and windows of which were not only closed, but rugs and curtains were hanging over them to keep out the draughts. There was, moreover, a double line of clothes hanging from the

roof to dry. A large fire was burning in the fireplace, and on entering I was almost overcome by the pungent and oppressive air. The child, as might be expected was very ill and suffering from bronchitis and general debility.

Another thing very difficult to impress upon ~~some~~ mothers is the necessity for keeping the feeding-bottle scrupulously clean. In every case I endeavour to persuade them to use the slipper pattern, stating as my reason the comparative ease with which they can be cleaned and kept so. There are, however, not a few mothers who insist on using the old-fashioned appliance with a long rubber tube. These misguided creatures tell me that they have no time to sit feeding the baby, with the result that they allow the infant to lie in its cradle and suck at its bottle until it falls asleep. The element of convenience here largely obtains; the mother can go on with her work or depart for a gossip with her neighbours, the bottle being left with the child until the cries of the latter show its needs replenishing. The bottle is then given a hasty and careless flush out with water, filled, and the pernicious practice repeated. As emphatically as possible I always insist on the bottle being cleaned immediately the child has finished with it, and then carefully laid aside in a basin of boiled water containing some boracic acid. The rubber parts of the appliance need some attention also: otherwise they very soon become sour. They should be regularly scalded with boiling water at least once a day, and the immensely popular so-called "comforter" calls for the same treatment. Indeed, I have not infrequently seen slight cases of rickets improve immediately these precautions were taken, and this without any alteration in the diet.

PARENTAL AGE.

In itself the age of the parent had little to do with the production of rickets; but I do think that when the mother is advancing in age and has had frequent and close pregnancies the tendency to produce rickety children will increase as she grows older. This fact will be referred to again presently.

DIET.

I look upon vicious alimentation as a most important etiological factor if accompanied by the want of fresh air and sunshine.

No matter what authority be consulted, they all agree that some dietetic error is at the root of the evil. Though the affection may occur in breast-fed as well as in bottle-fed children, it is undoubtedly more apt to attack the latter. This is due, I think, to the fact that in bottle-feeding the mother has more or less a free hand in the choice of artificial foods or milk-mixtures.

Breast-fed infants may acquire rickets from a variety of causes. Perhaps the mother's milk is at fault, being deficient in the proper constituents, or the child may be fed injudiciously, or, again, the infant may be unable to assimilate the food although it be of proper composition and good quality. The milk may be deficient in fats and salts, being of a watery constitution. This may occur in a woman who is of delicate constitution or who is debilitated by frequent pregnancies at short intervals. She may be so intensely neurotic that she

worries over the child, especially if it is slightly indisposed, with the result that she is unable to sleep and loses her appetite. This has sooner or later an unfavourable effect upon the milk, which now disagrees with the infant and causes the latter to vomit it after every feed. In three such cases I have had to order weaning, and have substituted artificial feeding with the happiest result.

Another factor in the causation of rickets in breast-fed children is the continuous feeding which sometimes take place during the night. It is, according to my experience, quite a common thing for mothers to tell me that the baby has had three or four feeds during the night, and it would appear that sometimes the number cannot be reckoned. If the infant is restless during the night, rather than nurse it the mother presents the nipple in the hope that the infant will satisfy itself and ~~and~~ follow her example by going to sleep again. The result is that the stomach, instead of obtaining its natural rest, is kept constantly at work, and ultimately develops a gastritis, which in not a few instances paves the way for rickets. In some cases, no doubt, it is due to some fault on the mother's part: she may not, for instance, be taking or able to get sufficient and proper nourishment to keep her milk up to the proper dietetic standard.

Another maternal cause of considerable importance, especially amongst the poorer classes, is the desire of the mother, for various reasons, to nurse her infant as long as possible. The usual thing is for them to nurse for at least a year or more; some I have discovered endeavouring to prolong lactation for two years. The natural result is that the woman suffers in health, and the milk becomes almost daily impoverished in quality.

On ten occasions such as these, I have had experience of women becoming pregnant whilst nursing and only consulting me after "quickenings". In eight cases I have found the child at the breast nursing and inclined to be rachitic, while the foetus after birth did not develop as it should have done. The condition of the older child I ascribed to the poor quality of the milk consequent upon the general debilitation of the mother. The condition of the younger infant can be explained by the poor composition of the milk produced by an over-stimulated mammary gland.

There is considerable difference of opinion as to whether a mother should continue to nurse her infant after menstruation has again become re-established. It is my custom to advise the woman to stop suckling unless both she and her child are particularly strong and robust.

Dingwall Fordyce (Brit. Med. Jour., 1806), in an analysis of one hundred cases of rickets, found that there was a decided tendency to the disease in the later children of the same parent. In fact, when he came to the third or later children he found them just as liable to become rachitic as the ordinary breast-fed infants. His figures are: 25% of first children develop rickets, 41% of second children, 58% of third children, and 65% of later children. "Bottle-feeding, per se, has," he says, "no more greater tendency to produce rickets amongst later children than amongst earlier!"

What the ingredient in the diet which causes rickets in the bottle-fed is ~~cannot~~ be definitely affirmed; but it has been pretty conclusively shown that the want of sufficient fats and salts leads to the appearance of the disease; not infrequently also a distinct improvement immediately follows when cream or oil is added to the dietary. Personal observation has taught me that the dyspepsia caused by the too early administration of carbohydrates to young infants has a great tendency to produce rickets. This is especially the case if the child is naturally of a weak constitution. The carbohydrate is given in the form of bread, biscuits, sweets, sugars, etc. The salivary glands have not materialised, and their action on the carbohydrate is not forthcoming - with the result that extra strain is put upon the stomach and the inevitable dyspepsia arises. The diet must contain an adequate amount of lime salts in order that ossification of the bones may proceed; One writer estimates that an infant derives something like ~~from~~ twelve to fifteen grains per diem from ordinary milk.

In general, then, great weight must be given to the elements of hygiene and nutrition in the causation of the disease. There is less rickets in rich or affluent families than amongst the poor. Bad foods - with, according to D'Espine and others, dilatation of the stomach and toxic absorption - such as large percentages of starch or undiluted cow's milk, are dangerous; but when babies are fed on the same faulty diet in the valleys and on the high Alps, those on the latter suffer less from rickets; it is particularly undiluted milk that is better tolerated on the high mountains than in the valleys. In the favourable climate and outdoor life of Athens, where the babies are mostly weaned after the second month and farinacea given, rickets, as I have already shown, is not frequent. Breast children suffer less from rickets than those raised ^{on} artificial food; still, I have seen plenty of infants whose breast milk contained an undue degree of casein that required weaning, and required some well-selected artificial food in order to overcome their rickets. Marked absence of fat from the food is also liable to produce rickets. Cheadle refers to the fact that rachitical animals of the Zoological Gardens got well when fed on fat meat. The small amount of sodium chloride compared with potassium in vegetables is the cause of salt hunger of animals and of the necessity of adding salt to cereals, potatoes, and cow's milk. It is required to counteract the superabundance of potassium and furnish a sufficient amount of hydrochloric acid in the stomach, which is required for digestion in general, and mainly for the solution of the calcium of the ingesta. Potatoes require a good deal of sodium chloride. Wagner attributes the rickets of poor people, Stockfleth that of the swine in Norway, to the influence of potato feeding. Still, when Glisson wrote his epoch-making work on rickets in 1650, there no potato feeding. In the opinion of some, the charge that starchy foods in general, no matter how given or in what quantities, must needs be the cause of rachitis, is exaggerated. This belief is the result of a fanatical insistence on the dogma that no young infant can digest starch, no matter in what combination. The teachings of

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Zweifel and Korowin, proving the very contrary, have been assiduously neglected this fifty years or more; and the advocacy of some writers of the addition of cereal decoctions to cow's milk has found as many loud or silent adversaries as disciples. They feed the babies on rice in China and Japan, on vegetables of many kinds in Africa; and according to Lange (Verhandl. d. 12, Vers. d. Gesellsch. f. Kinderh. in Lübeck, 1895, p.144), breast feeding is seldom seen; yet there rickets is a rare disease. Finally, uncleanness has been accused of being the cause of rickets, but the poor peasant and the Chinaman are very unclean, and the cases of rickets amongst them do not increase with their dirtiness. But this much is true: that uncleanness, bad dwellings, the foul air of tenements and factories, and improper food go very often together, and will make a child of tender age rachitical, the more so if there be a concomitance of respiratory or gastrointestinal disorders.

THEORIES.

In conclusion I may here briefly enumerate the chief pathogenic theories of rickets:

(1) Parrot's Theory.- This, as we have seen, is that of the syphilitic origin of the disease, vicious alimentation being imoperative.

(2) Nervous Theory.- According to Pommer and Tedeschi, rickets is a trophic affection of the bones originating through the central nervous system.

(3) Berm Theory.- Mircoli's idea is that it arises from a specific microorganism, and Chaumier's that it is an essentially contagious disease.

(4) Alimentary Theory.- For Glisson, Petit, Guérin, Trousseau, Chossat, Roloff, Gamba, Fonssagrives, Cheadle, and others rickets is a nutritional disorder due to vicious alimentation. The last-mentioned author and others produced rickets in animals by withholding all the lime salts, especially the phosphates. The young bone-forming cells seem to steal the lime from the already ossified bone, and so render the whole of the bone soft. This was demonstrated by Baginsky and ~~Wolters~~ ~~Wolters~~ respectively. This led some of the early writers to adopt the lactic acid theory, the idea being that this acid was formed in the stomach, entered the blood, attacked the ~~salts~~ salts of lime in the bones, dissolved it, and passed it on to the kidneys for excretion. Jacobi, Heitzmann, Boushard, and others regard rickets as the result of faulty feeding, and say that it is preceded by digestive disorders, chronic indigestion, dilatation of the stomach, or enteritis, which seriously impair the process of assimilation and favour the decalcification of the bones.

Much controversy has in the past centred around these theories; but I think that all nowadays who intelligently examine for the cause of rickets cannot fail to be impressed by the frequent combination of dietetic and hygienic errors.

SYMPTOMATOLOGY.

GENERAL OUTLINE OF THE DISEASE.

Rickets is essentially an insidious disease affecting all the different systems of the infantile body to a greater or less degree. It is difficult to say what is the first indication of the invasion of the rachitical condition.

In the vast majority of cases which have come under my observation, I think the child's mother has given me a clue to the existence of the dyscrasia. She informs me perhaps that "baby is not himself at all; he requires a great deal more nursing than usual; he is restless, peevish, perspires about the head at night, and sleeps badly!" In a great many cases I have found gastritis to be a forerunner to rickets. This may be accompanied by vomiting, constipation, or perhaps diarrhoea, and the infant seems not thriving as he should.

When rickets has become well established it presents a typical picture which, once seen, can scarcely be forgotten. The child, say nine months old, is on the mother's knee or in its cradle. It has a characteristic thin, drawn face. The head appears to be large in proportion to the size of the face. The arms are thin and wasted and accompanied by typical enlargement of the wrists. The chest is pigeon-breasted, from prominence of the sternum, with two lines of depression on either side of it. Outside of these depressions is a double row of nodules on the ribs, from the first to the twelfth. This is the so-called "rickety rosary!" The thorax itself is of a peculiar square contour, especially at the upper part. The lower part is often relatively large, being pushed out by the enlarged abdominal viscera. The abdomen is usually tumid and large, with hypertrophy of various viscera; tympanitis is also not infrequent. The lower extremities are thin, and the skin over the buttocks and thighs often presents a peculiar wrinkled appearance. At the ankles and sometimes the knees are found enlargements on the bones similar to those occurring at the wrists. The spine usually presents a posterior curvature involving the lower dorsal and lumbar vertebrae. This is best seen when the child is sitting up, as it often disappears in the recumbent position. The patient is badly nourished and looks more like one of four or five months instead of nine months.

On innumerable occasions I have noticed that the infant has a peculiar cry, which is quite different to any I have heard in the case of healthy children. This cry is emitted whenever the child is in the least disturbed or handled.

Another point of personal observation is the peculiar habit the patient has of appearing to chew its hands when crying. This, I think, has led mothers

to suppose that the child was being starved; and I have often at the very first visit been asked if this was not actually the case, despite the fact that inquiry showed that overfeeding, as demonstrated by undigested food particles in the stools, was actually being practised.

The patient has a troublesome cough, and on auscultation numerous adventitious sounds may be heard in addition to the disturbed respiration. There may be a bronchitis or even a bronchopneumonia.

On examining the mouth, it will be noticed perhaps that the teeth are not yet cut. Instead of the first tooth appearing at the sixth month, it may not do so until even the twelfth month or later. The dentition, then, is greatly retarded, and it may not be completed until the third or fourth year of life. Further, after appearing they very soon decay, early exhibiting this tendency.

The bowels are very often constipated, especially in the early stages; but diarrhoea may later on appear. The stools have a peculiar offensive odour; they are not infrequently green and mixed with flakes of undigested milk-curd. The abdomen is tumid from enlargement of the liver, spleen, and lymphatic glands.

The nervous system, too, does not escape, the infant being very susceptible to convulsive attacks.

Another type of the rachitic child is the big well-nourished infant about a year or so of age. It shows few or no constitutional symptoms, except perhaps a tendency to bronchitis. The most striking feature is the backwardness in walking, and the great tendency to deformities on the part of the bones. If the child has already been able to walk, it not infrequently goes "off its legs" again, and does not regain the faculty for a considerable time. In all such cases there is always retardation of locomotion observed.

Rickets in itself is not a fatal disease; but its numerous intercurrent ailments are very apt to terminate the life of the patient; especially is this the case as regards bronchopneumonia and convulsions.

THE LEADING CLINICAL PHENOMENA IN GREATER DETAIL.

Having thus briefly outlined the general features of the disease, I will now consider them more fully as they appear in connection with the various organs and tissues.

Changes

OSSEOUS SYSTEM.

~~Changes~~ occurring in the bones are typical and give rise to serious and far-reaching consequences. In the early stages there are acute changes occurring between the epiphyses and the shafts of the long bones. These changes result in perverted ossification. There is imperfect and delayed bone-formation accompanied by absorption of bone, also at parts increased osseous deposition. This latter causes the nodes or enlargements to appear at the ends of the long bones, while the imperfect ossification causes the bone generally to be soft and easily bent. They therefore become deformed by outside forces, which are the weight of the child, muscular action, and atmospheric pressure.

LEGS.

Under the action of the forces just mentioned

the legs show early signs of giving way. This bending varies very much according to whether the infant has been or is able to walk or not. In one hundred cases of rickets of this type, and which have come under my observation, I found that the commonest deformity was a bending of the lower end of the tibia or a general bending outwards of the femur and tibia.

In two hundred cases I found the lower limbs deformed in various ways. In some cases where the infant was carried a great deal by the mother I found an antero-posterior bending of the femur doubtless due to the pressure of the mother's arm. In other cases where the child sat or crept about on the floor to a greater or less degree I found the lower third of the tibia bent outwards and the feet rotated outwards. This was caused by the inner aspect of the foot being pressed against the floor during creeping.

It is in the case of the poor delicate child, who developed rickets in early life and who has proved amenable to treatment so far as to be able to creep about, or even walk a little, that the deformities often become most severe. There may be bow-leg, and knock-knee of the other. There may be outward curvature of the femur with little or no deformity of the tibia. There may be rotation of either of the bones on their vertical axes. In fact, there is no end of combinations of deformities appearing in the lower limbs.

Various authors report a condition termed coxa vara, in which the head of the femur becomes depressed until it is in a line with the trochanter or lower, the neck of the femur being at the same time bent forwards. Looking through my case-histories, I fail to find evidence of such a condition.

ARMS.

The upper extremities are not so prone to deformity as the lower ones; but deformities may be encountered in cases in which the child supports the body by the arms whilst creeping or sitting. The commonest type of deformity is a curvature outwards of the humerus and bones of the forearm, more especially the former. The clavicle may in some cases become more prominent than normal, due to an exaggeration of the normal anterior curve.

RIBS.

Very important changes may occur in the chest wall. As already stated, there is the formation of nodes or rosary at the anterior ends of the ribs. These are of very little importance except from a diagnostic standpoint. Far more important are the changes taking place in the soft and yielding ribs and their cartilages. They are drawn in by the suction of the diaphragm, aided by the action of the atmospheric pressure. This is most marked in the upper part of the chest, and occasions the square-shaped chest so often present in this disease. The lower ribs do not suffer so much; in fact, they are often bulged out by the enlarged abdominal viscera. From the same cause the soft cartilages between the ribs and sternum are pulled inwards, giving rise to the typical groove in that position and causing the sternum to appear more prominent than normal. Laterally, above the diaphragm, not corresponding with the diaphragm (for this extends from the ensiform process to the twelfth ribs) and both liver and spleen prevent the ribs

from moving inwards) there is a horizontal depression called Harrison's groove.

SPINE.

The bodies of the vertebrae suffer in general, participating in the softening process, with the result that they give way under the weight of the body. The most typical deformity is one of posterior curvature of the lower dorsal and lumbar vertebrae. This is best seen when the child sits or stands, and it very often disappears to a considerable degree when in the recumbent posture. In only one case have I observed curvature in the cervical region, the patient being a child with an enormous head.

PELVIS.

This is liable to give way under pressure transmitted to it through the femur and spinal column. The result of this pressure is to push in the acetabula, causing them to approximate. The sacrum is pushed downwards and forwards towards the symphysis pubis. These alterations in the shape of the pelvis have sometimes a very serious effect on the female during labour.

SKULL.

The skull usually shows marked changes in rickets. In the case of the normal infant the two lateral - temporal - anterior fontanelles, and the two lateral - lambdoidal - posterior fontanelles disappear within two or three, the small - parieto-lambdoidal - fontanelle within four or five, the large - parieto-frontal - within fifteen, months. In rickets they remain open many months, the large sometimes for two, three, four, or even nine years, as I have seen it. As a rule, they are larger than normal. Very often I have found it easy to hear over them a systolic murmur, which, however, I do not regard as positively pathognomonic of rickets. I think it is probably the result of the increased width of the artery, which is compressed in the stenosed carotid canal. The edges of the sutures are irregular. Such a head is frequently ^{actually larger} than the normal. Relatively, it is very much larger when compared with the usual small body. This is so, though both the longitudinal and the transverse diameters are but slightly increased. In rare instances the rachitical head is so large that it resembles the hydrocephalic head. Indeed, some of these heads are to a certain degree hydrocephalic; some are entirely so. Still, it must be remembered that there are many rachitical heads of normal size. Most of them are brachycephalic, quadrangular, rather flattened on the top. In a peculiar class of cases, first studied by Virchow, namely, that of the cretins and semi-cretins, rickets, which in these cases begins in foetal life, is combined with a premature ossification of the occipito-sphenoidal synchondrosis. In this condition the base of the skull is shortened. At the same time there is a deep grooving of the root of the nose, the eyes are widely separated from each other, there are shortening of the vomer, and a flat palate. Not infrequently the occiput is slightly flattened, and the oblique diameters are sometimes equal, so that one side may appear to be entirely flattened. This is especially the case in the rachitical softening of the cranial bones to which the term craniotabes has been applied. Besides the patency of the fontanelles due to retarded formation of osseous tissue

at the edges of the cranial bones, or to the softening of such as are already calcified, the rachitical cranium may exhibit any number of more or less marked perforations. These are particularly characteristic of craniotabes. Mostly in the parietal bones, sometimes in the occipital, rarely in the frontal, where I once saw more than twelve, there are a number of spots of the diameter of one-half to one and a half centimetres, mostly with steep margins, transparent, without any osseous tissue left, and giving way, as I have proved in several cases, under gentle pressure of the finger like a sheet of paper or thin cardboard. Tyson (Prac. of Med., 1897, p. 780) was mistaken when he said that "as craniotabes occurs in connection with syphilis and other wasting diseases in infants a few weeks old exhibiting no other signs of rickets, and in newborn infants, it cannot be regarded as pathognomonic", and characterises craniotabes as "large areas of delayed ossification in the parieto-occipital regions, producing yielding spots." Neither elsewhere nor in the cranium does the rachitical process consist in delayed ossification. On the contrary, the rachitical bone is that which was developed normally, and when the child is taken ill is normally absorbed and abnormally reconstituted. The bone thus softened is easily and locally absorbed by pressure, this working both from inside and outside, that is, from brain and pillow. In other words, craniotabes is a bone-softening resulting from precocious decalcification which may even be congenital, this craniomalacia being found in certain parts usually of the skull. The most characteristic feature of craniotabes for clinical purposes is the presence of soft spots, not along the sutures, but at some distance from them within an area of more or less normal bone. The transparent defects in the bone may have margins of different nature. They may be sloping or quite steep. In this way the rachitical cranium may be readily distinguished from congenital aplasia or syphilis of the skull. There is, when craniotabes makes its appearance between the first and third months of life much perspiration, particularly of the head, with loss of air upon the occiput. The veins are more dilated, the skin thinner and paler than on the average head, the scalp is very sensitive, the infants cry when laid down, feel better when they are taken up or when they are lying on their faces. In these cases of craniotabes one side may be flattened, usually the one which will be more softened, and the other bulging. The head may even appear to be triangular, where one side bulges out, and one side is flattened from pressure of the pillow; the forehead is very prominent, the frontal bone sometimes from three to five times its normal thickness, because of the immense amount of new periosteal soft growth between the periosteum and the bones. There is sometimes a groove in front of the coronal suture, which results from this steep thickening of the frontal bone. This is not always temporary. It is true that craniotabes may leave no trace if it is speedily recovered from. But when there is much deposit under the periosteum it will sometimes remain. When calcification takes place very suddenly, then thickening of the bone will remain unabsorbed for life; the contrary, however, usually obtains.

DENTITION is also affected in rickets, the teeth appearing late or irregularly, not infrequently one instead of two at a time. When they are early, the intervals between the first pair and the second, or between the latter and the third, are very long; sometimes, according to my experience, six or eight or ten months. The teeth are often discoloured, and they decay very easily because of the absence of cement. Sometimes, however, after recovery from rickets the permanent teeth are very hard and even yellow. Very often I have seen in the temporary teeth of rachitical children what may be compared to the Hutchinson form of syphilitic permanent teeth. Many of my cases have shown longitudinal groovings and semilunar furrowing of the incisors. The syphilitic theory of rickets was in this way suggested to Parrot.

The FACE may also present anomalies. Thus, the lower jaws may be short, narrow, and very low, flattened in the middle by the contracture of the muscle on the floor of the mouth. The angles are very sharp; therefore, the space for the teeth is cramped and dentition may become difficult. While the angles of the lower jaw are prominent, the alveolar processes turn inwards. Consequently, the teeth of the upper jaw do not correspond to those of the lower jaw, and the space for the teeth becomes still more inadequate. A rachitical subject sometimes shows a very low chin. In the upper jaw the alveolar processes of the posterior parts are pushed out; the anterior parts become triangular and prominent. Thus the upper incisors stand out very much beyond the lower. Sometimes the maxilla is asymmetrical. In many cases there is a depression of the canine fossa. The corresponding incisors are still more prominent in them.

TRUNK.

Rachitical individuals are not infrequently very short in stature. Shaw says that the impairment of growth occurs more in the upper than the lower half of the body, the former being a third, the only, merely a thirteenth less than it should be. Ritter von Rittershain reports that in his forty-two rickety cases only one of them was of normal stature for age, the rest being all defective in this respect. On the termination of the disease active lengthening of the body is resumed, but not infrequently the height lost can never be regained, and they remain dwarfish for life, when though not exhibiting perhaps the characteristic deformities of the disease.

NERVOUS SYSTEM.

The rickety child is very irritable and peevish. Some authorities hold that there is a corresponding increase of the size of the brain along with that of the skull. This may be so in some cases; but it is nevertheless a fact, accepted by the majority, that rachitic children, instead of being more intellectual than fortunate other not so afflicted, are inclined to be dull and stupid. Three nervous phenomena which are very apt to appear in rickety children between the ages of six months and two years are convulsive fits, laryngismus stridulus, and tetany.

Observation has convinced me that all three occur at one and the same time, sometimes one and sometimes the other being prominent. They are also closely related

to another nervous phenomenon - that of head-rolling and also to face twitching. I have noticed these latter very often ushering in a convulsive attack. This has impressed me so much that, whenever I see a rickety infant twitching its face and rolling its head, no time is lost in administering the bromides. When the child does take one of these convulsions it loses consciousness, its eyes are turned up, the mouth twitches, the limbs are stiff and may also twitch, the fingers are tightly clenched, and the toes are flexed. The face is cyanosed, and the mouth usually exudes a frothy saliva. When the infant comes out of the attack it very often does so with a peculiar crowing inspiration, as in laryngismus stridulus. The cause of these attack I refer to a reflex irritation of the intestinal mucous membrane, aggravated by constipation and accompanied by irritation of the gums by dentition. My reason for so doing is that if the bowels are thoroughly cleaned out and the gums relieved, the fits often disappear. The irritability of the nerves is well shown by the increased reflexes. The muscles are also very irritable, and the least tap in some cases causes their contraction.

Tetany is rare as a complete manifestation; it may be referred to the common digestive derangements of rickety children and is probably a neurosis of toxic origin.

Laryngismus stridulus is a symptom complex sometimes observed in rickets, consisting of many very short inspirations or more frequently of complete apnoea, followed by a long, crowing inspiration. Before the latter takes place, brought on by the accumulation of carbonic acid in, or absence of oxygen from, the nerve centre in the ~~medulla oblongata~~, death may occur; but that is not a frequent occurrence. Sometimes an attack of laryngismus stridulus is accompanied by a general convulsion. The large majority of cases are connected with, or rather dependent upon, cranial rickets with its hyperaemia and effusion. Still, this almost universal experience is doubted by some.

Every form of hydrocephalus may follow the rachitic process. Afterwards, when the craniotabes has healed, the secondary effusions generally also will disappear; but not a few cases of hydrocephalus may be traced to rickets occurring during the first year of life. When that occurs, the intellectual faculties may suffer, while, on the contrary, complete recovery not infrequently exhibits an unusual degree of mental development, for the same reason which improves the chances of the development of the bone. The condition doubtless arises from the hyperaemia of the bones and meninges surrounding the brain, aided by the sluggish condition of the circulation. In these cases the head is particularly large, with open fontanelles and sutures very late in ossifying.

SKIN.

The skin participates in the general malnutrition. It is often pale and wrinkled, giving the impression that it will require much filling up. The sweat glands, especially about the head, are prone to become very active, particularly when the child is asleep. Whether this is due to some abnormality in the blood, or to some reflex condition of the nervous

system or not seems doubtful. I am inclined to subscribe to the latter theory, and for the simple reason that ~~it~~ ~~is~~ to my mind, if it were a blood condition, the sweating should be general and not localised to one particular part of the body. In quite a number of cases I have noticed a small papular eruption, most usually about the neck and on the trunk. In other cases I have found large red patches - "heat spots", as the mothers call them. They seem to be very irritable, and I consider them a form of urticaria.

RESPIRATORY SYSTEM.

Numerous important changes may occur here. Laryngismus stridulus has already been described. Bronchitis is almost always present to a greater or less degree. The changes in the ribs, causing them to be drawn in, diminish the capacity of the chest, and this, along with the presence of bronchitis and the hampered action of the diaphragm, causes a great tendency to collapse of the lungs. In this condition, should the child contract the slightest chill, bronchopneumonia is almost certain to supervene, and not infrequently with a fatal result. This semi-collapsed condition of the lung, in my opinion, also tends to the development of tuberculosis at a later date.

INFECTIOUS DISEASES.

The presence of infectious diseases, especially measles and whooping-cough, are much more serious in the rickety infant than in an otherwise healthy subject. This can quite well be understood when the bronchitic symptoms of these affections are added to those already present. It is likewise evident that the presence of any of the infectious diseases must be of a more serious interpretation in a child which is already greatly debilitated.

CIRCULATORY SYSTEM.

The consensus of present-day opinion is that the circulatory organs present no special pathological change. There is, however, in some cases a mechanical displacement of the heart. This occurs in cases where there is extreme deformity in the chest wall, occasioning the heart to be pushed out of place. This may give rise to secondary tachycardia. In several cases I have heard a systolic murmur which was transmitted up the larger vessels. I think this can in most cases be regarded as of haemic origin, and not pathological. In these cases anaemia was present in a more or less marked degree.

Blood.- Most cases of rickets show a certain amount of anemia. The haemoglobin is often diminished to a proportionately greater degree than the corpuscles. I have never yet come across a case of leucocytosis in this disease.

MUSCULAR SYSTEM.

The muscles and ligaments suffer to a considerable extent from the general malnutrition. The muscles are weak, and in many cases this weakness is of such a degree that on various occasions I have been asked by the mother if the child is not actually paralysed. On post-mortem examination they are seen to be badly developed, and in not a few instances they show fatty changes. This weakness on the part of the muscles accounts for the child either being unable to walk until very much

later than usual, or to his "going off his legs" if he has already acquired that function. In some instances the child cannot even sit up, owing to the weakness of the muscles of the back. Accompanying this muscular weakness there is a relaxation of the ligaments. The precise changes which take place in the ligaments are not definitely understood. The relaxation causes looseness at the joints, so that the child is able to assume abnormal positions with apparent ease. I have noticed on several occasions a child quite comfortable and playing with his foot held quite close to the face, the thigh being abnormally flexed on the abdomen. There is in these cases quite an abnormal amount of movement at the head of the femur. Sometimes a distinct lateral movement can be appreciated at the knee-joint.

DIGESTIVE SYSTEM.

The mouth in a great many cases shows small white patchy islands. The tongue, as a rule, is not coated to any unusual degree. It shows, however, red marginated areas devoid of epithelium, which vary at times in their size and number. They heal up and make their appearance again. I do not consider them syphilitic, as Parrot suggested, but as due to defective absorption in that locality and abnormal secretion. They correspond exactly to the erosions near the solitary glands and those of Lieberkühn in the intestine.

The abdomen is usually large, and for various reasons. To mention some - there may be a dilatation of the stomach, flatulent distention of the bowels, enlargement of the liver, spleen or glands, and lastly, debility of the abdominal muscles. They may be all present in any case, but it is comparatively rare to come across more than one at a time.

I have elsewhere stated that catarrhal changes in the stomach and intestines are important and early symptoms in rickets. The child seems hungry, and the mother naturally feeds it lest the neighbours should say that the infant is being starved. I have noticed that these children seem to have an abnormal appetite, never seeming to be satisfied with a meal and fall asleep like healthy children. The result of this constant feeding is that the stomach undergoes a process of dilatation. In twenty cases I have found the stomach line in the region of the umbilicus, and in many others I have seen this quite early in the course of the disease. To my mind it is due to the engorgement of the stomach with more liquid than it could comfortably contain. The intestines are dilated and doubtless from weakness of the muscular coat accompanied by flatulent changes occurring inside. There are erosions of the solitary and Lieberkühn's glands. Catarrh of the mucous membrane is very apt to occur, and this is aided by the irritation produced by the presence of undigested food. The result of these changes is the presence of diarrhoea, the stools being of a peculiar offensive odour and often mixed with undigested food.

Enlargement of the LIVER and SPLEEN is found in a small proportion of cases; so also is enlargement of the LYMPHATIC GLANDS.

ACUTE RICKETS.

Some authors describe cases under this heading cases in which apparently healthy infants suddenly become ill. The bones show very rapid changes of a rickety nature; and this rapidity is so marked that it has led to the terms "multiple epiphysitis" and "multiple periostitis" being applied to it.

Some observers say that it is an acute initial stage of certain stages of rickets; others regard it as an independent disease developed on a basis of constitutional predisposition; while yet another writer thinks that it is nothing more nor less than a pure inflammation of bone.

Whether or not it is a true rachitic condition is a mooted point; but there seems no doubt that changes occur, especially at the ends of long bones, very similar to those found in ordinary rickets.

It is a comparatively rare disease, and during the course of several years' by no means inconsiderable observation I have only seen one case eligible for inclusion in this category. The child, aged four months, took suddenly ill with a temperature ranging from 99.2. to 102.F. It was peevish and fretful, and it cried lustily when touched or disturbed. On very careful examination I could discover no definite lesion to account for the temperature, until, on the second day, my attention was drawn to a slight fulness on the patient's right leg. This seemed to be a tibial periostitis. On further examination I found one wrist and one ankle tender and rather larger than the other. At the end of the week these fulnesses had become quite prominent, and the other wrist and ankle were now affected. The child perspired pretty freely; though the temperature varied, it tended to recede. The state of the general health very soon gave cause for considerable anxiety, it being evident that the patient, instead of thriving, was going down very rapidly. Gastric disturbance was manifested by the occurrence of slight vomiting. A variety of food-stuffs experimented with were barren of beneficial results. At the end of about three weeks the child took a convulsive fit, and from the effects of this death occurred. No post-mortem examination was allowed, so the diagnosis must rest entirely upon the above clinical observation. There was a distinct history of venereal disease on the father's side at the time of the child's death. At first I was inclined to consider the condition a periostitis of syphilitic origin; but on thinking the matter over several times I changed my mind, and concluded that the case was actually one of the so-called acute rickets, the syphilitic paternal taint doubtless predisposing to its occurrence.

FOETAL OR CONGENITAL RICKETS.

There seems a unanimity of opinion that rickets can be present at birth, but not to the extent reported in certain quarters; thus, it is probably an exaggeration to insist, as some have done, that rickets is present in 80% of newborn children.

An actual experience in one thousand cases of ^{new-born children} rickets has not revealed a single instance. I am of the opinion that any abnormality in the osseous system at birth is by enthusiasts put down to foetal rickets, especially if the abnormality in question consists of softening or deformity of the bones. It has at various

times been described as "foetal cretinism" and "achondroplasy". Osler and McCrae quote Ballantyne as having shown different varieties of bone disease occurring in utero, and these, I have little doubt, have all been some time cited as instances of the congenital disease.

The salient feature of the affection, as generally described, is the extraordinary tendency to fractures of the bones. These are not necessarily produced at birth, as cases have been reported in which signs of re-united fractures were present when the child came into the world. There may also be deformity of the bones.

I have come to the conclusion that the condition of the mother is not without influence in its production. If she has come through some serious illness during pregnancy the foetus, I think, has a chance of suffering also, especially if the illness in question took place when ossification of the bones is going on.

Some authorities entertain the hypothesis that the mother's blood is at fault, being deficient in bone-forming material. That may or may not be so, but I have observed on more than one occasion a mother who has suffered from pneumonia or acute tuberculosis, in the last four months of pregnancy bring forth a weak, delicate infant entirely free from the rachitic taint.

LATE RICKETS OR RACHITIS TARDA.

This condition has often been described in recent years, but it still is an open question as to whether or not these are really instances of true rickets similar to the infantile disease. The affection occurs usually in the growing youth any time between the ages of five and seventeen or eighteen years. There may be no special constitutional disturbance, the main thing noticed being a gradually developed deformity of the limbs, especially the legs. The most common deformity is knock-knee. There is an excessive bone formation on the inner aspect of the femur and tibia, with a corresponding deficiency on the outer aspect. This deformity may have been preceded by pains in the legs, which are usually considered by the parents as "growing pains". In some cases changes in the thorax similar to those in ordinary rickets are observed.

According to Osler and McCrae, the bones give a less distinct picture under the X-rays; and this is due, they say, to the want of ossification.

These changes in the bones leading to their softening and yielding have caused the affection in many cases to be confounded with osteomalacia. The latter, however, is a disease of adult life, and more especially adult female life, in which the bones of the pelvis and spine are most readily affected.

DIAGNOSIS.

GENERAL DIAGNOSIS.

It is only in the earlier stages of rickets that there is difficulty in recognising the affection, which, when fully established, presents such typical characteristics that it is comparatively easy to make a positive diagnosis. The changes in the osseous system are so unequivocal that, in conjunction with the other symptoms, no difficulty should be experienced. It is in the early stages and in cases in which the osseous changes are not general, but are confined to one or two bones, that error may be made.

The affection is so very common amongst the lower classes that I am continually on the watch for it. I have found that amongst the earliest suggestive symptoms are: pallor of the integuments; insomnia and restlessness during nights; perspiration, mainly of the head; muscular debility; constipation, beginning between the fifth and eighth weeks of life, often with a placid kind of indolence; prominent frontal tubera; craniotabic spots on the parietal bones mostly (posteriorly), also on the occipital, together with widening of the sutures and patency of the fontanelles; falling out of the hair on the occiput, which feels unduly hot; sensitiveness of the ribs when they are touched, or an outcry when the infant is raised from the cradle; swelling - sometimes painful on pressure - of the epiphyses of the ulna, radius, femur (the two latter, however, are large normally, tibia and fibula, also of the insertion of the ribs; the curvatures of tibia, femur, or arm which have already been described; early bronchial catarrh, particularly when persistent without apparent cause; late or irregular teething; the swelling of the spleen, which may be felt distinctly below the edge of the ribs - which are all, some positive and some probable, expressions of the disease.

It is important that the affection be recognised as early as possible, for it is in these early cases that treatment proves most satisfactory. If the infant be brought under suitable hygienic conditions and properly fed, the disease may be cut short.

DIFFERENTIAL DIAGNOSIS.

POTT'S DISEASE.

When the osseous deformities are limited to the spinal column the diagnosis presents some difficulty. In the case of rickets the curvature affects a considerable portion of the vertebral column and forms a wide angle; further it disappears more or less when the patient is laid on his face. In Pott's disease, however, the curvature is not so extensive and forms a sharp angle. The body of the vertebra is not merely softened, but is actually destroyed - hence the acuteness of the ^{angle} ~~angle~~. There is also weakness of the lower limbs, but this is accompanied by nervous phenomena due to pressure on the cord. These nervous symptoms are not found in rickets. In some cases, however, it is

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almost impossible to effect a positive diagnosis at the first examination, and it is only after repeated future examinations that this can be done.

CONGENITAL HIP-DISLOCATION.

It sometimes happens that rickets is at first sight mistaken for congenital dislocation of the hip, on account of the deformity of the legs, but this affection can be eliminated by finding the head of the femur luxated.

SYPHILIS.

If the deformity is confined to the tibia only, hereditary syphilis must be eliminated from the diagnosis. The syphilitic changes have been graphically described by certain French writers, particularly Lannelongue, in recognition of which the term "Lannelongue tibia" has been applied. This tibia is apparently curved, but in reality is not, but deformed by more or less irregular swellings due to gummatous deposits. The rickety tibia is, however, actually bent in the way already described. A suspicion of syphilis will also be raised by other things. Thus, parental history of venereal disease, mischarrriages in the mother and dead-born premature children; onset not later than the third month; presence of rashes, fissured mouth, coryza, snuffles, cachexia, mucous and anal patches. Syphilis is a bone producer, while rickets is a cartilage producer; the former gives rise to more extensive and diffuse thickening of the lower end of the diaphysis, as well as nodes and gummata. The osseous lesions of syphilis are destructive and lead to separation of the epiphysis and shaft and to the formation of abscesses. The early form of bone disease is an acute epiphysitis, with inflammation of the shaft and soft parts secondarily. The epiphyseal enlargement of the wrist in syphilis attacks usually only one bone, which is quite different from what occurs in rickets. Chronic periostitis is seen in late hereditary syphilis, often attacking the tibia and producing there the characteristic deformity already described. The head in syphilis is sometimes of irregular shape. Osteophytic growth is seen round the anterior fontanelle, which may appear to be in a hollow. The natiform skull is seen, and there may be also craniotabes and nodes which may break down and cause necrosis of the affected part.

HYDROCEPHALUS.

In cases presenting hydrocephalic symptoms some difficulty may be encountered in the absence of other signs of rickets. If the hydrocephalus is present to any marked degree the child, as a rule, very soon exhibits pressure symptoms. If combined with rickets, the configuration of the head and body generally is that of rickets, while the fontanelle is large and elevated; further, if there be much liquid, the sutures are open and fluctuating.

SCURVY.

I have now and then had some difficulty in eliminating scurvy in certain cases. In this affection, however, there is always a great tendency to haemorrhages, especially subperiosteal ones, and very often the gums are spongy and bleed readily.

In conclusion, Holt's dictum may here be appositely cited. "Most important early symptoms for diagnosis", he says, "are sweating of the head, craniotabes, great

restlessness at night, delayed dentition, and enlarged fontanelle. All these, taken separately, may mean something else, but collectively, they can mean nothing more than rickets.

PROGNOSIS.

It is my custom to give a guardedly favourable prognosis in this disease, especially if at all well-marked. Rickets in itself is not to be feared, but the nervous and respiratory complications which sometimes occur, and not infrequently prove fatal. The earlier the age at which the affection makes its appearance the more serious its effects. The course and severity of the malady usually determine its morbidic expressions; and these may last for months or years and in not a few instances lead to permanent deformity and debility.

The ultimate result of the deformity of the limbs depends greatly upon the carrying out of efficient treatment, with the splinting of the affected bones, especially those of the lower extremities. The child must not be allowed to walk. This rule I have on some occasions found most difficult to enforce, the mother emphatically affirming that the creeping could not be kept at rest.

This was the factor which obtained in a case attended by me last year, that of a male child of two years whose legs were badly bowed asunder by the disease. I formulated my rule as above, but the result was anything but satisfactory. Not content with knocking the splint out of place during his bizarre locomotion, he actually pulled at the bandages until he got his legs quite free. Then he commenced to creep and walk about as fancy seized him.

In such cases the deformity is usually permanent, unless rectified by the surgeon later in life; osseous deformities never disappear of their own accord. They may improve to some extent under proper treatment; but it seems to me that the prognosis should be very guarded in view of what may ultimately occur. The best results in the way of a cure of an osseous deformity I have seen have been in the ribs where, under physical culture and gymnastics, the chest has been returned to its normal shape and size, allowing of the proper expansion of the lungs.

In cases in which there is great deformity of the chest accompanied by chronic bronchitis, the prognosis should be guarded also, as these patients are the happy hunting ground for the tubercle bacillus. So long as the bronchial trouble is amenable to treatment and there are no signs of bronchopneumonia, I consider it justifiable to hold out fair hopes of recovery, especially if the patient is showing signs of improving under the treatment. Whenever either of these two complications appear, I consider the case as very serious - the more so if the child is very young and greatly debilitated from any cause.

To sum up, - the outlook in the case of rickets depends on the organs and tissues affected and their, and the slow or rapid course of the development of the symptoms. Spontaneous recovery is not unusual, mainly under the influence of favourable hygienic conditions.

when the bones are mostly affected the epiphyses may remain thickened, and curvatures may be visible through life. But, unless they be very bad, they will partly disappear during the progress of growth. A rachitical pelvis may become dangerous during parturition. When the diagnosis is made early the prognosis is good, even when the anaemia and dystrophy are marked. Symptoms belonging to the nervous system may prove dangerous, a single convulsion may cause permanent cerebral lesions; laryngismus stridulus may be the cause of sudden death; hyperaemia of the skull and meninges may lead to meningeal effusion and hydrocephalus; a rachitical chest to atelectasis, bronchial catarrh, bronchopneumonia, and mediastinal adenitis. Complication with tuberculosis is not uncommon for that very reason. The general health is always suffering through the first months of rickets; that is why every disease, and mainly infectious diseases, run a graver course when attacking rickety children. Nevertheless, with all these possibilities, the general prognosis is rather favourable in the vast majority of instances.

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T R E A T M E N T.

P R O P H Y L A X I S.

The old aphorism, "prevention is better than cure," can be very aptly applied to rickets. A great deal can be done to prevent the appearance of this affection by giving good advice to ignorant mothers. It is my invariable custom to persuade the mother to bring up her infant on the breast, if reasonably possible. Unfortunately, it is not always possible, and probably will never be so long as poor-class mothers have to absent themselves from home to earn a living. In other cases, where the mother is quite willing to nurse her infant, the mammary gland fails to produce the necessary milk.

Long experience has convinced me that, if all infants were breast-fed at proper intervals, there would be a marked diminution in the number of rickety children; if this natural nutrient method were accompanied by hygienic surroundings, the affection would most likely be of comparatively rare occurrence.

A very interesting case which I once attended comes in very well here - that of a young woman, Mrs. S., aged 28 years. She came over from Ireland with a little girl of about nine months. The latter was fed at regular intervals, put to bed every night, at 5.30, in a nice airy room, and not hurried to sleep like other children, but merely coaxed into a sleepy desire and left alone. This case impressed me very much, as I often met the mother during the evenings in the houses of friends; one night I asked her who was looking after the child during her absence, and the reply was: "Oh! nobody; she's in bed asleep and will not waken until about 11 o'clock!" This seemed to be ideal nursery management; and I saw very clearly that the infant was the picture of health. A few months later I was called to see Mrs. S., whose mother had died of diabetes mellitus, and found her suffering from glycosuria - an affection for which I forthwith prescribed the usual remedies. She was a most satisfactory patient, and did all she was told in a very faithful way, with the result that after a few months the sugar had practically disappeared; in fact, it could not be detected in the urine for months at a time. But what concerns my present purpose is the fact that Mrs. S. again became pregnant, and during the last three months of pregnancy sugar appeared in the urine. She was confined, on June 25, 1904, of a male child apparently in good health. The mother insisted on nursing her infant, and this she did in an ideal manner. He was fed at regular intervals, and between meals lay in his cradle in the open air, weather permitting. I thought it highly probable that he might develop rickets owing to the comparatively delicate constitution of his mother, aided by that of a by no means robust father. Instead, he grew into a remarkably strong child; and when last I saw him, in July, 1909, he was the picture of health, notwithstanding that both parents had died,

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when he was a little more than two years old, the mother of diabetes and the father of cancer of the stomach. This case, I think, shows what can be accomplished where a child is predisposed to a weak constitution, and therefore rickets by virtue of this.

My greatest difficulty in BREAST-FEEDING is to make the mother realise that the infant is not necessarily hungry every time that it cries. Whenever it utters a sound, the breast is usually the comforter applied, even though the infant may have had a drink quite recently. This is particularly the case during the night. I always endeavour to convince the mother that the best way to feed the child is ~~every~~ ^{at} two hours during the day until bedtime at 6 or 7 o'clock; then another feed when the parents retire to rest at 10 or 11 o'clock, and another in the middle of the night at 2 or 3 a. m., making in all about ten meals per diem. If this plan is carried out properly from the time of birth, the infant will thrive and, what is more satisfactory, will learn by a natural instinct when a meal is due. Then, and usually only then, will the infant become uneasy and cross.

Another point in connection with breast-feeding which must be borne in mind is the necessity for keeping the mother's milk up to proper standard. This in some cases is difficult, as amongst the poorer classes many of the mothers are unable to procure proper food, and sufficient of it to keep herself in health and rear a child. She has to go with what she can get, and this at the expense of the child's milk, the result in not a few instances being that rickets appears.

After the first three months the time between meals should be ~~gradually extended~~ ^{at least} ~~half~~ ^{about} the sixth month there should be at least two and a half hours between. About this time I usually begin to prepare the child for weaning. If the mother is a big strong woman with plenty of milk, she may go on nursing for months without showing any injury to herself or her infant; but nonetheless I advise ~~her~~ to give the child some infant food, at first once a day, and then morning and evening. A favourite of mine is a little thin gruel made with Scott's or Jack's oatmeal-flour; later on a little beef-tea may be given in the middle of the day. Under this system of gradual weaning I have found that the child is not nearly so upset as when it is suddenly taken off the breast and some artificial food substituted. In any case the child should be entirely done with the breast when it has completed the first year of its existence.

As to HYGIENIC CONDITIONS, whenever possible I insist on the baby being a certain time in the open air daily, and that when indoors it should lie in a well-ventilated room. It should be kept scrupulously clean in every respect, it being bathed morning and evening and to have a change of napkins after each meal. This I have found careless mothers not infrequently neglect, and with unfortunate results. The child's clothing should be light, warm, and not too tightly applied, especially over the chest.

If BOTTLE-FEEDING it is necessary to see that the bottle has no long indiarubber tube, and that it be kept perfectly clean. The question as to what to put into the bottle is an important and mooted one. During recent

years I have derived very pleasing results from a mixture of one part each milk, barley-water, and water, with cream added. I commence with equal parts for an infant at birth and gradually reduce the proportion of water until about the end of the second month the proportions of milk and barley-water are equal. By the time the child has reached the age of six months, I give two parts milk and one part barley-water, and at the end of ~~th-~~ a year pure milk. I add to each feed half a teaspoonful of cream at first, and gradually increase it as the child ages. A mistake often made is to make the barley-water too strong, a large teaspoonful of barley in a pint of water, boiled for fifteen minutes, a little salt added, and then strained through muslin, being quite sufficient.

The total quantity of the mixture to be given at each feed should be duly arranged. I usually advise two ounces for a start every two hours, and gradually increase it to six ounces, provided there is no regurgitation after feeding and no signs of undigested ~~food~~ in the stools. If the child steadily thrives, sleeps well, and appears to be comfortable, nothing more can be desired.

It is when the child has reached the age when it is able to sit upon the mother's knees that damage commonly takes place; and this holds good in both breast- and bottle-feeding. What I allude to is the unfortunate practice of some parents, and in my experience the father is the more culpable of the two, of giving the infant sips of tea, etc., pieces of bread and other starchy foods, when at their own meals. One of the most difficult everyday endeavours is to convince some mothers that milk is the food, ~~and~~ that it should be the principal food for the child during the first two years of its existence. The mother seems never satisfied unless her infant is eating something

The hygienic principles governing breast-feeding hold good in bottle-feeding, perhaps even more so. When symptoms of rickets have made their appearance, the first part of the treatment is to correct the mistake, or mistakes, which have been made. These are usually found to be either dietetic or hygienic. Whenever I diagnose a case of rickets I inquire about the feeding. If the child is on the breast, I try to find out how often the infant is fed during the daytime and during the night. If it is being fed at irregular times during the day, I advise the mother to give a meal every two to three hours according to age, and whether awake, and only to feed for ten or fifteen minutes at a time. A very common error is the frequent feeding during the night. The infant should only be fed twice at night - once when the mother goes to bed, and once in the early morning. This makes about ten feeds per day for the young infant, and as it gets older the number of feeds should gradually be reduced by increasing the time between each meal.

If there is no error in the method of feeding, then I look to the mother for some cause which might impoverish her milk. If menstruation has been re-established, I advise that the child should be weaned as soon as possible. I at once prescribe some artificial food for administration, according to the age of the child, once or twice daily, with gradual increase until

weaning is accomplished in two or three weeks. If the mother is not securing her proper sleep at night, the matter will require proper investigation and correction: otherwise weaning is inevitable. The same holds good if she is not getting sufficient nourishment to maintain good health and at the same time provide her infant with a satisfactory milk. Should she become pregnant whilst suckling an infant, the latter should be weaned at the earliest possible date.

The question, then, of bottle-feeding is an important one, particularly as bearing upon the disease under consideration. I have used an infinity of ARTIFICIAL FOODS, and during the time I have been in practice I have seen good and bad and indifferent result in connection with them all. The great fault in the majority of patent foods is the presence of starch to a greater or less extent, some having as high a percentage of starch as 40, as in Nestlé's Milk, while in human and cow's milk there is none. I have therefore come to the conclusion that COW'S MILK, properly treated, is the best substitute for mother's milk. The main difference between the two is the small amount of fat in the former and a higher percentage of casein. The fat-deficiency can, however, be remedied by the addition of cream or oil.

Some writers advise the use of tall vessels to contain the milk, and after the settling of the cream to use the upper half or third of the liquid.

Whenever possible I advocate the employment of pasteurised milk, but amongst the poorer classes this is usually impossible of achievement. In these cases I have tried two different plans. One is to have a bottle into which good cow's milk, together with a pinch of bicarbonate of soda to correct the acidity of the liquid, is put, a plug of cottonwool being inserted as a cork. This is placed in a pan of water, which is slowly brought to the boil and kept slowly boiling for fifteen minutes. I have found that this simple procedure was in many cases badly carried out, owing to the labour involved.

During the last two years I have tried milk which has been heated in a clean pan, if possible specially reserved for the purpose, until the glossy skin forms on the surface. This is thrown away, and the remainder administered in the usual way after the addition of a little bicarbonate of soda.

As already stated, cow's milk is deficient in fats as compared with mother's milk. Human milk, when up to the proper standard, should contain 21% to 23% of fats, while cow's milk may only contain 3% or 4%. This latter may even vary more according to the general condition of the cow and feeding.

Some people run away with the idea that only one cow's milk should be used for feeding infants. My own preference is, however, for a mixture of several milks, because, it seems to me, there is not so much chance of there being such variations in the quality of the milk as ~~when~~ the lacteal secretion of one animal alone is used.

The sugar in human milk is usually over 40%, as compared with 4% in cow's milk. This may be remedied by the addition of milk-sugar; but the only cases which have ~~come~~ under my observation were those in which the

infant was being fed on condensed milk, which, as is well known, contains a large percentage of cane-sugar.

Having now obtained the milk ready for administration, the question arises as to how it is to be used. This depends upon the age of the patient. If about six months old, I commence by giving it about equal parts with barley-water. Just at first I prefer the milk to be rather less than the barley-water. If this agrees with the child, and there is no vomiting after two days or so, I give equal parts and add half a teaspoonful of cream, gradually increased to a teaspoonful, each feed at this age being at first four and a half ounces, gradually increased to six in the absence of regurgitation after meals. In younger children the proportion of barley-water to milk is increased, and vice versa for older ones, until at the age of one year the infant can usually thrive on the milk without any dilution.

If the parents have not the means to obtain cream in such quantities, I prescribe a teaspoonful of pure cod-liver oil for administration twice a day after a meal. If this does not upset the stomach, I increase the oil to three times a day. It is often advantageous to rub a teaspoonful of the oil into the infant's abdomen, in addition to administering it internally. Some writers report results from suet or butter in the milk as substitutes for cream. I have had no experience of them.

Sometimes vomiting has been such a persistent symptoms that I have had to use predigested milk before the stomach would tolerate nourishment, Fairchild's peptonising powders being used. The milk is heated (not boiled), then the powder is added, and the temperature kept at a point below the boiling for twenty minutes, the peptonising action being now stopped by bringing the milk to the boiling point. When sufficiently cooled, the milk is ready for use, and is given to the infant in quantities according to age. I have seen it very effective in cases in which the child's stomach was so irritable that it immediately rejected milk in any other form.

I have derived very satisfactory results from the use of RAW BEEF-JUICE and VIROL, which can be given very easily by dipping the point of the bottle or comforter into the preparation and allowing the child to suck it.

HYGIENIC TREATMENT is of great importance, but in many cases most difficult to carry out. Some of the lower classes in our city population live under most insanitary conditions, mostly due to their own carelessness and slovenly methods of household management. I can describe some of the rooms I have seen in Edinburgh as nothing less than absolutely filthy. Treating cases of rickets and other affections under such circumstances has baffled me not a little, as the parents cannot, or will not try to, keep their apartments as clean as possible. The room in which a rachitic child is kept should have plenty of light and sunshine; also, it should be kept at an even temperature of about 68 degrees. This, however, should not be accomplished at the expense of fresh air. The air in the room should be kept fresh, the apartment being well ventilated and the temperature regulated by means of a fire. Unless the patient is troubled with acute constipation, it should

be taken out for a couple of hours in the middle of the day, weather permitting of course. The child's clothing should be light and warm. Woollen garment should be worn next the skin - light in summer and heavier in winter.

If the child suffers much at night from sweating, I always advise the use of two sets of undergarments - one to be used during the day and the other at night. The object of this is to have the damp sweat-soaked clothes thoroughly dried and aired before use; in this way the risk of a chill is obviated.

The baby should be kept clean by a bath morning and evening; but in some cases where it is debilitated a sponge-bath is to be preferred to immersion. The bath should be of short duration, and practiced just enough to insure perfect cleanliness.

After the child has been dressed, and just before applying its napkins, it is a good plan to warm its feet and legs at the fire and by gentle friction until there is a good circulation; this acts as a mild counter-irritant, relieving to some extent the hyperaemia present in the bone. The child who suffers from cold feet is never in perfect health, and measures should always be instituted to remedy this - e.g., by flannel-wrapping, artificial heat, and massage.

T H E R A P E U T I C S .

Prolonged observation has convinced me of the fact that ordinary uncomplicated cases of rickets require no medicinal treatment other than the administration of fats and cod-liver oil.

Some authorities, more especially Kassowitz, advocate the prescription of PHOSPHORUS in one or other of its several forms; but the objection I have to the satisfactory cases he reported is that the phosphorus was given in combination with cod-liver oil. This, in my opinion, does not prove such a good case for the beneficial effect of the phosphorus, seeing that the generally accepted as itself thoroughly reliable and productive of, in the most instances, as good results as could reasonably be desired.

This, I think, is proved by the experience of H. H. Purdy, who says: "The resumé of the results of treatment is as follows: Some were given cod-liver oil alone, some oil with phosphorus, and other phosphorus alone, and of course all the mothers were given instructions in feeding and hygiene. The infants that received only phosphorus were slowest to improve. Indeed, in several cases this method of treatment was abandoned because of the absence of signs of improvement. The group treated with cod-liver oil did best. In fact, all the infants that could tolerate the oil derived much benefit from it. The group that were given cod-liver oil with phosphorus did very well, but seemed no better than those that were given only cod-liver oil. This, I think, goes to prove my contention."

When there is any degree of anaemia, IRON, in one or other of its numerous forms, proves very satisfactory. I usually prescribe the compound syrup of the phosphates of iron (Parrish's Syrup), in doses ranging from ten to thirty minims, in combination with cod-liver oil, or in milk when the child is under twelve months of age.

In the case of older children I very often use⁴⁵ the syrup of the iodide of iron, in half-drachm doses, with or after a meal.

I very commonly prescribe the emulsion of cod-liver oil as sold by our local chemists, containing 50% to 60% of the oil, the hypophosphates, eggs, sherry, and syrup. This has a rather pleasant taste, and the child in many cases takes it quite readily when it would undoubtedly refuse the pure oil.

As nervine and muscular tonics STRYCHNINE is useful in doses of a half to one minim, according to age, of the officinal solution or the tincture of nux vomica.

Stölzner has used SUPRARENAL EXTRACT with good results, and he therefore is inclined to think that the disease actually arises from some suprarenal insufficiency.

ADRENALIN solution (a half to one minim in a teaspoonful of water) has been recommended, three or four times a day on an empty stomach, by Ewart, who says that it is not without effect on the muscular asthenia, which commonly exists in this disease.

Von Mettenheimer has used extract of the THYMUS gland; though good results were reported, his observations have not been confirmed.

Eustace Smith has noted improvement from the use of TANNIN, given in doses of from a half to one grain two or three times a day in dilute nitric acid.

MALT is sometimes useful as a digestive, tonic medicament.

SYMPTOMATIC TREATMENT.

The various symptoms and complications which may from time to time occur in rickets should be treated according to indications and the following general plan:

HEAD-SWEATING.

This is best controlled by applying the liniment of belladonna locally to the head at night, or by giving the tincture of the drug internally. Children tolerate this remedy very well. Twenty drops can be given in one dose at bedtime to a child a year old; or ten minims may be administered three times a day instead. Another good plan is to exhibit one minim of the atropine solution at night. Phosphorus is said to benefit these cases, and the author already mentioned says the same of suprarenal extract.

DIGESTIVE DISTURBANCES.

Not a few cases of rickets have pasty and most offensive stools. In these cases the bowel should be cleared out. A small dose of calomel may be given at night and a teaspoonful of castor oil in the morning, the powder being placed on the tongue and washed down with a little milk or water. Failure to operate can usually be accounted for by the child spitting it out, and in these cases chocolate-coated tablets or other attractive proprietary preparations may be used. Some prefer to give daily fractional doses of gray powder, preferably at night, and the usual castor oil in the morning; further stomachic treatment is then said to be unnecessary. Small doses of rhubarb and soda may be employed if preferred. Having thus purified the alimentary canal, it is advisable to administer an alkali, on account of the

catarrhal condition, and to check the undue peristalsis whereby the food is hurried through the bowels undigested. A small dose of the tincture of opium and some carminative may be added to, for instance, a mixture of bicarbonate of soda, aromatic spirits of ammonia, and chloroform-water. In a few days this treatment will cause the stools to assume a normal character; the tongue will clean, and flatulence will disappear. Then tonics and cod-liver oil should be given. In the treatment of gastrointestinal derangements some writers advise the use of such intestinal antiseptics as salol, urotropin, and naphthalin. Doubtless these agents hinder putrefactive changes, but I have never found it necessary to depart from the method described. The daily evacuation of the bowels is most essential. Sometimes one to three minims of dilute nitrohydrochloric acid, with a little pepsin and nux vomica, is useful, especially in conjunction with some rhubarb preparation. Another good laxative is the carbonate of magnesia, of which a few grains may be added to the bottle. A good tonic is that recommended by Ashby, namely, the aromatic syrup of cascara. From five to fifteen drops may be given three times a day, and continued indefinitely. Some cases require a glycerine and oil enema, but this form of treatment is seldom employed and is not to be recommended as a routine plan.

RESPIRATORY AFFECTIONS.

The stimulating plan of treatment with iron, etc., goes good in these cases; and in not a few instances of acute bronchial catarrh I have seen benefit arise from the inhalation of moist air, a tent being erected around the child's cradle and a steaming kettle placed inside. The patient should be kept in one room at a temperature of, say, 70.F. The condition of the bowels should, of course, receive the usual attention. Counterirritation should be applied to the chest in the form of a weak mustard poultice, that is, one part of mustard to five or six parts of linseed-meal, mixed dry and made with hot water. The idea is to act slowly on a large surface. This should be kept on for perhaps six hours, and then be replaced by wool. The back of the chest should be similarly treated. Meantime the child should be taking the following mixture for the cough:

R/

Vin. Ipecac., ʒi.
Tinct. Camph. Co., ʒiii.
Syrup. Tolu., ʒiii.
Aq. ad ʒiv.

Sig.- One teaspoonful every four hours.

Half the prescribed quantity should be given to a child of four months, that amount being supplemented by water so as to fill the spoon.

As soon as the cough becomes looser and not so hard, stimulating expectorants are useful, such as ammonium carbonate with tincture of squills

The bronchial ~~catarrhal~~ condition may culminate in bronchopneumonia and pulmonary collapse; and then vigorous measures should be employed. Counterirritation is indicated, and the temperature must be watched. Tepid sponging is the best way to reduce it, at least in private practice where the parents object to wet-packs

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and cold baths. Stimulants are necessary from the first, and in the form of whisky or brandy, or brandy and egg mixtures, or even whey. An infant a year old will take a half to two ounces daily of whisky or brandy. The dose must be regulated according to the condition of the pulse; should it be weak, compressible, rapid or irregular, stimulants are indicated, and also if there is pallor or lividity. They are most useful at the commencement of the disease, if the temperature suddenly falls, or if there are any signs of collapse of the lung. My usual custom is to give the stimulant every two or three hours. Sometimes nitroglycerine is useful in heart failure with pulmonary congestion, and in doses of $1/500$ of a grain every hour until the urgent symptoms are relieved. Should actual collapse of the lung occur, an injection of strychnine, $1/300$ of a grain every three hours for a child of twelve months, should be given; as well as a hot mustard bath and stimulants. Eustace Smith says that if the child cannot swallow, it should be introduced into the mouth by means of a glass syringe with an elastic tube attached. Caffeine (gr. $1/20$) and atropine (gr. $1/800$) are also useful respiratory stimulants in these cases. Inhalations of oxygen are serviceable, as well as gentle friction of the chest wall. An emetic often does good, at the beginning of the attack, by clearing the tubes. A teaspoonful of ipecacuanha wine, repeated if necessary, will meet this indication. Stimulants expectorants do good. During convalescence such tonics as quinine, iron, cod-liver oil should be employed, and hygiene and nutrition require the usual supervision.

NERVOUS SYSTEM.

If the child is cutting its teeth and showing symptoms of nerve troubles, such as head-rolling or face-twitching, the exhibition of the bromides is indicated. The bowels also require attention, and I usually prescribe calomel and bicarbonate of soda every other morning to overcome the constipation which almost invariably exists. The gums may require lancing in these cases. During the convulsions it is advisable to put the child into a hot bath and pour cold water on the head.

Tetany is treated on much the same lines, and a stimulating liniment seldom fails to do good. Tonics and hygienic regulations are strongly indicated also.

Laryngismus Stridulus can usually be cut short by sponging with cold water, the temperature of this being raised to tepidity during the winter months. Fresh air is also most important, and the patient should be as much as possible out of doors. Antispasmodic drugs are useful, such as belladonna and musk; the former may be given in doses of a third of a grain to a child of twelve months every six hours, and the latter in fifteen minims of the tincture three times a day at the same age.

For hydrocephalus no treatment is entirely satisfactory. Incision, puncture, aspiration, strapping, blisters, and inunctions of mercury have all been tried without benefit: the fluid simply accumulates again. Hensch recommends local blood-letting, by means of leeches behind the ears, iced compresses, and such purgatives as colomel in $\frac{1}{4}$ - to 1-grain doses several

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times a day. To absorb the fluid he prescribes small doses of mercury, inunction with blue ointment, painting with iodoform collodion, and the prolonged exhibition of iodide of potassium.

CONVALESCENCE.

After the child has sufficiently recovered from the acute symptoms, and when circumstances will allow, great benefit is derived from a lengthy sojourn in the country or, better still, at the seaside. Here, if the child is able to walk, it may be allowed to paddle in the water; but if a cripple or not able to walk, the mother should dangle its legs in the sea.

SURGICAL TREATMENT OF THE DEFORMITIES.

Deformities of the bones should be prevented as far as possible. Children predisposed to them should not be allowed to walk, but be kept at rest as much as possible on a soft mattress with a soft pillow for the head.

If deformity, however, has already taken place, attempts must be made to correct or at least to keep matters from becoming worse. I have found that here forcible reductions in the curvature in the soft bones are useless, for relapses ~~in~~variable follow. In cases of urgent necessity splints may be used until medical treatment has had its effect. Total fractures of rachitic bones are, according to my experience, rare. Infracture (subperiosteal, greenstick fracture) requires splints until the bone will have had time to get normally hard. When the tendency to it is very marked, immobilisation of the entire body may become necessary. The pigeon-breast, which has a tendency to remain for life, requires the earliest possible medicinal and hygienic interference and gymnastic expansion of the lungs. Even crying is welcome, and in children of two or three years trumpet-blowing and soap-bubbling should be encouraged. The curvatures of the diaphyses are less marked than in the adult, because of the extension which takes place during growth. This clinical experience has been amply verified by the close observations continued through years in various clinics. If splints are to do good at all, they should be applied before the bones become hard and resist every degree of reasonable force exercised in mechanical straightening. The tendency to flat-foot, acquired through the flabbiness of the ligamentous apparatus during the attempt at walking, requires straightening of the arch of the foot by a moderate spring and a support for the ankle; scoliosis of growing children of more than six or eight years, Sayre's plaster-of-Paris or a felt jacket; ugly and determined curvatures of the long bones, either osteoclasy (fracture of the curved bone, while the periosteum is mostly left intact, and resetting), or osteotomy (straightening of the bone after a cutting operation). Of these two, osteoclasy was the only operation resorted to formerly. The fracture of the bones was either manual or instrumental, mostly successful in the middle of the femur or tibia, mostly unsuccessful for genu valgum or varum, inasmuch as it not infrequently tore off the epiphyses or broke the bone in an undesirable place, and was not rarely followed by septicaemia and the death of the patient. Consequently, osteotomy has mostly replaced osteoclasy, as it is a simple and an open operation. It is seldom

required on the upper extremity, mostly on the lower, not so often on the thigh, as for genu valgum, varum, or curvatures of the diaphyses. The genu valgum of children results from the curvature of both the femur - usually the only one at fault in adolescents - and of the tibia. It requires the supracondyloid operation of Macewen, and often a supplementary operation on the tibia. The curvature of the tibia has mostly its convexity interiorly and posteriorly, and is usually found at the lower half. The operation may be either simply linear (transverse or oblique) or cuneiform. In bad cases the latter wedge-shaped operation is preferred, and not seldom a single operation is insufficient. After an interval of a few weeks repetition of the operations may be done. These operated cases invariably do well. Osteotomy has proved strikingly successful since its introduction; indeed, it is one of the happiest achievements of modern surgical methods.

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