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R I C K E T S

P r o l o g u eI. GENERAL REFLECTIONS ON THE DISEASE.

The general trend of modern medical research has been to substitute intimate laboratory and clinical investigation for the more or less theoretical ruminations of former days. Practical aids to the study of maladies in all their protean manifestations have come into a popular vogue everywhere, and it seems possible, indeed highly probable that, ere long, there will be few, if any, affections which will not be properly understood. In Australia, and New Zealand, investigations are just as common as in other parts of the British Empire - not only in the towns, but in comparatively isolated parts as well. This is largely due to the convenience of "outfits" which so many manufacturing houses provide, and to the postal facilities for the speedy and punctual spread of literature to the remotest climes. Thus our Art, like some great, but beneficent, monster of allegory has come to stretch its arms throughout the world and so tackled the gargantuan machinations of disease as civilisation and explorations have penetrated. In the part of the world where I reside there are schools where tropical affections are studied, also in Australia and elsewhere; and under their salutary auspices much good is being done, social and climatological factors are assuming the importance they deserve. From the Homeland in Europe expeditionary enthusiasts have gone forth to cope with and, if possible, destroy the causes of such warm-country ailments as sleeping sickness, beriberi, and yellow fever, as well as the like destructive diseases hitherto imperfectly understood - on the evolution of which new light is being shed from day to day. The advantages of such investigations are known to all and the workers in this great field of inquiry widely applauded, as well they deserve to be. But, alas! there still remain in our very midst affections whose names are unfavourably known in every home, taking as they do their toll of infant lives from day to day. Rickets is prominent in this respect; and until some sort of effective and well directed crusade is inaugurated throughout the population, until the people awake to the significance of the awful wastage of the future race, and become aware of the deformity and physical enfeeblement of their children, such curses of civilisation as rickets are sure to remain to do their worst. New Zealand is rapidly becoming peopled with hordes of weekly emigrants from Home; and it only requires time for this beautiful island to become another Britain, with perhaps all its social ills. Both in this country and at Home my practice has been and is largely devoted to the prevention and cure of disease amongst the industrial classes; so that I have had innumerable opportunities of observing rickets in practically every form that it can take. My first impression of it still remains, viz., that of a very frequent disease accompanied by profound general and special effects. Its causes, both in the theoretical and practical aspects, have long occupied my reflective and working hours, with results that will presently be described. Competent observers everywhere are agreed as to the great importance of rickets, and with them, indeed, I would urge that it behoves every medical practitioner to make himself well acquainted with it, and to bear in mind that its influence upon the mortality of little children is enormous, as well as the fact that it must enter largely in the prognosis of an infinity of affections which attack the very young. The tyro in pediatrics is apt, in the hastiness of his impressions, to conclude that rickets is essentially a deformity of the bones producer and in the sense that osseous anomalies assume a great prominence in typical cases, he is not in error. But the bone troubles are usually of late evolution, though it is on their account that children are brought under observation by their alarmed parents. Yet, there are many cases in which

the mother calls in the family medical attendant for the relief of some stomach or intestinal complaint, quite unsuspecting of the underlying and responsible rachitic taint. In view of the frequency of this disease and the innumerable opportunities for close observation which it affords, it is difficult to account for the fact that so few have recourse to practical experimentation. Such line of research not infrequently requires a sort of aptness and special training; but the labour is well repaid, it trains the observation faculties, which, once blunted or tainted by carelessness, are apt to have an unfavourable and faulty influence upon the early and much-desired detection of the disease; the prognosis must become faulty as well. Such an affection as pulmonary phthisis is admittedly amenable to a certain line of special treatment, provided the disease is detected in its comparatively early stage; the same is true of rickets - the earlier the detection the better the results of medical aid. To some minds it might appear labouring the question by comparing phthisis pulmonalis to rickets - the former a terrible disease, the latter one of the easiest of affections to control. Yet the destruction of young children by phthisis is no greater than the like effect of well established rickets; furthermore, as both tend, under suitable conditions, to terminate in recovery, the comparison is quite in order. Clinical experience everywhere will bear this out; so does pathological investigation. It seems to me, then, that a high proficiency in the study of the diseases of children is absolutely essential to every conscientious practitioner: a dictum which gains in emphasis when it is remembered that about 33 per cent. of one's practice lies amongst the juvenile members of the community. Probably a much higher percentage might be allowed, and some writers would raise it to at least 50. From one's proficiency in the treatment of children's ailments the general public are apt to judge one's "cleverness" as a practitioner. The mother with a sick child is the keenest of all critics perhaps; so that one's success in professional life may depend upon attention to the little details and ~~sustaining~~ trifles which one is so apt to overlook or ignore.

Though one is apt to regard this affection as a disease of children and include it in our nosological interpretation as such, it is only apparently so to a certain extent; for it leaves many unwelcome legacies to adolescence, which are apt to influence grown-up ailments to a serious degree. A close study of the literature will convince anyone of this; and such study, in combination with well-directed clinical observation should be one of the most interesting and fascinating branches of our Art, especially if combined with laboratory or other experimental work. To the value of experiment I have already alluded, and must admit that, in its full sense, it is, as a rule, beyond the competence or opportunities of the general practitioner. Yet, on a minor scale, it can be engaged in by all, and it has often surprised me what great advantages are forthcoming from experimentation on even the most modest scale. In the case of rickets, also, we can learn many a valuable lesson from the study of Nature herself. We live in an age of social reform and one in which the disease under consideration is a subject of considerable interest and great importance; one also which covers the vast field of healthier homes, improved sanitary conditions, more wholesome food and dietetic articles of better quality - in short, the constitution of an ideal population. On all hands we find evidence of an increased public interest in the housing of the people, of growing knowledge of the dangers of suckling mothers working in factories and mills, and a conviction of the necessity for the proper feeding of their children, especially in the earlier months of their existence, as well as a general desire for the regular and efficient medical inspection of school populations, the provision of meals for badly fed children attending the public elementary schools, and the formation of local and central nursing and other similar associations of popular and practical medical instruction and assistance. It is now generally recognised that rickets is amazingly widespread distribution, being encountered in the children of the slums, as well as amongst those living in the country; it is met with in the luxurious

nurseries of the affluent, being there due to either ignorance or carelessness or indifference. Though of comparatively modern understanding, rickets is a distinct affection. Its protean machinations are evident from the vast literature which has accumulated regarding it, in which mention is from the first made of such classical conditions as deformities of the osseous system, gastrointestinal disturbances, delayed dentition, weakness of muscles and ligaments, and disorders of the nervous system.

In view of its vagaries and clinical disguises, there is all the more reason for a thorough understanding of rickets. True, it seldom of itself proves fatal; but it is, nevertheless, a very serious menace to infantile existence; and, in its more serious forms, is apt to have far-reaching effects upon the system, such as stunted growth and twisted bones, as well as the too common so-called rickety pelvis, one of the most common causes of difficult labour or even fatality in labour cases. The exodus of the population of rural parts to the towns is a factor which must be taken into consideration in any account of the disease. Such increasing desertion of the country is not to be wondered at when we consider that in man lives the spirit of adventure and of disgust of monotony, which in some is dormant, but in others active. Such persons become discontented with their peaceful and monotonous surroundings and sooner or later make for the cities and towns or go abroad. Some abandon a rural existence and remove to disease-producing cities in resentment over their cottage hovels, houses erected perhaps nearly a century ago, defective in both comfort and decency, and perhaps dark, unventilated, damp and dismal and depressing. I have seen houses (sic) in various parts of rural Great Britain in which I would not keep either my horses or my dogs; no wonder, then, that we in New Zealand are receiving such great accessions to our number. A few years ago I happened to read the speech of a great medical reformer, in the course of which he touched upon the relation between his profession and the State and offered the people a cure for many of their ills. He proposed the establishment of a general staff of medicine "to", as he expressed it, "rebuke the purblind and inveterate habit of our countrymen of devoting their magnificent energy and their treasure to mopping up effects in disregard of causes. We pour our money out like water in order to found sanatoriums for consumption, asylums for the insane, prisons for the criminal, yet are moved with the greatest difficulty to subscribe to the agencies which study to prevent those evils." He adds that "at present it is nobody's business to educate the public in those rules of life and living which are the springs of national efficiency". He would also have it that the functions of medicine have been too long "neglected by the unidea'd governing classes", and insists that a ministry of public health can alone deal with the work to which he refers. "This immense and beneficial work would include," he says, "the protection of infant life; the medical inspection of school children; the physical and mental conditions of education; the conditions of labour, the dynamics of food and the minimum wage; factory inspection, with estimates of the effects of different trades upon health, and collateral problems of compensation; housing, ventilation and sanitation; food markets and adulteration; epidemic diseases; the working of the poor law; the campaign against drunkenness, fornication and other social vices; criminology and punishment, and the antiquated and distrusted dogmas of the judges of the higher courts on responsibility before the law." The importance of the subject was recognised by some of the writers of sixty-five years ago, when one of the most interesting of their number stated that it has been imagined that rickets is a disease peculiar to England, despite the fact that the statistics of the continental authors and the number of squalid, rickety forms seen running in the streets of foreign towns and elsewhere show that the populations of other countries do not entirely escape this dreadful disease. It is as true nowadays as in former times that if we search for the causes by which the affection is propagated we shall find that it most often occurs among persons living in low, dark, damp, filthy cellars and ill-ventilated and overcrowded dwellings, such as may be found in many parts of the cities and large

towns, where they are not only badly fed and poorly clad, but are also more or less bereft of the enjoyment of a proper amount of light and fresh air. When a great number of persons live in such malevolent circumstances as these, it is not surprising that their constitutions should be subject to various derangements - one of the principal amongst them being rickets. Cases of this kind, however, as I have already noted, occur in the families of the rich, who are exempt from the handicaps of life just mentioned; and, therefore, the opportunity occurs for a wide field of research. The exact nature of the external conditions and internal predisposing conditions necessary to engender this specific form of disease constitutes a problem for the workers of the future to entirely unravel: to which which they will have to face an intimate and practical research.

One of the most burning questions of present-day civilisation is the problem of the frightful mortality amongst infants under one year of age, which toll of precious human life goes on year by year almost unchecked and undiminished by the improved conditions of living introduced by medical and other reformers. The adult death-rate has been reduced by scientific and preventive medicine, which have scarcely affected the death-rate amongst the members of the community under discussion. There is no getting away from the fact that, though the general death-rate is decreasing, the infantile one is not. In England and Wales, in 1905, the death-rate among infants was large; there were one hundred and twenty thousand deaths, which number equalled a quarter of all the death in that year. This just about corresponds to the entire population of a very important town in any important country. Just imagine the world-wide sensation there would be if such a place were wiped out by disaster or decimated by a widespread disease or epidemic causing the above number of deaths! In other words, bring this appalling, but distributed, loss of life to a local focus, and how public feeling would be aroused; the newspapers would bring out special editions; a commission would be formed; every effort would be made to discover the cause; and steps would be taken, if possible to avert a similar disaster in future. And yet, because this loss of life is spread over a wider area of space and time, it has been allowed to continue for over fifty years, sapping the nation's strength amongst the newborn and depriving it of a population which would be useful to it in future generations. In all branches of medical and general science there have been wonderful achievements during the last fifty years or so; but when we come to realise that the death-rate amongst infants is almost stationary, surely the hardest heart must be touched. It is difficult to understand how such a lamentable state of affairs has been allowed to continue, in the face of beneficent enactments which have vastly diminished the adult rate of death. Must the nation's newborn strength continue to be sapped unchecked? There are a number of tables published which give interesting particulars in this direction. One of the best known of such official tabulations gives figures in three columns, the first of these being the birth-rate per 1000, the second the death-rate per 1000, the third the infantile mortality per 1000. During the various decennial periods the figures were as follows: 1851-60, - 34.1, 22.2, & 154; 1861-70, - 35.2, 22.5, & 154; 1871-80, - 35.4, 21.4, & 149; 1881-90, - 32.5, 19.1, & 142; 1891-1900, - 29.9, 18.2, & 154; 1901-05, - 28.4, 16.0, & 138. It is a well-known fact that the birth-rate has also been decreasing, - to the extent of four births less amongst every thousand persons, - so that it is even more urgent and necessary that we should endeavour to safeguard and, if possible, help infants through early life in order that they may grow up healthy adults and useful members of the community. Statisticians are also well aware that the largest infantile death-rate occurs in the first three months of life; and that many of the surviving children who are tided over this critical period grow up weakly, immature and prone to disease, and therefore scarcely able to withstand the the morbidic storms of the first year of their existence. During the remaining nine months of the first year

of life it is possible and common to trace numerous affections to the carelessness and ignorance of the parents, who expose their children to cold and pay little attention to their proper feeding: so that the majority of deaths can be attributed to gastrointestinal disorders (such as diarrhoea), disease of the respiratory system, atrophy, malnutrition, scurvy, marasmus, rickets and convulsions. If the child should survive, it may be left weak and puny; and, though in the early months of life it is not possible always to make an accurate diagnosis, the initial illness in reality may have been the forerunner or the first indefinite symptoms of rickets, which, if not treated, progresses and still further weakens the constitution of the child, distorts its limbs and leaves it sadly handicapped in its future struggle for existence.

Though rickets is of great danger to the infant during the period just mentioned and during its growing days, in later life it may lead to evil effects, even if the affection has apparently taken its departure from the system; in other words, though all these children do not die, many of them grow up to be weaklings and have their vitality endangered by the relics of their infantile years. It is later on in life that rickets, antedating perhaps to the time of nursing or birth, especially makes its presence felt, particularly as regards permanent shortening of the limbs, with deformity of the skeleton and distortion of the limbs, so that the child is dwarfed in stature and stunted in its growth. The permanent teeth may suffer equally with those of the first dentition, leading to early decay, malformation of the jaws and various disorders of digestion. The presence of bowlegs, knock-knee and flat-foot may interfere considerably with locomotion and seriously handicap the individual in competitions for various appointments, or prevent him obtaining employment in the mills or factories of his town. Not a few of these children make slow mental progress and show signs of mental enfeeblement, and sometimes are so stupid that they are unable to take full advantage of the educational facilities offered them. So it comes that the presence of rickets should be watched for from the earliest days of life, and all possible be done to eradicate such a dangerous taint from the affected system.

But we must not forget that it is not only the child which must be considered and watched, but also the pregnant woman, as she also requires careful supervision. In her case the dangers of childbearing are much increased by rickets, with collateral dangers to her unborn babe; even after delivery it is in the next generation possible to detect the evil influence of the malady under consideration. Numerous authors have in the past insisted that rickets can be transmitted from parent to child; so that, out of respect for their authoritative position, if hereditary or constitutional taint be suspected, the same should be eradicated as soon as discovered. Such a disease as this, with so great an influence for evil in childhood and adult years, with such insidious and diverse symptoms, leaving behind so serious a tendency to various catarrhs and nervous instability, requires to be dealt with as speedily and effectively as possible with all the means available, no pains being spared to employ any remedy that may be expected to vanquish or allay its machinations. The management of the disease should commence with the pregnant mother; her health should be supervised during the critical period of gestation; she should be encouraged by all available means to suckle her own child, and be educated to understand and appreciate the manifold dangers of artificial feeding. Further, the health of the mother during lactation must be attended to with every care; and, when necessary, free meals might be provided for women with suckling infants on the Pansian lines now so well known. The enactments governing the administration in factories &c should be so arranged as to prohibit a woman being there employed during the later months of pregnancy; and the interval after delivery before return to work is allowed should be increased, if possible, without interfering with the finances of the home. The early notification of births is sure to prove a salutary measure, when the obvious opportunities which it affords are fully taken advantage of.

To various parts of the world expeditions of scientific enthusiasts have been sent out in order to stamp out or alleviate the ravages of tropical diseases. They have done incalculable good, working on the lines of house-to-house inspection, the filling up of damp-containing pits, the draining or paraffining of pools, and so forth as described in works on tropical hygiene and medicine; and in such ways, and by vigorous sanitation and administration, frightful afflictions have been checked in enormous areas where before they were so prevalent and death-dealing. But it is not possible for us to attack such a widespread malady as rickets in this way. But it can be limited somewhat. Painstaking associations of workers, sanitary inspectors, lady visitors and medical men may keep in touch regularly with the mothers and, if possible, supervise the infants from the day of birth before ignorance and evil influences have done irreparable injury to the young members of the community concerned. It will be necessary to insist upon the importance of fresh air and sunlight; and the mothers must be educated (by verbal advice or leaflets) as to the urgent necessity of feeding their infants at the breast - the natural source of early dietetic good. In the absence of medical sanction, the rearing of a child on farinaceous food for the first few months of life should be rendered an offence at law. During its early existence the infant should be weighed and inspected at regular intervals, and the mothers should be encouraged, in every possible way, to interest themselves in the future welfare of their children. But if artificial feeding becomes absolutely necessary, the mother should be trained in the simplest methods of using cow's milk with ordinary diluents, as well as in the details essential to the contamination of the milk or its containers; without difficulty or extra expense they should be able to obtain supplies of clean milk from such dairies as are beyond suspicion in the dietetic sense. In short, only by widespread organisation shall we be able to check the ravages of rickets amongst infants and help to rear a truly imperial race. Indeed, rickets might even become a rare disease if such means were generally adopted and practised.

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II. SYNONYMS AND POINTS OF COLLATERAL INTEREST.

The nomenclature of rickets is somewhat extensive, as the following few of numerous synonyms will show: "Rachitis," "Rhachitis," "Rachitismus," "Morbus Anglicus," "Maladie Anglaise," "Osteoporosis," "Scrofula Rhachitis," "Rachitisme," "Articuli Duplicati," "Chastre," "Nourure des Jointures," "Doppelte Gleider," and "Englische Krankheit." Etymologists tell us that the name "rickets" is derived from or is an English modification of the old Norman word "riquets," meaning one with a hump on his back. Some, however, would trace its origination still farther back into antiquity and see in it a derivation from the old term "wrickhan" or "wrikken", meaning to twist or wrest. Be that as it may, there can be no doubt that rickets was known in Great Britain many centuries ago. Indeed, so well were the general public acquainted with the peculiar deformities produced by the affection that the term "rickety" soon came into general vogue and was, and still is, used when referring to defective articles of furniture and so forth, for example, rickety tables and chairs. Glisson, - who, as we shall presently see, first observed the malady in the middle of the seventeenth century, - pleaded that the name should be changed to "rhachitis" - partly no doubt from its resemblance in sound to rickets, and partly in view of the existence of a Greek word "rachis", which means the spine, which part of the body, in his experience, was especially affected by rickets. The ailment was said to have been unknown on the continent before the publication of his observations; but it soon crept into prominence there under such synonyms as "morbus anglicus", "maladie anglaise" and "englische krankheit" - each of these designations being used to indicate the supposed territorial origination. The common expression in Germany is "doppelte gleider", or double-jointed; it was also there called "articuli duplicati", with a view to the indication of the swellings at the ends of the long bones which are so conspicuous in the course of the disease and give the double-jointed appearance just referred to. The vernacular term in France is "chastre", - a derivative of "castrum", - suggested by the deprivation of liberty caused by the disease; it means a prison and is intended to convey the impression of a person being imprisoned in a morbid dwelling under conditions of liberty deprivation and movement restriction. In the same country it is also referred to as "nouures des jointures" which is a synonym based upon the similarity of the enlargements of the epiphyses to the swellings or knots sometimes seen in the branches or trunks of trees when the circulation of the sap is interfered with, as well as the idea that the joints, or the limbs a little above, appear as if they had been bound round with a string; "noués" meaning bound up. I have come across the term "scrofula rachitis" in my perusal of the older works; it would seem to indicate that rickets is a variety of scrofula, which theory had not a few advocates in earlier times. Some of the older authors call it "osteomalacia" and tell us that rachitis and that affection constitute one and the same anomaly of the osseous system, modified, it may be, by the age of the patients; also that rachitis is the osteomalacia of infancy. There is a good deal of interesting literature bearing on this point, which it is foreign to my purpose to review; sufficient to note that, whatever may be the derivation of now generally accepted designation of the term rickets, it is as good as any other form ordinary use and has the triple recommendations of pronounceability, distinctiveness and simplicity.

III. WHAT IS RICKETS?

A disease like rickets, of such protean character and varied manifestations, is somewhat difficult to define in a few words. It is essentially a chronic nutritional disorder occurring during infancy or early life (six months to two years, usually), of insidious onset, slow in development, tardy recovery, but marked amenability to appropriate treatment, with early symptoms of indefinite character, attacking principally the nervous system and the various systemic organs concerned in the processes of digestion and assimilation of food - its final and most definite signs being found in the osseous parts, where it is readily detected owing to the bending and deformities of the affected bones. The malady is one that gives rise to a peculiar retardation of development to the extent of late dentition, inability to walk at the orthodox time and slowness in learning to converse - such signs of defective development perhaps continuing after the period of infancy and drawing attention to the presence of the disease.

It is highly desirable that the element of malnutrition be included conspicuously in any definition of this malady, and that it should not be dogmatically assigned to the diseases of the skeletal system lest the real nature of the affection be lost sight of. The distortion of the bones of the cranium and limbs, though often the most conspicuous features of the ailment, are only part of the real clinical picture; and, in order to be able to treat rickets on intelligent lines and at the earliest possible date, the other signs of depraved nutrition, - such as sweating of the head, restlessness, delayed dentition and the various digestive and nervous disturbances, - should be borne in mind in order that the child may be properly overhauled and no time be lost in the correction of the defects in its diet or hygienic circumstances. It is also of prime importance that rickets, in the sense of a bone affection, be recognised as not of sudden origination; for, prior to the development of the osseous anomalies, there is a period of invasion marked by such gastrointestinal troubles as diarrhoea, night-sweats, weakness and a greater or less elevation of the body heat. The digestive disorders take the form of frequent vomiting, diarrhoea alternating with constipation, with evacuations of an acid reaction and admixture with food particles which have failed to be fully digested in the stomach or upper digestive parts. The sweat is also of acid reaction, with a marked profuseness on the belly and skin of the head: so that during the hours of sleep the child's head is soaked with liquid and the pillow is made wet. The first osseous anomaly to make its appearance is a deformity at the wrist or ankle, following which the condyles of the femora are attacked. The anterior ends of the ribs also undergo enlargement. Sometimes the affection is restricted to these epiphyseal enlargements; but in other instances the shafts of the bones are bent in by the pull of the muscles or by the weight of the body or the pressure of the air. In the later stages of the malady deformity is sometimes caused by longitudinal growth of the bones consequent upon eburnation of the epiphyses. When the disease attacks the spine and chest wall great deformity of the trunk results from the antero-posterior and lateral curvature of the spinal column, accompanied by flattening of the thorax and lateral deviation of the ribs. The pelvis also becomes deformed and the cranium shows flattening of the occiput, together with prominence of the frontal regions and increase of the biparietal diameter, giving a peculiar square and box-like appearance to the entire head. The angles of the jaw sometimes exhibit excessive squareness and prominence and, consequent upon the yielding of the softened bones during the nursing's suckling, there may be a great vaulting of the roof of the mouth with protusion of the narrowed jaws. In a considerable proportion of rickety cases the affection is most evident in the legs. The alteration in the contour of the bones is more common in the ankles and legs than the thighs and in the lower portion of the body more than the upper, while the distal portions of the upper extremities suffer in preference to the upper portion of the trunk and skull. Such conditions, however, do not always obtain, as we sometimes encounter curvature of the spinal

column and deformity of the thorax without anything being amiss with the lower extremities and pelvic girdle. Rachitic affections of the cranium are due to delayed closure of the fontanelles which, instead of filling up at the end of the second year of the child's existence, sometimes remain patent up to the third or fourth year. The consequence of this delayed fontanelle closure is that the brain goes on developing and dilates the skull until its measurements are out of all proportion to the size of the face. The casual observer sometimes notes a certain amount of resemblance between the outline of the rachitic skull and that of the hydrocephalic head; but inquiry shows that the intellectual conditions are vastly different. The brain of the hydrocephalic subject is imperfectly developed and is buried in a dropsical effusion, which obscures the intelligence of the patient. On the other hand, the brain in rickets undergoes no such compressional interference, is at liberty to develop and possess unimpeded and perhaps remarkably acute cerebration. The intellectual activity and mental sharpness of the rachitic individual, - even the horribly deformed, - is not infrequently remarkable and has long been recognised as such. Indeed, from the class of humpback dwarfs the court jesters of old were recruited. The rachitic alterations in the jaws are apt to have a peculiar effect upon the child's teething, great delay in the cutting of the temporary teeth being one of the early important signs of the malady. The narrowing of the jaws sometimes leads to the teeth being crowded out of place: so that great irregularity in this position and order of appearance may be observed. The form of the teeth also may be altered: so that they appear to be too large and roughly formed, as well as changed in a variety of ways. The deformity of the thorax is one of the classical signs of the well-established disease. The normal antero-posterior and lateral curves of the spinal column are sometimes greatly exaggerated and the dorsal lateral curvature has its convexity always directed towards the right side of the body. A compensatory curve in the opposite direction makes its appearance in the lumbar part of the spine. The vertebral column is also twisted upon itself: so that the ribs project conspicuously upon the side of the lateral convexity of the spine, while there is a corresponding depression of the ribs upon the opposite side of the body. The posterior line of incurvation of the projecting ribs forms the prominence of the humpback person, and a corresponding projection of the costal cartilages and angularly curved ribs exists in from upon the opposite flattened side of the chest. At the point of junction between the costal cartilages and the anterior extremities of the ribs an ossified protuberance can be felt beneath the skin upon each rib, distinctly marking the line of morbid proliferation and ossification in the sterno-costal epiphyses. The sternum itself is often pushed forward and rendered prominent in a way that gives to the laterally compressed thorax a remote resemblance to the pectoral conformation of a bird: hence the term "pigeon-breast" that is often employed in designation of this peculiar osseous alteration. The lower portion of the chest wall is often very much inverted in consequence of the compression of the lateral walls' compression by the weight of the atmosphere; while the abdominal organs, pressing upwards and outwards, expand the lower ribs, which rest upon the diaphragm. Gaseous distension of the stomach and intestines, and downward displacement of the liver from the rachitic deformity, give rise to excessive enlargement of the belly in not a few instances of the disease. Pelvic anomalies are also the outcome of abnormal pressure effects. The lateral walls of the pelvis are sometimes pressed inwards, causing anterior projection of the pubic symphyses; but in those cases in which there are lateral curvature of the spinal column and unequal distribution of the weight of the body upon the sides of the pelvic bones, its walls are flattened upon the same side with the deviation of the lumbar spine, while a corresponding projection of the bones at the inferior strait exists upon the opposite side. The deformity of the chest interferes with the functions of the lungs, which are usually of irregular size and of stunted growth. It sometimes happens that the heart is displaced upwards or to the right side; and, owing to the fact that it is forcibly crowded against

the front wall of the chest, its pulsations are unusually conspicuous - the difficulty attending its movements sometimes causing actual hypertrophy of its muscular tissue. Such rachitic patients may complain of emphysema, and disease of the respiratory organs is particularly serious because of the difficulties that attend the movements of the lungs and the accomplishment of the pulmonary circulation of blood stream. Such persons very often exhibit the symptoms of spasmodic croup, laryngismus stridulus, spasm of the glottis and severe forms of pertussis - these affections being due to the special tendency to spasmodic contraction of the laryngeal muscles. In the case of females, pelvic deformities are especially dangerous, for the same may cause the person's death at the time of being brought to bed with child: short of this, unusual difficulty in parturition is very often encountered. The rachitic individual has a clumsy and laborious gait when there is deformity of the long bones, giving rise to such affections as knock-knee, bowleg and club-foot. It sometimes happens that the bones are so fragile that they readily fracture; the bones may fail to unite and so constitute special complications. Rickets does not even spare the unborn child always, though it is exceptional to find deformities of its long bones, multiple fractures, enlargements of the fontanelles and other changes which characterise the so-called ~~infant~~ infantile forms of rickets. It is also possible that in certain instances the children of badly nourished and weakened mothers may show at birth the symptoms of congenital rickets, which possibility should be remembered during the period of pregnancy. It is also possible that the commencement of the malady may be delayed until the later period of the life of the child, rickets being then observed as a sequel of perhaps one of the exanthemata - measles for example. The lateral curvature of the spine, which is in some cases seen to develop at puberty, has sometimes been referred to rickets. An acute form of rickets has also been described.

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IV. EVOLUTION OF OUR KNOWLEDGE REGARDING THE DISEASE.

In a certain sense rickets is almost an affection of modern times and its concomitant advances in civilisation; it has continued to increase more and more in countries since women have left their homes to work in the factories and mills, feeding their nurslings in irregular fashion, or adopting the pernicious practice of artificial rearing - one of the well recognised drawbacks to the so-called civilised state. In former times they worked at their spinning-wheels or by no means unhealthy domestic avocations in their own homes and tenderly nurtured their infants. Nowadays, they follow their husbands to the factories, placing the child out to nurse with a neighbour or leaving it in a crèche, where it is necessarily deprived of its natural maternal nourishment. New customs, new ailments, &c., all bring in their train reports, conferences, histories, and an infinity of publications and investigations. Taking a general view of the history of rickets, we shall find that, though its exact origination is lost in the dim and distant ages of the far remote past, it began to be intelligently recognised about the middle of the seventeenth century, when a disease appeared in the southwestern English counties, particularly in Dorset and Somerset, which spread gradually over the whole country. At this time Glisson's famous work saw the light, though many writers seem to have affirmed that the disease was known long before his time. As a matter of fact, Beylard mentions that it was studied even in the lifetime of the great Hippocrates, who is said to have referred to rachitic osseous anomalies in his narrations of certain deformities of the skeletal system. Such older writers as Galen, Celsus and Zacutus Lustianus have referred to bone diseases which some recognise of rachitic origination. During the above-mentioned century the disease is said to have prevailed on a by no means minor scale in Switzerland, Germany, Holland and France. Nevertheless, the fact remains that prior to the seventeenth century no clear and unmistakeable description of rickets was ever published - only vague generalities and nothing more. But it must, all the same, be noted that, in the fifth volume of his famous "Clinical Medicine", Trousseau affirms that in the year 1630 the English mortality tables for the first time refer to an affection which none of the doctors and inhabitants of the greatest age ever experienced before. The common people, he tells us, called it the rickets. This fact caused considerable speculation, with the result that numerous practitioners commenced to investigate it on special lines, which their great opportunities for research afforded, and prepared a report for general professional consideration. A commission of eight members was appointed to inquire still further into the machinations of the disease, amongst whom the names of Glisson, George Bate and Regemorter are conspicuous. Their report, published by Glisson, is the really first authentic account we have of rickets. This is one of the most interesting and famous of all earlier medical publications. It was entitled "De Rachitide sive Morbo Puerili," was written in Latin, - the language of the educated of the time, - and was issued about the year 1647. The limited edition was soon disposed of: so that in 1650 another was required, and yet another in 1671. Glisson's narration of the disease was remarkable for its clearness and comprehensiveness, no point of then recognised importance being omitted. To give an example, he showed that the affection has undoubtedly been observed at birth, but that it was usually seen to commence after the first six months of life, also that it was now and then almost epidemic in various parts of the country. He drew attention to the fact, moreover, that it led to delay in dentition, and stated that, if the teeth failed to appear before the onset of the malady, they very soon commenced to undergo decay. The interference with the respiration of the child, leading to insufficiency of the circulation and ~~subargement~~ ~~of the~~ ~~veins~~ ~~under~~ ~~the~~ ~~skin~~, especially those of the scalp, was mentioned in his work; likewise the "pot-belly", which he said was due to the fact that the rickety patients were usually gluttons or over-eaters. He blamed disturbance of the nutrition by the arterial blood for the production of this malady and maintained that the changes in the osseous tissues

were due to their excessive blood supply - this point leading up, as it were, to the experiments of Kassowitz, in which he produced hyperaemia of the bones artificially, with resulting changes not unlike those seen in rachitic change. Glisson was indeed a remarkable man, with an infinite capacity for keen observation and taking pains. Therefore, no account of his researches would be complete without a brief note of his useful life. We are told that he was born, in 1597, in the Dorsetshire hamlet of Rampisham. After the usual elementary educational routine he was sent to Cambridge University (Caius College), of which he ultimately had the honour to be elected a Fellow. He took his degree in medicine and succeeded Winterton in the chair of physic, which position he continued to adorn for forty busy years. In 1634, the London College of Physicians made him a Fellow. In all his methods he followed in the footsteps of the famous Harvey, relying more upon practical work and minute investigation than vague theories and hearsay affirmations. At one time he resided in Colchester and was conspicuous during the memorable siege and surrender of that place during the time of civil war. His great fame was achieved through the exacting study he devoted to rickets. For many years he was president of the London College of Physicians, was one of the foundation members of the Royal Society and died in 1677 in the London parish of St. Bride. He had the reputation amongst his contemporaries of being a very learned man, he is said never to have made an enemy, the public respected him and he was beloved by all his friends. His fellow-worker, George Bate, seems to have been born at Maids-Morton, near Buckingham. He was educated at New College, Queen's and St. Edmund's Hall, Oxford, in which University town he had a physician's practice of some note. His skill was talked of in distant places, and reports thereof, coming to the ears of Charles I., he was made Royal Physician. Oliver Cromwell employed him, and he was appointed to the Court after the restoration. We are told that his great recommendation in the case of the second Charles was the allegation that he gave a lethal dose of medicine to Cromwell. His writings were very numerous and finished only with his death in 1699; he was buried at Kingston.

Some of the writers of the period under consideration express considerable doubt as to the alleged origination of rickets in the seventeenth century and give vent to their surprise that an affection with such striking characteristics and infinity of varieties should have remained in oblivion until so late a period. They tell us that it is inconsistent with several terms in the Greek and Latin languages denoting deformity and decrepitude of the human frame, as well as with instances given in the ancient history of persons so afflicted who won distinction as authors and soldiers. On the 48th page of the 5th volume of his "Clinical Medicine" Van Swieten affirms that rickets was undoubtedly a new disease, though admitting that in his time the point was much mooted. In 1852, Beylard published, in Paris, a work on "Rickets, Fragility of Bone and Osteomalacia", in which he insisted that rickets, far from being a new disease in Glisson's time, was as old as the hills, being recognised by such ancient writers as Hippocrates and Galen. He points to Whistler, of Leyden, as one of the writers on rickets before Glisson. Whistler's work, which was entitled "A Dissertation on the Infantile Disease called The Rickets" and published in 1645, is now so rare as to be worth almost its weight in gold; fortunately for the ordinary historian, there is a copy of it in the Bodleian Library at Oxford. It has been insisted that the very title of Whistler's book shows that rickets was well-known in England as a children's disease, and that it must have been recognised for a considerable period in order to have been nicknamed rickets. It would seem, these commentators tell us, that Glisson was familiar with Whistler's narration of the malady, and that the recognition of the affection by practitioners in England prior to the existence of the former. Moore, in the 30th volume of the Transactions of the Pathological Society, insists that Whistler has no right to be regarded as the discoverer of rickets, as his only claim to this honour rests on his assertion that he published a Thesis, in 1645, entitled "paedospianchnosteocace", of which there is no trace; and that, as he appropriated the funds of the College

of Physicians, while their president, his own statement should be regarded as being of little value. It would seem, then that Glisson is the only recognised discoverer of rickets in the form that we now know it. He attributed the prevalence of the disease in the south to the more luxurious habits of these communities, which nowadays is of interest, as the affection is regarded as more particularly affecting the poor. He first proposed the term rickets, as indicating one of the principal parts of the body attacked (rachis, the spine), and also because the sound of the word resembled the vernacular of the people. In the mortality bills of London, rickets first appeared in the year 1634, there being 14 deaths from it. In the period 1658-59, there were 476 and 441 deaths respectively, the increase probably being due to the malady being better known to the practitioners of the time. In 1660 Mayo, of Oxford, called attention to the softening of the bones in this disease.

During the eighteenth century there were numerous publications regarding the various points of interest and importance in the evolution of rickets, some of them dealing with it as affecting the animal kingdom. In 1707 Bicherod found bone softening in the case of an ox, and Lordat saw the same sort of thing happen in a monkey. About this time, writing of "Tuberculous Disease, Dupuy investigated the osseous anomalies in the case of a rickety horse; and Good, - in his "Study of Medicine", 5th volume, - saw rickets evolve in a lion - an interesting observation in view of Bland Sutton's well-known researches in the London Zoological Gardens. In 1740 the Bishop of Cork reported the case of a man who suffered so severely from rickets during childhood that every bone in his body was deformed and distorted. We are told that, at the age of 18, the patient began to grow stiff and at length, having lost the use of all his limbs, he became like a statue; he lived until the age of 61 and, at the post-mortem examination, his skeleton was found to be, as it were, one continuous bone from the top of his head to his knees; many osseous growths, some of them of the most grotesque forms, branched from his head, back and haunches; and a portion of abnormally-formed bone was also found embedded within one of the large muscles. In the same year Petit warned very seriously the people regarding the dreadful effects of early weaning. Ten years later, Duverney studied the minute pathology of rickets and drew attention to the fragility and rarefaction of the bones, as well as their liability to fracture under comparatively trivial traumatic conditions. In 1772 Comber gave a good description to rickets as it occurs in the case of sheep. In 1797 Portal classified the disease as syphilitic, scrofulous, scorbutic, rheumatic, arising from intestinal disorders or following one or other of the exanthematous diseases.

The nineteenth century was a period of marked progress in the proper understanding of rickets. Dealing with "Congenital Rickets", Romberg (1817) described the congenital form of the affection; and his researches in this respect were continued by other observers: for example, Lepelletier (1830), Henckel and Klein. Boerdenhave and Pinel claim to have seen rickets in the foetus; the latter says he has seen it in an eight months' child. Ruz (Researches on the Rickets in Infants, Gaz. Méd. de Paris, 1834) gave a good account of the affection arising in this way. A notable observer was Jules Guérin who, in 1834, first suggested the classification of the disease now very commonly recognised. He described its evolution in the following stages: (1) A period of incubation or of effusion, in which there is a general distribution of the blood throughout the bones of the body. (2) A period of deformity, in which the spongoid tissue forms in the epiphyses, diaphyses and the subperiosteal spaces, the bones at the same time undergoing softening and bending. (3) A period of resorption, consolidation and eburnation, in which the spongoid tissue becomes compact and the bones very hard. (4) A period of consumption, - but one not constantly present, - in which the osseous tissue remains fragile and rarefied. Guérin was the first to call the reticulated tissue of the rickety bone by the name spongoid; and he is credited of trying the experiment of making animals rachitic by altering their diet - success in the case of puppies being achieved. In 1843 Elsaesser wrote regarding the peculiar change met with in the skull as the result of rickets; to this he gave the

well-known name of "craniotabes". In 1849 Trousseau pointed out the resemblance of rickets to osteomalacia; and, three years later, Broca communicated to the Anatomical Society of Paris a careful and accurate description of the microscopic changes occurring in rachitic bones, stating that the same were due to an arrest or suspension of the normal development of osseous tissue. In 1853 Virchow pointed out the analogy of the rachitic bone alterations to parenchymatous osteitis, which observation met with considerable support at the hands of other observers. In 1881 Parrot contended that rickets was only a form of congenital rickets, which statement was speedily denied and promptly disproved. In 1895 Jenner published his course of famous lectures on the disease, which are largely quoted from by writers, recognising as they do the classical nature of his observations. Barlow, - writing on craniotabes and scurvy, - and Cheadle, - one of our greatest authorities on rickets, - advanced the generally accepted theory that the affection is one which has to do with feeding - the principal error, according to them, being a deficiency of fat in the food of infants and young children. Bland Sutton's experiments will receive due consideration presently.

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E T I O L O G Y

Though the actual exciting cause of rickets is at present unknown, it is interesting and instructive to consider the numerous conditions that seem to favour its development more or less - such as heredity, insanitary conditions, dietetic errors, prolonged lactation, and toxins. For a varying length of time such theories as these, and other fanciful ones, enjoyed some acceptance, only to be negatived by later observations to some degree. We shall see that, although rickets occurs in children who are subject to improper feeding, as is admitted by everyone, dietetic anomalies alone are not recognised as alone productive of the affection: for rickets would be a much more common assailant of young children that it actually is.

The various factors which fall under the above heading are as follows:

A g e

Observers are agreed that rickets is a disease which is particularly prone to attack infants or very young children under certain conditions such as will presently appear. I have found it to be most common between six months and two years and have seldom come across a case which started after the third year of life. In general, then, it is a malady coincident with the period of first dentition. Though usually occurring between ~~six~~ months and two years, it is known to break out at an earlier age, even in utero, or at a much later date than during the above-mentioned regulation period. Ashby (Ency. Medica) contends that, in its most typical form, rickets occurs between six months and two years, - which observation coincides with my own above-mentioned experience) - but admits that early symptoms may be noted in the latter half of the first year. Comby, in one of his earlier communications, stated that the malady commenced about the tenth to the twelfth month, that it may arise as late as two years, but is rare after three years. Out of 1662 patients examined, 1268 were between one and two years, at which latter age one of his contemporaries saw 2974 rachitic children out of 4176 cases. In the fourth volume of "St. Bartholomew's Hospital Reports", Gee publishes an analysis of 635 cases, amongst which were 365 boys and 270 girls of the following ages: 32 under 6 months; 144 from 6 to 12 months; 183 from 12 to 18 months; 133 from 18 months to 2 years; 116 during the 3rd year; and 27 during the fourth year. He supplies the interesting information that rickets was present in 30 per cent. of all the sick children in his hospital; but it is probable that the disease was in existence before admission, the age at which only is stated. He seems to think that a third, or thereabouts, of all children under twenty-four months are rachitic. The large proportion of cases are from the period of weaning and through the process of cutting the teeth. Starr says that rickets may begin in intra-uterine life; but Vincent doubts, as I do, if there is any foetal condition to which the same of rickets may be applied. In the course of his work on "Diseases of Children", Hutchinson expresses his doubt as to there being definite signs of rickets frequently observed under the age of six months; he thinks that the most likely time for it to come under observation is at about the eighteenth month of life, though it may start earlier than that, several writers having satisfied themselves of the correctness of their diagnosis of the condition in the second six months of the child's age. In one of his contributions (Arch. Ped.) eleven years ago, Jacobi stated that the disease not infrequently shows itself at a very early stage entirely in the muscles; and he says that obstinate constipation, beginning in the second or third month of life, can be regarded as a suggestive sign of rickets. He applies this rule even to breast-fed infants, not constipated at birth, but becoming so in the second or third month of their existence. In the work of which he is, with Poland, joint-author, Chance (Bodily Deformities) says that he has for a long time considered that rickets originates during uterine life whilst development is going on and while the basis of the osseous tissue is being laid down and its constitution given to it. He also

emphasises the fact that rachitic children always have bad teeth; he holds that this is due to some original constitutional cause, not merely to imperfect nourishment after the child is born. He has taken careful notes of 600 cases and observed that two-thirds of their number appeared in the first year, which he takes as proof that rickets is a disease of infancy and that the cause thereof must originate immediately after birth, or more probably in the pre-existence of a congenital disease. He compares his tabulation with that of Guérin and finds that ~~it~~ both in the main are in agreement to the extent that the vast majority of cases of rachitic occur under two years of age. He found that 472 of his cases out of 600 occurred within the first year of life; out of this number 204, or a third of the whole, were before 6 months, and 108 were noticed by the mothers within the first month of life, which persons usually contended that their infants were born with the disease. There were a few cases between 2 and 5 years of age; he adds that his tables support his opinion that rickets is always a congenital disease, arrested for a time and revived later on, a few months after the infant comes into the world. In their work on "Diseases of Children", Goodhart and Still give an analysis of 141 cases. There were 68 boys and 73 girls. At 5 months there were 3 cases; at 7 months 1 case occurred; at 8 months 3 cases; at 9 months 2 cases; at 10 months, 5 cases; at 11 months 6 cases; at 12 months 11 cases; and at 18 months 36 cases. At 2 years there were 26 cases; at 2½ years 19, at 3 years 13; at 3½ years 2 cases; at 4 years 6 cases; at 5 years 5 cases; and 3 cases at 6 years. Fordyce (Brit. Med. Jour., Apr. 28, 1906, p. 971) has published an analysis of 116 cases of rickets between the ages of six months and 3 years: In 55 per cent. the age was from 6 to 12 months; in 63 per cent., 12 to 18 months; in 65 per cent. from 18 to 24 months; and in 52 per cent. from 2 to 3 years. In a short series of 46 cases of which I have knowledge there were 10 during the first 6 months; 14 during the second 6 months; 12 during the third 6 months; 7 during the fourth 6 months; and 3 cases during the third year. In other words, there were 24 cases during the first year; 19 cases during the second year; and 3 cases during the third year. In this observation of the occurrence of the disease during the first three years of life the 10 cases which occurred during the first 6 months of the child's existence are dated from the onset of the digestive disturbances, which were always followed by osseous disturbances of undoubted rachitic character. Rittershain has studied 521 cases of rickets, of which 91 were seen during the first 6 months of life; 175 during the second half year; 154 from 1 to 2 years; 62 from 2 to 3 years; 15 from 3 to 4; 7 from 4 to 5; and 17 from 5 to 9 years, Guérin had 3 cases of rickets occur before the child was born; 98 during the first year; 176 during the second; 35 during the third; 19 during the fourth; 10 during the fifth; and 5 from the sixth to the twelfth year, making a total of 346 cases, of which 148 were males and 198 females. Ritchie has tabulated the age incidence of 219 cases. In the first 6 months 7 were seen; in the second 6 months 65; from 1 to 2 years 109; from 2 to 3 years 25; from 3 to 4 years 9; and from 4 to 5 years only 4. In Brueniche's 163 cases there was only a single instance of rickets during the first 6 months of life; 19 cases occurred during the second 6 months; 79 from 1 to 2 years; 47 from 2 to 3 years; 7 from 3 to 4 years; 6 from 4 to 5 years; and 4 from 5 to 8 years. In the fifth volume of Heath's "Dictionary of Practical Surgery", Parker states that he has knowledge of 1000 consecutive rachitic cases. 20.2 per cent. (202 cases) occurred during the first 6 months of life; 27.8 per cent. (278 cases) during the second 6 months; 23.8 per cent. (238 cases) during the third 6 months; 14.4 per cent. (144 cases) during the fourth six months; 9.8 per cent. (98 cases) during the third year; 1.8 per cent. (18 cases) during the fourth year; 1.4 per cent. (14 cases) during the fifth year; and .8 per cent. (8 cases) during the sixth year of the patient's existence. Parker's researches show that there is a great frequency of rickets during the earlier months of child life, especially during the second half of the first year; also that it is by no means rare during the third six months and that after the first half of the second year there is a rapid and marked fall in the number of cases observed.

I have given long consideration to an infinity of statistical compilations of the age incidence of rickets, both in this country and at Home, and during the pursuance of such studies have noted that the number of cases happening in the first and second years very greatly exceeds that of other periods of life, and that there is a very rapid diminution of the disease in the subsequent years. The predominance of female cases over those of males, according to certain tabulations, may be due to, I think, the greater delicacy of female constitution. I have seen a lot of cases in which the first occurrence of the disease was put at six months; numerous others occurred at two and a half years, the mean age of incidence being fifteen months. In some of these personal experiences it seemed possible, indeed justifiable, to assign the origination in large part to prolongation of the function of lactation; and I have noted an earlier onset of rickets in infants artificially reared, with delayed age incidence under the lactation circumstance just mentioned. Although the disease may not be evident in an infant before the end of the first year, it is advisable that the minor suggestive symptoms of the dyscrasia should be carefully searched for at a much earlier period, in order, if possible, to arrest the insidious advance of the affection before the later effects, - such as malnutrition, retardation of development or deformities of the skeletal system, - have made their appearance and permanently disabled or interfered with the stature or physical conformation of the individual.

C l i m a t e

Any account of the geographical distribution of rickets must be premised with the affirmation that it is practically worldwide in occurrence, though much more often encountered in countries which are cold and damp, particularly under circumstances of deficiency in sunlight in long-winter climes. More of it, then, is seen in temperate countries, and is therefore very well known in regions of cold, damp, changeable weather, such as Great Britain, Holland, parts of Germany, Austria, France, and Italy - the north especially. On the other hand, it is favourably influenced and tends to die out under conditions of dryness, sunlight and abundant warmth. To a large extent it is an affection of all the large modern cities and manufacturing towns. It is quite common in the large towns of Australia and New Zealand, and there are even numerous cases in the bush and forest districts. Travellers tell us that the disease is almost unknown in the extreme north - e.g., Greenland, Iceland, as well as in Norway and Denmark: possibly because in all these countries **heat**-producing fat enters largely into the dietary of the people. Cases which occur in these parts are said to be of a very minor character. Coming to Asia, it is interesting to note a marked contrast in the great rarity of the **malady** in China, Japan, Turkey, and the like places. Contrary to what obtains in the north of Italy, rickets is seldom seen in Greece. I am told that it is very common amongst the Egyptians of the lower orders; and my informant explains this on the ground of the prevalence amongst them of such debilitating conditions as tuberculosis, prolonged lactation, bad ventilation and early marriage. Tropical observers report that the malady is very rare in **hot** countries, possibly because it is, when occurring, so mild in evolution that its symptoms are overlooked or disregarded. It is very rare in mountainous districts or at high altitudes, especially if the soil is dry; whereas low-lying and marshy localities show innumerable instances of the disease. Such places are apt to be at times cold and damp, therefore favouring bronchial and intestinal catarrhs and predisposing to the ailment. Similarly, it is more often seen to arise in the colder season of the year, when the people are less often in the open air. Some writers of note consider that climate and a deficiency of oxygen in the respired air are very important factors in the origination of rickets, the infrequency of which in hot climates they explain by the open-air lives which both mothers and children lead in such places. This would explain the rarity of rickets in India, where it seems to be limited to the children of soldiers living in damp localities and marshy places, and who are comparatively seldom in the fresh, pure air. I have already noted the prevalence of the affection in New Zealand and

that it is by no means infrequent in Australia, in certain parts of which, being modified by the semitropical climate, the osseous anomalies are of less severity than in temperate countries. Muskett (*Australas. Med. Mag.*, 1891, pp. 285-291), writing of the Australian occurrence of rickets, states that the absence of deformities is due to the fact that in such a subtropical climate the children require less animal fat and proteid in their food, as the starchy elements satisfy the requirements of the system and the osseous tissues are not so adversely affected as in colder regions. Although a cold climate and an unhealthy environment are markedly predisposing to the occurrence of rickets, that affection affects all races alike as a rule. In the large cities of America it is common enough, though comparatively rare in sparsely populated Canada. In New York and such great cities as Philadelphia most rickets is seen amongst the Italians and negroes; and extreme cases are said to be almost invariably restricted to such nationalities. But it must be understood that the disease is by no means confined to these two races. It is interesting to note the frequent severity of Italian affection, even breast-fed infants being liable to the disease. Both Italians and negroes seem to bear badly their transformation from the warm semitropical countries to more temperate climes; they undoubtedly suffer severely from the confined life of the northern cities: for they are obliged therein to live in dark cellars or tenement houses, where there is little sunshine, and they miss the open air and the genial warmth of their native land. Their dietetic customs remain unaltered; invariably they continue to eat the food that is customary in their homeland and on which they do not develop the disease: so that the prevalence of rickets in these two races, under new conditions of life, is not so much due to improper food as to the deficiency of fresh air, sunlight and warmth. It is possible, though not proven, that there may be some racial peculiarity which tends to make them unduly susceptible to rachitic disease. In the Southern States of America the affection is not common amongst the negroes, because they are able to secure plenty of fresh air and sunlight; and it is probable also that they there lead more open-air lives and have houses which are better ventilated by wide-open windows. In the Maine Coast towns the children are able to have plenty of fresh air and sunlight; and, although many are badly fed and often by artificial means, rickets is but rarely seen in this part of America. The disease is considerably modified by climate and in semitropical countries may make its appearance in a form so altered or toned down that the true nature of the affection may be overlooked by the careless observer. In New York City the osseous anomalies of rickets are late in making their appearance and cases which show such slight symptoms as fretfulness and perspiration about the head, without any gross deformities of the bones, may pass unobserved and fail to be diagnosed as being accompaniments of the rachitic state. The records of the Infant Asylum in that city show that 1 case in 9 of the children examined were rachitic. The affection everywhere is most common amongst the children of the poor in the slums, though the affluent members of the community are by no means exempt from the disease. On the other hand scurvy is more frequent amongst the children of the well-to-do, owing to the antiscorbutic element being deficient in the food. No less than 50 per cent. of the children attending the London hospitals are said to be rachitic, though in certain years the figure has been 30; the percentages in other cities of Europe were Prague 30, Vienna 80, and Manchester 30. These figures, which are given by Barlow and Bury and which concern children under three years of age, have their accuracy called in question by Kassowitz, who affirms that, if craniotabes and swelling of the ribs were included, the statistics of the English towns would be higher still. This seems to be the general opinion, it being believed in most quarters that 50 per cent. of the attending children under two years of age have rachitic symptoms. Such points as these are well borne out by the reports of the Commission on Physical Deterioration. It has been affirmed that children suffer from rickets more acutely, if not more generally, in the North of England than in the southern parts, there being in the former a larger

proportion of deformed children encountered than in London, for instance. The employment of mothers in various unhealthy avocations would partly account for the greater prevalence of the disease in the part named, though poor and unsuitable food is largely operative. During the course of one of the medical inspections in the city of Leeds, Yorkshire, there was found 50 per cent. of rickets in a poor school, 8 per cent. in a good-class one, but only 7 per cent. in a poor Jewish school. Some 2335 board school children were examined; half of these were rachitic and a much larger proportion had defective teeth. Amongst other children in Leeds it is estimated that from 15 to 20 per cent. are rachitic. It is said that the disease is diminishing in Glasgow, - though some have denied this, - the former "home of osteotomies." Rickets is a very common ailment in Sheffield and some of the deformities encountered very repulsive; this has been referred to the factory working of the mothers, though doubtless other factors are at work. Curiously enough, rickets is not so common in Liverpool as in some of the other Lancashire towns. This has been referred to the fact that there is not so much overcrowding in it, and to the fact that the people are better fed and housed; the slum population there are mostly Poles and Germans and not nearly so rachitic as might be supposed. Manchester is less overcrowded than Leeds and therefore less rachitic. In the former city, according to Ashby, in 1903 there were 901 infants and children, outpatients, suffering from rickets in its early stages; 539 children over 3-4 years of age had such deformities as knock-knee and bandy legs. It is affirmed that there is more of the disease in Edinburgh than in London, though it does not seem to be on the increase. In Boston some 80 per cent. of the children attending the dispensaries showed signs of rickets; even a higher percentage is reported from Russia, where it is 95, including the slight cases; and 35 per cent. are more or less severely afflicted. In Norway rickets is said to be more common from April to June, and the least frequent from November to December. At Johannessen's clinic there were 32 per cent. of rachitic children.

C o l d E x p o s u r e

Very young children are very susceptible to exposure to cold; the infant becomes chilled, its vitality is thereby lowered and soon gastrointestinal disorders or catarrhal respiratory conditions make their appearance. The legs of the infant are not infrequently bare, especially in the case of slum children. If no napkins are other protectives be worn, the wind blows upwards and strikes the abdomen, the organs of which are apt to be adversely affected by such neglect. In other cases the child spends much of its time in crawling about the floor, where cold air circulates in strong currents, carrying usually dust and germs, with aggravated danger to the recipient. The soles of the feet are particularly susceptible to cold and damp; for this reason they have been referred to by Money as "external kidneys." In this way cold may easily cause impairment of health; and rickets may follow in the wake of malnutrition or the lowered vitality thus engendered. The coldness of the climate, the lack of outdoor life and the constant breathing of impure air appear to be the causes that diminish the resisting-power in the negro and Italian races of America and so induce a special susceptibility to rickets in the case of their infants. Barlow and Bury, whilst admitting the unfavourable influence of such factors as we are considering, state that insufficient clothing of the infants' limbs must be taken into consideration.

D i e t

The feeding of the infant has on innumerable occasions been blamed for the production of rickets, with the result that the theories regarding the same are as plentiful as in not a few instances fantastic. To deal with all of these would absorb quite a bulky treatise: so that it is proposed merely to consider only the better known. When one comes to investigate the causation of the disease on modern and practical lines, one soon concludes that most of the hypotheses

fail to supply evidence strong enough to justify the claim that one factor alone is operative in the production of the malady. Climate, - especially that of cold and temperate regions, - bad air, insufficient ventilation of houses, want of sunlight, heredity, syphilis or constitutional disorders, lack of lime in the drinking-water or in the tissues of the body, and excess of lactic acid in the blood may all have some predisposing influence; but none alone is able to make the growing infant rachitic. The insufficiency of such theories, therefore, led investigators to turn to dietetic anomalies and to inquire if any error in the food could have an unfavourable influence upon the health of the child, which might culminate in rickets eventually.

One of the first lines of research lay in the natural food of the the infant, inquiry being directed to the possibility of there being a fault as regards want of balance of the dietetic constituents, overfeeding, underfeeding and the like. The natural food of the infant is its own mother's milk; it is, therefore, at once evident that it is of prime importance to examine it, if the nursling is of rachitic inclination, so as to determine what alteration should be effected in the feeding. The growing disinclination of mothers to suckle their infants and the tendency to resume their employment too soon after the birth of the child are some of the causes of the increase of rickets in modern times. They undoubtedly are largely accountable for the great and stationary infantile mortality seen during the last half-century or so. But, as the infant is often deprived of its natural nourishment and fed by artificial means, it is just as important to ascertain if the use of these substitutes for the same and cow's milk, - i.e., patent foods, condensed milk, &c., - are more liable to cause rickets, and what alterations in the dietary, or defect as regards nutritive value, can be productive of so common a disease as that under consideration. Mother's milk must be taken as the standard of the healthy diet for the developing infant and, therefore, any substitute for this must possess the same elements in proper proportions. It is advisable, then, to state right now the composition of human milk, to consider its various constituents and then to ascertain what variations thereof injuriously affect the infant so far as rickets is concerned.

In common with many others, I have made a series of analyses of milk and find that the composition of the human secretion is, approximately, as follows: Proteid, 1.5 per cent.; fat, 3.5 per cent.; lactose, 6.5 per cent.; mineral salts, 0.2 per cent.; miscellaneous constituents, 0.6 per cent.; water, 87.7 per cent. Another series of cases dealt with the milk of 25 women, 20 to 46 years of age, primiparæ and multiparæ, with healthy infants, during the first to third month of lactation. In such the proteids amounted to 0.06 - 2 per cent., the fat to 0.63 - 6.65 per cent., and the sugar to 2.55 - 9.77 per cent. I have found that the milk of primiparæ is richer in sugar, proteids and fat; also that poorly nourished females, - especially when there is an excess of starchy food, - give a milk that is poor in the proteid content; further, human milk is richer in fat between 20 and 25 years of age, richer in proteids between 25 and 30 - beyond which latter age there is more sugar than before. The cardinal principle which I desire to emphasise at this juncture is the fact that, when we come to examine into the bearing that food may have on this disease, breastfed infants seldom suffer from rickets up to the time when weaning should occur. The consequence is that as a food normal breast milk of a healthy mother may be regarded as unable to make an infant rachitic. There seems reason to believe that one of the reasons that rickets is practically unknown in China, Japan and India is because breast feeding enjoys an almost universal vogue in such parts. In Japan 99 per cent. of the infants are thus reared; though they are sometimes kept at the breast for years, fat or oil constitute extra foods. In Burmah and India rickets is a very rare malady, though it must be remembered that not a few of the infants die from opium poisoning before they have lived long enough to suffer from it; all the mothers nurse their own infants and, as the former live so much in the open air, the element of over-crowding scarcely obtains; moreover, the evolution of the

disease is less severe, even if the nursling becomes affected. Infants born before their time, or those who are reared with difficulty or who have suffered from diarrhoea and gastric or intestinal catarrh, are liable to become rachitic. Digestive troubles, flatulence, vomiting, diarrhoea, &c., though often the precursors of rickets, are not always present. Many young children with no preliminary digestive disturbances become rachitic, while, on the other hand, others who may have suffered very much indeed show no signs of the disease. If, with vomiting and diarrhoea, there is lack of power to assimilate food, rickets may be engrafted upon the malnutritional state. Starvation causes atrophy and not rickets, overfeeding being more liable to excite the disease. After the first year of life the defenders of the nursling against rickets are not so powerful. The maternal milk deteriorates in quality and the nutrition of the child begins to suffer. It is possible also that the prolonged drain of nursing has told upon the health of the mother, who now is unable to supply to the infant the proper proportion of food, with ferments and antitoxins, which have been its safe-guards against infection during the early months of life. Pregnancy during lactation also seems to affect adversely the breast-fed infant and tend towards the rachitic diathesis. In spite of abundance of good maternal milk, rickets occasionally makes its appearance in infants who are being fed at the mother's breast; but, as a general rule, I have found that the disease occurs in nurslings only when the mother is sickly and debilitated or has suckled the child into the second year, the milk having fallen off as regards its proper composition. So good an observer as Adriance (Arch. Pediat., 1912) refuses to subscribe to the common notion that breast-fed infants never become rachitic; he tells us that clinical experience teaches that it is no uncommon thing to see these infants with a mild type of the disease, more especially cranial rickets, this being difficult to explain except upon the ground that the milk contains a deficiency of bone-forming elements; he considers that a deficiency of proteids and salts predisposes to rickets, though due regard must be paid to many other etiological factors; he pays but little attention to the possible deficiency of fat, emphasising that it is the quality of the proteids which gives information concerning the functional activity of the milk; he cautions against accepting the amount of fat as a guide for estimating the true value of a milk as a food for an infant. The breasts of the nursing mother, with little effort, are able to secrete fat and sugar; but, in the later months, proteid is only produced in small amount. The milk of prolonged lactation is deficient in salts and proteids; the latter will be very low when lactation is about to cease, at any rate in the nursing period, unless under emergency or inability to suckle the child. Throughout the entire period of lactation the percentage of fat in human milk is extremely irregular, whereas that of the proteids remains constant until the approach of the time for weaning the child, when it falls rapidly. By the end of the eighth month it is below 1 per cent. The salts of the milk are intimately associated with the proteid and also show gradual decrease during the progress of the lactation function. Cases of rickets have been from time to time attributed to excessive feeding with fat; and I would suggest that a possible excitant is a failure, on the part of the infant in these cases, to absorb sufficient proteid. The proportion of the constituents of the milk must be kept at a normal standard; otherwise malnutritional disturbances are very apt to become prominent.

An infant could not survive in the absence of proteid from its diet, as it is the only substance capable of replacing the constant nitrogenous waste of the cells of the body and ~~preventing~~ any interference with the due supply leads to digestion and assimilation of nourishment being disturbed. Although proteid alone can sustain life, the vital functions are greatly assisted by the addition of fat and carbohydrate to the dietary to the end that body heat may be adequately maintained. It would appear that 22 parts of proteid can do the work of 10 parts of fat; but only one-half or two-thirds as much proteid are required if fat and carbohydrate are also present in the food. Proteid appears in the form of casein and albumen in milk; and it should here be noted that the proportions differ in the milk of the woman and the cow; in

human milk there is 0.59 of casein to 2.88 in the case of the cow's product; whereas the albumen amounts to 1.23 in the former as compared with 6.53 in the latter - making a total of 1.82 for the mother and 8.41 for the cow. Proteid starvation occasions backward development; the infant becomes weak in muscle, sitting up becomes delayed and the child is late in crawling and in learning to stand or walk. The most constant sign of proteid shortage is anaemia. There will also be poor-ness of circulation, flabbiness of tissues and failure of nutrition, with inability to digest the other food constituents. The child becomes fretful, peevish and irritable, there is a frequent tendency to perspiration about the head and the fontanelles and sutures of the head are late in closing. These signs of malnutrition from proteid deficiency closely resemble what we observe in rickets; the lesson is obvious, namely, that the infant's food should contain a proper proportion of the constituent named.

We find that the salts are in a state of organic combination, the calcium and phosphorus being combined with casein. Phosphorus is an active cell constituent and is always present when there is most active growth going on. The importance of calcium lies in the fact that it supplies the lime which is so essential to a proper process of bone formation. Milk which is deficient in casein will be poor in calcium, phosphorus and iron; the consequence of its ingestion will be that a predisposition to rickets may occur.

One other constituent of human milk which requires mention is lecithin; this is an organic compound necessary for the development and repair of the nerve tissues of the body.

Everyone knows that innumerable infants are fed at the breast, but receiving the additional nourishment of some such farinaceous food as boiled bread or biscuit. This class of patient is, I find, by no means easy to deal with; for the mother, if questioned about the feeding of her child almost always replies that it is having the breast, and only close questioning discloses the important fact that starchy food is given in addition. Such mothers usually resent any advice offered and in the smug assurance of an easy dietetic conscience she lends ear to the plaudits of the grandmother, relatives or interfering neighbours. Nevertheless, it is a fact that some of these infants thrive better with the addition of well-boiled bread; and what is known as "bread-jelly" constitutes a valuable dietetic aid in cases of diarrhoea or when the child resents ordinary milk. In general, however, a large proportion of such mixed-fed children suffer from convulsive troubles or disorders of the stomach or bowels. I have often tried to formulate some definite opinion as to whether or not infants fed on the breast, with a small addition of farinaceous food, more often become rachitic than others not so brought up. I have seen a few instances of such a happening; in the others the breast feeding was somewhat irregularly performed and a definite conclusion could not be drawn. By far the majority of cases of rickets which I have seen in this country and elsewhere, both in public institutions and in the home, comprised children reared upon cow's milk, the condensed product or patent foods so called. In such cases I have made investigation into the surroundings and habits of the child; though I have found such defective in very many instances, the real and almost invariable defect has been in the improper manner of feeding by hand. In the sixth edition of his work on "Artificial Feeding", Cheadle contends that there is only one constant factor present in the etiology of rickets, namely, the food factor; he maintains that all other conditions, - such as want of sunlight, bad air and damp and insanitary surroundings, - merely assist by degrading the nutrition of the child and are not always present or necessary for the production of the disease; he has never seen a child, properly fed and free from dietetic disorders, even in unhealthy surroundings, become rachitic. This is, I think, rather a strong statement; it certainly does not thoroughly explain the fact that the Italians and negroes almost always become rachitic when transplanted from their warm to the colder climate of America and probably to much less healthy homes, but under conditions in which their accustomed diet

is persevered with. Though rickets is an affection of the poor it nevertheless occurs amongst children living under the best hygienic circumstances who, as a rule, are fed by artificial means, more especially on patent foods, which are too often rich in carbohydrates and deficient in proteids and fat. The two elements deficient in the food of children who become rachitic are animal fat and proteid; but there may be a paucity of phosphate of lime likewise. I have already referred to the fact that proteid is essential for cell growth and bodily development; and have shown how symptoms similar to cranial rickets supervene when the percentage thereof is reduced to a low proportion in the mother's milk. Proteid, then, is the cardinal basis of the food; it is a tissue repairer and builder, as well as a blood producer.

The fat occurs in the system in the unchanged condition as such. Its functions are in intimate association with those of the proteid, it possessing the property of saving nitrogenous waste and so allowing the proteid to expend its entire energy on cell production. In other words, it is a proteid-sparer and prevents this element being drawn upon to supply body heat; for if the proteid is used for this purpose the body suffers and the child may suffer from malnutrition, anaemia and other troubles. Fat also assists in the formation of bone, probably by aiding the absorption from the intestines of inorganic salts, especially the earthy phosphates. On a milk diet, if fats are withheld or greatly reduced, these salts appear in great quantities in the evacuations of the bowels. A child requires more fat than an adult in the course of the day. It is said that a child under $1\frac{1}{2}$ years of age needs 30 to 45 grammes of fat and 60 to 90 grammes of carbohydrate (i.e., 1 to 2 or the proportion in milk); whereas an adult man, doing moderate work, requires 56 grammes of fat to 500 grammes of carbohydrate (or 1 in 9 only). The total required by the infant in the twenty-four hours is 30 to 45 grammes against 56 grammes by the grown-up person: so that the infant needs from one-half to three-quarters as much fat in its food as an adult individual. On the other hand, the child requires less starch. The maternal milk perfectly supplies the child's requirements in these particulars. All the scientific witnesses in the report of "Physical Deterioration" agree that rickets is caused by defective digestion and improper feeding, especially with artificial foods. The affection is not infrequently not due to actual want of food, - for there is often an abundance of the wrong sort of food, - but to a want of nourishment of the proper kind. In other words, it is a question of quality, not of quantity: so that the production of this disease must be referred to a special, but not a general, dietetic incorrectness.

As substitutes for the maternal milk a large variety of so-called foods have been employed, the commonest perhaps of these being cow's milk diluted with water or gruels, condensed milk, milk with farinaceous substances, and patent foods alone, including desiccated milk. It is not often that we find such maternal substitutes conforming to the composition of that important form of nourishment. Cow's milk contains 3 per cent. of proteid, 4 per cent. of fat and 5.1 per cent. of carbohydrate; whereas the percentages in the case of human milk are respectively 1.5, 3.5, and 6.5. The milk of the cow, therefore, is richer in proteid, but weaker in fat and carbohydrate than human milk. For the purpose of the reduction of this excess of proteid it is customary to dilute cow's milk with water; and for the supply of the deficient constituents cream and sugar are not infrequently added. There are a number of old-established formulae for mixtures of cow's milk and water - e.g., one of milk to two of water; equal parts of milk and water; two parts of milk to one part of water. Such would undoubtedly correct the proteid, but lower the fat too much. In the four examples just given the proteid, fat and carbohydrate proportions are respectively - 3, 4, 5; 1, 1.3, 1.6; 1.5, 2, 2.5; and 2.2, 2.6, 3.3. Although the fat is deficient in all these mixtures, not a few infants thrive very well thereon and become ailing only when the milk receives carbohydrate addition. Even then, the child at first fattens and is apparently doing well; but the fat so formed is of a poor quality and the muscular tissue weak: so that, sooner or later, symptoms of rickets make their appearance. Unfortunately, the mother only seeks advice when the child fails to walk or if, when able to do so, the

bones bend beneath its weight. Condensed milk is not without its uses in cases of **emergency**; but, if its administration be continued for a long time, the child becomes weak, anaemic, fat and flabby, with a tendency to suffer from respiratory disease on comparatively trivial excitation. This substance contains a large excess of sugar and is deficient in the antiscorbutic element. In order to reduce the sugar it should be used very freely diluted. As a rule, the directions on the tin result in its being given too strong; **in infants** the **proportion** is 1 of the milk to 24 of water, which dilution would make ^{from} 13 per cent. of fat, 2 per cent. of proteid, 1.6 per cent. of fat and 8.17 of carbohydrate. The proteid, fat and sugar figures for the neat milk would be respectively, in the sweetened article, 10, 13, 52; in the unsweetened 9, 11, 15. Adding 1 part water to the sweetened, we would have 1.2, 1.6, 6.5. Adding 6 of water to 1 of the unsweetened condensed milk, the figures for the three elements would become 1.2, 1.5, 5.6. In order to render this kind of milk capable of digestion, it must be well diluted; and in cases in which it has been, according to my experience, successful the cow's milk was used in a strong mixture and given up as a failure before trying it in a weaker strength, which would probably have been tolerated by the patient. The amount of casein in the diluted condensed milk mixture is small and the precipitate of casein, formed on the addition of acetic acid, is about equal to that of peptonised milk. As bearing on this question of feeding with condensed milk, we may note that there is the element of great unreliability in the use of the domestic teaspoon as a measure in the home. It seldom measures exactly a drachm and the amount taken varies considerably, according to whether the spoon is dipped into the tin or the milk carefully poured out until level with the edge of the spoon. Actual experiment will show that the average teaspoon, holding two and one-half drachms, if carefully dipped into the tin, may hold on withdrawal half a drachm more than that; the larger kind of teaspoon may be made to hold four drachms; but only two drachms will be held if the milk is poured into the spoon and none is allowed to adhere to its under surface. In actual practice it will be found that condensed milk is not easy to work with scientifically and that an infant, fed on a mixture thereof and water, will be getting too little fat and too much sugar. In short, it is well nigh impossible to construct an absolutely reliable mixture that can efficiently replace the natural secretion of the human mother's mammary glands.

It is interesting here to note that a disease bearing a close resemblance to human rickets may arise in the case of certain animals fed on improper food and housed under insanitary conditions. Crisp (Trans. Path. Soc, xxviii) tells of the bones of pheasants that had been confined in a small space and were unable to obtain natural food. The bones in question showed softening and distortion. He found that London-bred poultry, inadequately supplied with good food and air, often suffered from deficiency of phosphate of lime in their bones, as evidenced by bending of the breast-bone and permanent twisting. Rickets would appear to be somewhat common amongst young dogs fed on large quantities of animal and vegetable food; and the malady is particularly prone to attack lambs in cold and bleak situations. Anyone can perform the experiment of feeding puppies for four of five months on meal, keeping some of the litter under normal conditions as a control; it will be found that the latter will grow up strong and healthy, the meal-fed ones invariably rachitic. Tripiier performed such an experiment as this, but substituted cats, dogs and chickens for puppies - with the result that all the animal which were fed with meal only died, though rickets was not produced. Baxter gave starchy food to animals; they all developed a condition of severe marasmus and died therefrom without any rachitic sign coming under observation. The diet of these animals consisted of pure arrowroot-jelly plus a variable quantity of milk. The starch soon passed through the intestines unchanged and there was speedy production of a condition of inanition. The bones participated in the general atrophy, becoming thinner and frail, though the epiphyses showed no rachitic lesions. Our author blamed for his failure to cause rickets the difficulty which he experienced in keeping the animals

alive long enough to occasion the disease; and he doubts that all cases of so-called rickets in lower animals are the same as that affection as seen in the human being. The tendency on the part of certain animals in the London Zoological Gardens to develop rickets has ever been a source of anxiety to the Fellows of the Society owning them. The keepers blamed the clay soil and the coldness of the location in Regent's Park. Bl - and Sutton, however, noticed that young monkeys, deprived of their mother's milk and fed upon vegetables, became rachitic and that young bears, on a diet of raw meat, rice and biscuits, suffered from weakness and bone softening and bending, with death ultimately from extreme rickets on persistence with this erroneous diet. For a long time it was found impossible to rear the lion cubs whelped in the Gardens; the usual tendency was for them to take rickets and succumb. They were then given fed on raw meat, tough and deficient in fat, with old marrowless bones so hard that the adult lions could not chew them up. This diet, which was practically a starvation one, was continued, milk, cod-liver oil and pounded bones being added on Sutton's advice. In three months all signs of rickets disappeared and the whelps became strong and healthy. It will be observed that the only change made was in the food: so that the keepers' soil and climate theory of production was disproved - especially in view of the fact that the animals remained in the same cages and under identical conditions of air, light and warmth, both artificial and natural. Now, the successful treatment of these animals by diet alone support, in a very striking and remarkable manner, the theory that the deficiency of fat, and possibly also shortage of proteids and lime salts, must be looked upon as the chief agent in the production of this disease. An American observer, Herter by name, once tried to excite rickets in pigs by fat starvation. A pig was fed, for fifty-one weeks, on a limited quantity of skim-milk containing a 1/40th per cent. of fat; as the milk of the sow averages 8 to 10 per cent. of fat, the animal received only 1/300 to 1/400th the normal amount of that constituent. By the end of the above-named period under this diet, the pig had gained 16 pounds; its skin was dry and it suffered from great debility, weakness and constipation. Another pig, fed on an unlimited quantity of the same milk for twenty weeks, also developed muscular weakness and drowsiness. A third pig, fed for fifty-six weeks with skim-milk and carbohydrates, became very weak and had occasional diarrhoea attacks. Several other pigs were subjected to experiment, but our author found that the bones and other tissues presented no rachitic lesions. The fat of the adipose tissue was replaced by a mucoid material; but this change was not found when the animals received the addition of sugar to the milk. His conclusion was, therefore, that fat starvation does not cause rickets. That this is correct, in the case of pigs, seems fairly well proven; but in the case of the child fed on skim-milk that came under Cheadle's observation, there is strong evidence that fat starvation of human infant tends to cause rachitic disease.

The observer in question tells of a child living in the country; as the father was a domestic servant in the employ of a wealthy person, the supply of milk available was practically unlimited. Such copious milk the child digested without difficulty. Nevertheless, it developed rickets; and inquiry showed that the milk was merely the skim variety, the cream having been removed before the father was given the milk. In addition to this skim-milk, the child received farinaceous food; it was therefore under a diet in which fat was almost absent. The child ultimately recovered from the disease when fat was added to its food.

I, too, have found that the addition of fat to a rickety child's nourishment in the vast majority of instances cures the affection - not seldom, too, without any necessity for recourse to medicaments or other things. The lesson which I have learnt from such practical experience is to effect improvement or cure by giving rachitic children cod-liver oil with their food; if raw meat-juice be added also, they almost invariably do very well indeed. Although it is not altogether wise to ignore the other factors which may operate in the production of the disease, - such as heredity, cold, bad air, sunlight deficiency, and so forth, - it would seem that, in general, the

addition of fat to the diet,- especially if proteid be introduced at the same time,- will cure the malady. Nevertheless, it must not be forgotten that there is some danger in feeding an infant on excessive fat. Administration of food too high in fat, if rapidly inaugurated or too prolonged, may give rise to dyspepsia, intoxication and diminished absorption of fat; in this way we may have rickets actually occasioned by a diet intended to cure or prevent its origination. Cream, especially dairy cream, not infrequently contains an excess of fatty acids, giving rise perhaps to dyspepsia, weakness of auto-intoxication. Milk too rich in cream can give rise to gastritis, vomiting and diarrhoea with large oily motions, or constipation with hard, crumbling stools full of fat, and ammoniacal urinary secretion. The child continues to lose flesh and rickets is now very apt to occur. Absorption of the proteids is interfered with and thus an additional danger appears on the scene. The ingestion of a high fat percentage is especially apt to occur with the cream and whey, or top-milk mixtures so commonly employed in these times of modified cow's milk dosing. I have knowledge of four such cases, two of which presented such signs of rickets as craniotabes, delayed dentition and head sweating, whilst another had convulsions in common with these two, but nothing else suggestive of the disease.

The starchy and sugary constituents of the diet, classed as carbohydrates, perform a very important part in the economy of the system. Such substances, when converted into fat, constitute an efficient source of body heat; still they are unable to make up for nitrogenous waste and are not operative in the building up of cellular material. Carbohydrate is present in the milk as lactose. Starch is not a constituent of milk, but an element foreign thereto and useless in the early period of infancy. The ptyalin ferment is not present in the infant's salivary secretion until it attains the age of two months and there is no diastatic ferment in the pancreas until the end of the first year of life. Numerous experiments have been performed with a view to determining the existence of diastatic ferment in the stomach and ptyalin in the saliva of infants, aged from sixteen days to two months. The result of such is the conclusion that the saliva of very young infants contains a diastatic enzyme capable of converting small amounts of starch into maltose; that the diastatic action of the saliva may continue inside the stomach for two hours after food ingestion; and that, on pathological grounds, there is no reason why infants cannot digest small amounts of starch. But starch and maltose, the principal constituents of most patent foods, are not found in the milk of any mammal; and, although infants do well on dextrinised gruels, it is possible that they possess a mechanical, rather than a nutritive, value by assisting the digestion or modification of the curd in the stomach. The best results are obtained if the amount of starch is small,- under 1 per cent.,- and the effect upon the curd is lessened if the starch is converted into dextrin and maltose. A diet of carbohydrates may lead to rapid increase in weight; but the strength gained is not proportionate and the musculae tissue is flabby. Infants thus fed have feeble resistance and many of them become rachitic. The easy digestion of a food consisting chiefly of soluble carbohydrates, and the rapidity with which children gain weight, not infrequently convey a false impression of physical progress to the child's parents and medical attendant. A diet containing excess of carbohydrate, and particularly if possessing a shortage of proteid and fat, will cause malnutrition if persevered with. The beautiful photographs of fat infants, which one sees so often on the hoardings and in the papers nowadays and supposed to testify convincingly to the merits of certain patent foods, are apt to create a false impression of the possibility of healthy infants being reared in this way, despite the fact that the child concerned is, though fat, actually unhealthy and very likely in a rachitic state, the same perhaps concealed by judicious posing to the camera. Thus, Cheadle tells us that a fat baby once won the first prize at a baby-show, but was not long afterwards brought to his hospital for the treatment of rickets of an advanced character. Carbohydrates enter into the composition of human milk as lactose, usually to the extent of $6\frac{1}{2}$ to 7 per cent. The proportion of proteid to carbohydrate

for a child is 1 : 3 and for an adult 1 : 5; the fat to carbohydrate is in a child 1 : 9, in adults 1 : 5. The infant requires more fat and less carbohydrate than the adult. This condition is not fulfilled by most of the patent foods, which show a great deficiency in fat. The fat percentages in various foods is as follows: Dried human milk, 26.4; Allenbury No. 2, 12.3; Horlick's Malted Milk, 9; Mellin's Food, a trace; Benger's Food, 1.2; Allenbury's No. 3, 1; Mosley's, 0.92; Neaves's 1. Some of the foods of this class, - e.g., Mellin's Benger's and Neaves's, - are recommended to be made with milk; whereas others, - e.g., Allenbury's No. 1 and Horlick's, - are sold to replace milk and are usually dried preparations of cow's milk variously modified. The following analyses were made of foods prepared with a milk of 1032 specific gravity, 3.6 of proteid, 4 of fat and 5.37 per cent. of lactose: (1) Neaves's Food has a basis of oat flour and contains free starch; the dry food contains 12.10 of proteid, 0.86 of fat and 82.03 per cent. of carbohydrate; mixed with water, the respective figures were 0.78, 0.005, 5.40; somewhat vague directions are given for its use with milk. (2) Benger's Food is pancreaticised wheaten flour; it contains free starch and 48.5 per cent. of soluble matter. The percentage composition is, for the dry food, proteid, 12.48, fat, 0.86, carbohydrate, 76.91; when prepared with 1 part milk and 2 parts of water the figures are respectively 2.1, 1.8, 6.2. (3) Frame's Food is an admixture of malt and biscuit. Analysis of the dry product shows 19.86 of proteid, a trace of fat, 76.54 of carbohydrate; when mixed with 1 part of milk and 2 parts of water, the figures are 4.5, 1.1, 14.49. (4) Mellin's Food shows a reduction of starch to almost pure dextrin; for a child of three months it is directed that use be made of three level teaspoonfuls of it to eight tablespoonfuls of milk and water. So served up it contains 1.63 of proteids, 1.22 of fat, 5.36 of carbohydrate, as compared with respectively 10.01, 0.08, and 78.86 in the dry condition. (5) Allenbury's Foods. - No 1 is a dried modified milk, No. 3 a malted preparation containing starch. No 2 contains milk and deposits fat on standing; the dry food contains 9.86 of proteid nitrogen, 15.04 of fat, 68.85 of carbohydrate; when made up according to directions the figures are respectively 1.71, 2.49, 11.46 per cent. Despite the commendations of the vendors, such proprietary foods are unable to replace cow's milk by approximating more closely to human milk: for they are generally deficient in fat and often contain an excess of carbohydrate - principally in the form of free starch, which during the early period of its existence the infant is unable to digest. Moreover, they are lacking the antiscorbutic element; they are usually very troublesome to make up and expensive to patronise. Indeed, it is difficult to see what real advantage they have: for if carbohydrate were required in the food, it is far easier to supply it as oatmeal or barley-water. It is, however, true that they are of some use in emergency, especially during the summer months, when it may be difficult to secure pure clean milk and diarrhoea is prevalent. Their prolonged exhibition is dangerous to growing infants, especially as at first the latter may gain in weight and for months present no undesirable symptoms. In other words we may take it as a general principle that the suitability of a particular food is not proved by the fact that it is taken well and productive of no ill effects, as it may be many months before there is any manifestation of rickets, scurvy or other untoward results. As the onset of rickets is so insidious, and as many months may elapse before symptoms pointing to the disturbance of the osseous system draw attention to the presence of the disease, this delay in the appearance of starch-diet ill effects should during infant feeding be carefully remembered. Many opportunities for practical observation have convinced me that, though symptoms may be slow in developing, when once present are of very serious import, particularly if there be present such symptoms as dryness of the skin, muscular rigidity, difficulty in free movement, weakness of the heart and loss of appetite. I have always found that starch-fed children have a low resisting-power to disease; they recover from illness very slowly and rapidly succumb to prevalent ailments or to auto-intoxications. Even with rosy complexion and no digestive disorders, a child may have its

constitution undermined by improper feeding with foods of this class. The weight of the child may be no reliable index of its vitality or physical progress; therefore careful watch should be kept over the condition of the skin and muscular tissues. The exact rôle which carbohydrates play in the production of rickets is not quite clear. The child, nourished on carbohydrate excess, is easily affected by digestive disorders and its power of resisting adverse influences is much below par. Trophic ailments, once initiated, are not easily overcome and rickets may be the outcome thereof. There is no doubt that any disturbance of the dietetic ratios affects adversely the organism; and, if one is deficient or in excess, the other elements are not so advantageously dealt with or assimilated. Although it has been suggested that rickets is not infrequently due to fat shortage in the food, it may also be caused by a diet sufficient as regards fat, the assimilation of which latter is interfered with by an excess of carbohydrate. Too much sugar, soluble or insoluble, will stand in the way of the proper assimilation of the fat; therefore, a diet, poor in fat but sufficient to prevent malnutrition, will, in the presence of carbohydrates, be too weak, the child suffering from trophic disorders and rickets. If the latter affection is occasioned by fat shortage, it is probable that, in the case of an infant six to seven years of age, a mixture with 1 to 5 per cent. of fat will not be enough to prevent its appearance; further, if there is too much sugar in the dietary, 2.5 per cent. also would not be sufficient. The three important constituents of the food, then, namely, proteid and fat and carbohydrate, are intimately associated with one another. In searching for the cause of the disease and the lines on which the treatment thereof can best be conducted, it should not be forgotten that, though the main evidence points to deficiency of fat in the diet, the proteid constituent has an important part to play in the child's nutrition and that excess of carbohydrate may interfere seriously with or hamper the proper absorption of the other elements, even though growth and nourishment of the child and rachitic invasion might be considered as adequately catered for by the amounts of that substance arranged for operation.

It seems difficult or impossible at present to estimate the true value of the ferments which are present in the milk of all mammals in addition to proteid and fat and carbohydrates, salts and lecithin. The child at birth has a stomach that is deficient in digestive ferments and probably also in those which are called trophozymases and which may be supplied in the mother's milk. Unless these are present, the infant cannot digest and assimilate its food and finally transform it into cell tissue. These ferments possess some biochemical action, which is probably rendered inert when the milk is boiled for sterilisation purposes. All animals do not possess the same ferments; but, with special foods or by feeding them with the ferments absent in a particular species, the missing elements can be made to appear in the milk. More than a decade ago it was demonstrated in Concetti's laboratory (Arch. de Méd. des Enf., Mar., 1902) that human milk contains ferments which are absent from or only slightly present in the milk of the cow and goat; also that sterilisation destroys any ferment that may exist. The ferments present in human milk are not peculiar to it, but under appropriate feeding can be made to appear in the milk of the cow. Thus, Spolverini (Ibid., 1901) fed goats and cows on a germinating barley and subsequently noted the appearance therein of amylolytic and hydrating ferments which were not present normally, but which are always present in human milk. Our author classifies the ferments of milk as follows: First, the Ferments of Elimination; second, the ferments of secretion. Under the latter category he includes: (a) Trypsin ferments - active in the goat, dog and cow, less active in woman and the ass. (b) Pepsin ferment - less active than the foregoing. (c) Amylolytic ferment - never found in cows or goats, but always found in women and dogs, in whom it is highly active. (d) Hydrating ferments - transforming salol into carbolic and salicylic acids, always present in woman and dog, but absent from the milk of the cow. (e) Lipase - a fat-splitting ferment present in all milks. (f) Oxydase - very active in cows and goats, scarcely noticeable in woman. (g) Glycolytic ferment - present in all animals in varying degrees of

activity. Milk must therefore be considered not only as a mixture of nutritious chemical substances, but also as containing active biochemical elements operative in the building up of the tissues of the system. For some time past, owing to the difficulty in obtaining reliable and pure cow's milk and the dangers associated with that liquid when impure or even slightly tainted, a large section of the population have had recourse to the use of what is termed sterilised milk, the method also having been adopted for general distribution amongst the poor wherever municipal laboratories exist. It would seem, however, that the advantages of this form of feeding infants are doubtful; for the spores of the more virulent microorganisms are thereby not destroyed, though probably the useful varieties - the so-called "tame microbes" - are rendered inert and the milk damaged. There is always the risk that a child reared on sterilised milk will fail in development and become more or less scorbutic. I have seen several such sterile-milk-reared children eventually lose firmness of flesh and vigour and fail to acquire the normal robustness of constitution. The sterilisation process seems to impair the value of the nitrogenous element and interfere with the proper performance of the antiscorbutic function; the heating of the milk probably also kills the ferment present. Owing to the difference of good opinion existing, I would not go the length of affirming that the use of sterilised milk does cause rickets, and refuse to subscribe to the old German theory that scurvy is an acute form of that disease; for although these two affections are not infrequently seen together in the same patient, the symptoms are quite distinct and the treatment indicated must be conducted on essentially different lines. The outstanding characteristic of scurvy is the tendency to subperiosteal hæmorrhage, subcutaneous extravasations, sponginess of the gums and hæmaturia - such hæmorrhagic expressions not being observed in rickets. The alteration and correction of the diet can produce a slow and gradual improvement in the rachitic child, the administration of antiscorbutic remedies effecting no change; whereas, in the case of scurvy, the addition of fresh vegetables or fruit juices to the diet will lead to improvement forthwith and ultimate recovery. The occurrence of these two maladies at the same time is owing to the fact that the foods are given sterilised, or that the diet consists of a sterilised or patent substance lacking the antiscorbutic principle and deficient in fat, proteid and phosphates. It seems possible that the periosteal activity and the vasculatity of young growing bone may also be a factor in the contemporaneous present of scurvy and rickets. Though it is doubtful whether sterilised milk can produce, de novo, rickets, it is possible that a child, nourished on an inert food lacking the digestive ferments and enzymes present in fresh milk, will grow up less resistant to the various catarrhs and that rickets will appear as a sequel to these disorders and chronic malnutrition. If, in addition, the milk is deficient in fat and proteid, the tendency for the child to develop rickets will be very much augmented. Comby (Brit. Med. Assoc. Meeting, Manchester, 1902) holds that modified artificial milks are responsible for the production of anaemia, rickets and infantile scurvy, he having noticed that these affections, especially the last-mentioned, appear in those countries where modified milk enjoys the largest vogue; further, in America, where modified and sterilised milks and foods have a tremendous popularity there is an enormous prevalence of these diseases. Still (Med. Rec., Dec., 1902), in one of the American Dispensaries, observed 179 consecutive cases fed, for periods varying from three to eighteen months, on carefully modified laboratory milk, all bottles containing it being carefully sterilised. For nine months of the year the milk was pasteurised, a process of simple sterilisation being practised during the summertime. In 97 of the cases there were unmistakeable signs of rickets or scurvy, most of the cases being instances of the former affection or a combination of it with scorbutus, the one merging into the other. For the production of these juvenile affections he blames the pasteurisation of the milk. Although scurvy and rickets are commonly associated under certain circumstances, there are many who doubt that they are really connected and insist that they are not dual expressions of the same disease. The American Pediatric Society have given much attention to the subject of

the alleged association between rickets and scurvy. Out of 340 cases it was found that in 152 (45 per cent.) rickets was present. In 50 of the patients that disease antedated the appearance of the scurvy. Dealing with the subject of infantile scurvy, Aussât (L'Echo Méd. du Nord, Jan., 1903) concludes that the same is a haemorrhagic form of rickets; that all children affected thereby are rachitic, with or without osseous deformities; and that the coexistence of infantile scurvy and rickets would indicate a similar etiological factor. He also tells of 682 cases which he studied, comprising 456 bottle-fed, 59 breast- and bottle-fed and 167 breast-fed. He thinks that it is an exaggerated view to hold that sterilised milk can cause rickets, but that it is the wrong and injudicious dieting which actually gives rise to the affection. A breast-fed infant becomes rachitic if bad hygienic principles cause prolonged digestive disturbance. He considers sterilised milk of great value and preferable to wet-nursing in the case of families able to afford the latter. The establishment of milk stations where that liquid is carefully dispensed and where mothers are advised how to accurately administer the same will help, our author believes, to effect a diminution in the number of rachitic cases observed. One of his contemporaries, Variot (La Trib. Méd., 1902), concludes that, - aside from mother's milk, which nothing can perfectly replace, - the use of sterilised milk is almost as safe as a wet-nurse. There are so many objections to the employment of that individual that he advises sterilised milk for a normal infant and reserves the wet-nurse as a last resource when the infant fails to thrive under other methods. The general opinion amongst the scientific officials of municipal milk depôts is that the use of sterilised milk does not make children rachitic. I have seen no case of genuine rickets which I could refer to the use of sterilised milk, though I have seen a few cases of slight scurvy, but doubt whether the number which arise in this way is not less than under any other method of artificial feeding. I find that sterilised milk helps to prevent the occurrence of summer diarrhoea and believe that it is safer in hot weather than any other form of food. Beyond a suspicion that this kind of milk, especially if its use is prolonged, may cause the disease, my conclusion is that sterilisation in itself is not capable of making an infant rachitic. Nevertheless, this purifying process has some drawbacks. Thus, it has been found that its prolonged employment as an infant food lowers, or completely deranges, metabolism, throwing an extra strain on the organism, thereby paving the way for innumerable ailments; the nutrition of the body is inadequately maintained and the frequent result, therefore, is a constitutional disorder. We may take it, then, that though sterilised milk cannot be definitely regarded as causative of rickets, there is a possibility that feeding on a milk deprived of its vitality, its ferments and its energy, will lower the vital resistance and predispose to that disease. If three children were selected for observation, - a breast-fed infant, one artificially fed with fresh cow's milk and another given sterilised milk, - the last-mentioned, - assuming ~~the~~ healthy at the commencement of the test period, - would suffer most in nutrition and probably become scorbutic or perhaps rachitic or both. It is therefore advisable to bear in mind the anomalous constitutional conditions to which the use of sterilised milk or sterile patent foods may lead, and that the dangers of either will be largely increased if they prove deficient also in the all-important proteid and fat. For proper infant feeding the food should be fresh and free from dangerous organisms, the vital principles - the ferments - being also unimpaired. Such desiderata exist under ideal conditions in normal breast-milk; if, unfortunately, cow's milk must be used by way of a substitute, no pains should be spared to obtain, if possible, clean milk that does not call for cooking or sterilisation or any form of treatment that will injure the vital principles named.

From the foregoing facts, which I have marshalled in the briefest possible manner consistent with their importance, I would now draw the following conclusions: Despite the undeniable fact that the faulty nutrition of the infant is the great underlying excitant of rickets, as new facts arise bearing on the great differences between human and cow's milk it becomes more and more evident that it is unwise to point to one element in the food as the sole or principal originator of the disease.

Laboratory experimentation will not settle the moot question of the causation of this disease; for test-tube reactions are not identical with the conditions obtaining in the human and living stomach. I very much doubt also that experiments on animals throw much light upon this subject; for animals and men react differently to the same reagent. There is very little really known regarding the chemical nature of the human mammary secretion, or how it differs from the same liquid in the case of the cow and other mammals. We do know, of course, that the proteid substances, casein and serum albumin, differ, both in nature and proportion, in cow's and human milk and, moreover, we know that the fats differ, - the milk of the cow being relatively rich in fatty acids, whereas human milk is poor in these constituents; we know, further, that both contain milk sugar and salts in varying proportions; - but it is impossible as yet to explain the vital principles hidden in the human secretion - be they ferments, enzymes or antibodies, which render human milk, taken into the system of an infant, an animating liquid, while the milk of the cow or other mammal may, under certain conditions, become a poison as lethal as virulent organisms. It is therefore necessary to study the action of infants foods from a biological, as well as from a chemical standpoint, remembering always the important principle that the nutriment is intended to act as a food and to aid in the evolution of the alimentary functions: so that, as the digestive secretions of the infant's stomach and intestines are gradually formed and brought into operation, they may act chemically upon the milk and retain it for lengthening periods in the stomach, thereby preventing its being passed on too quickly into the intestines. By this process the stomach is gradually enabled to fit itself, anatomically and physiologically, for the food of the adult when the child grows up. Human milk retains the same composition throughout lactation, but is changed by the action of the developing gastric secretions into forms requiring slight, moderate or complete gastric digestion - until at last the animal is able to digest and assimilate the food of the mother and can be considered physiologically independent of the that individual. I do not think that there is so much danger in deficiency of one element as in the disturbance of the ratio of all the important constituents of nutrition, that is to say, the sugar, the fat and the nitrogenous material. It would seem that the disease under consideration is caused by a food usually deficient in quality, though the quantity may be ample to support existence and be even able to make it put on considerable apparent flesh. An infant nourished by the mother herself secures a milk in which the constituents just mentioned preserve a tolerably constant and proper ratio. Towards the end of the period of lactation, in pregnancy occurring during the presence of that function, and in the case of ill-nourished mothers, the proteids fall to vanishing-point and, even though the fat may have varied little, malnutrition and rickets may break out in the child. If the infant is being fed on the milk of the cow, that liquid may be ingested in a condition of fat shortage, or that substance may be so excessive that it interferes with the process of proteid assimilation. The result is that digestive troubles appear and, though the child seems to be pretty well nourished, gradual wasting and rachitic expressions may be observed. The excess of sugar in condensed milk, together with the fat deficiency of the same, may likewise prove an improper dietary, with culmination in flabbiness, weakness and rickets. But the worst dietetic offenders are the patent farinaceous so-called foods, which are rich in carbohydrates, soluble and insoluble, and very often provide excess of starch, which in itself is an unnatural food for infants, hinder the absorption of sufficient fat by the infant, which, slowly but surely, lapses into a rachitic condition, although for a long time previously it may have been fat and well-nourished, strong in digestion and apparently doing very well upon the diet in question. My opinion, therefore, is that, though we should not despise entirely the theory that fat shortage tends to the production of rickets, no one constituent of the food should be unduly emphasised to the exclusion of the rest. The sugar, the fat and the proteids, and in a lesser degree the salts, are so intimately associated that any disturbance of the balance inevitably causes digestive disturbance, malnutrition and not seldom the disease we are considering. At the present time it is not easy to explain why one infant

should become marasmic and another rachitic or the subject of scurvy disease. It may be that marasmus, scurvy and rickets are in some way related; and the special disease is determined, perhaps by different agents, - microbic, scorbutic and toxic, - conveyed in the food or are at work in the system already. The alleged production of rickets by the presence of toxins and the contemporaneous shortage in antitoxins will receive attention presently. We shall see that it is affirmed that these toxins would possibly supply an irritant to the blood stream and give rise to disturbances of the organs most concerned in the osseous lesions of the disease; cold would assist by lowering vitality, and so give the toxins more ~~shape~~, and lack of sunlight and fresh air would act pretty much in the same way. The germ theory of rachitic toxæmia has been assailed; and it would seem that the toxin involved is not the product of germ like, but originating in some poisonous element of the milk, which the child's antitoxins are capable of resisting when naturally and properly fed. If this be true, over any lengthy period it is not safe to feed an infant on synthetic food stuffs, which obviously could not approximate the maternal mammary secretion in preciseness of ingredients.

It seems apposite at this juncture to consider the interpretation of some interesting experiments which Chalmers Watson (Lancet, Dec. 18, 1906) performed upon some female rats, the progeny of an excessively meat-fed mother. For some months prior to delivery and during the period of lactation these animals received a diet of ox flesh and, after weaning, the young ones were similarly dieted. These meat-fed rats numbered 100 and in age varied from a day to twelve weeks, and control rats were fed on breast and skim-milk, with an allowance of bread. After a certain time they were killed and submitted to a minute examination. There was found a general softness of the bones of the body, a condition invariable in all meat-fed rats and accentuated as they grew older. The long bones, especially the ribs, were of a dark colour from increased vascularity of the tissues. During the second month of the experiments curvature of the spine and long bones developed - such comprising scoliosis, lordosis, bending of the ribs at their angles and, to a lesser degree, curvation of the bones of the lower extremities. The osseous curvatures were not infrequently associated with enlargement of the cartilages. In some 15 per cent. of the animals small white nodules were observed on the ribs, standing out clearly as pale and bead-like prominences in the substance of the darker part of the bone. On cutting across these, it was seen that they consisted chiefly of cartilage. In pronounced cases the changes were similar to those seen in advanced cases of rickets in the human subject. But this likeness was not confirmed on microscopical examination; for, although it was seen that ossification was delayed and imperfect involving endochondral and periosteal bone formation, the minute structure of the epiphyses was normal, even though the animal showed pronounced rachitic changes in the skeletal system. The bony trabeculae were thin, there was great increase in the size of the medullary canal and the bone marrow, - in the case of meat-fed animals of six weeks and onwards, - showed a great increase in fat. In not a few there was a considerable increase in the number of thin-walled vessels, which were full of red blood-corpuscles in the medullary spaces. The white nodules observed in the ribs consisted of cartilage cells, derived from the periosteum, and transformation of these into bone was seen at the peripheral part of the sections. The first impression conveyed by these experiments was that there was a condition of rickets produced by the meat diet; for the delayed and imperfect ossification, the increased vascularity, the augmentation of the number of the blood-corpuscles in the medullary spaces and the costal cartilaginous nodules resembled the conditions observed in the case of human infants suffering from rickets; but the fact that the epiphyseal ends of the bones were normal showed that some condition other than rickets must have been in operation - an observation which would seem to cast some doubt upon Ghérin's statement that he produced rickets in animals by feeding them upon a diet of raw meat, and allow us to urge only an apparent similarity of conditions upon superficial examination. Our author refers to a case in which a consumptive mother, - treated successfully with considerable quantities of meat, - married, continued the same diet and in eighteen months

was delivered of a child. It was brought up on the bottle until the age of one year and then, for some overt reason, did badly and became anaemic. Raw meat-juice was administered, but matters became worse and death occurred. The symptoms observed during life were not unlike those seen in meat-fed rats and after death the tissues, particularly the radius, showed osseous anomalies very similar thereto. This case demonstrates the great influence that the diet of the mother during pregnancy may have upon the offspring, which fact should always be borne in mind.

The alleged deficiency of lime salts in the rachitic subject has for long been a matter of controversy amongst perinatrists, especially as the most obvious anomalies occur in the the child's bones. The older writers sometimes referred to a shortage of mineral matter, particularly of lime salts and contemporaries and others held that rickets was induced thereby. It is very common to find that rickety children are overfed on unsuitable food, in which it is probable that they receive enough phosphate of lime, but are unable to assimilate it from digestive disturbance resulting from their gluttony. Many experiments have been performed with the object of throwing light upon this important question; and it is necessary to consider here as briefly as possible some of the more instructive and important. Thus, Chossat, in 1842, advocated the theory of the absence of certain bodies in the food, as against those who insisted upon the presence of some acid, or acids, in the circulation of the patient. He fed pigeons on food deficient in lime salts, with the result that he was able to produce in them a condition in which the bones were readily liable to undergo fracture, or were softened and curved; he was, however, not able to excite rickets, merely, it would seem, some osteoporotic condition. Friedleben repeated these experiments and found that the bones were undoubtedly more fragile than normal, though he could not convince himself that rachitis was present. Voit was another to repeat the experiments in question, in 1880, with the result that he concluded that rickets could be so produced. Baginsky did much original research of this kind and stated that the administration of lime-free food gave rise to rickets; he adds that the changes in the bones were more marked when lactic acid was added to the dietary. In 1871 Wegner, of Berlin, demonstrated that, if while administering doses of phosphorus to young animals, lime salts were withheld, there arose an affection of the bones exactly like rickets; his conclusion was that phosphorus acts as a stimulant of the ossifying process. An all-round view of this question would seem to show that the lime-salt deficiency theory of rickets causation can be much weakened by such facts as the following: Despite the fact that cow's milk is six times richer in lime than human milk, breast-fed infants rarely suffer from rickets, whilst amongst infants artificially fed and taking cow's milk in some form or other the disease is very prevalent. Nevertheless, it is possible that the infant is unable to use effectively the lime salts in the cow's milk: for the calcium salts are intimately associated with the albumin of the casein, and it is this substance that the infant finds difficulty in digesting and not infrequently rejects as tough curds. Secondly, in limestone districts, where the water may be impregnated with lime salts, the children are frequently rachitic, and the addition of lime-water to the food does not prevent the occurrence of the disease. It was formerly thought that rickets in the Glasgow children was due to the want of lime in the Loch Katrine water-supply; but, although this water is known to be almost distilled water, the tendency now is for the affection to disappear. The Commission on Physical Deterioration did not consider that the want of lime in water can make anyone become rachitic. So good an authority as Ormsby was inclined to believe that in cities with a water supply containing little lime deformities of the limbs are much more prevalent; yet there is very little lime in the water supply of Dublin and rickets is a comparatively rare affection in that part of Ireland. - a fact probably referable to the unpopularity of artificial rearing of infants. It appears, then, that the presence or absence of lime in the drinking-water has practically no bearing on the occurrence of rickets in Glasgow or elsewhere. Thirdly, the administration of lime salts, alone or in medicine containing preparations of lime, seldom relieves or cures the disease. Fourthly, farinaceous foods are rich in lime and phosphoric acid; yet children fed with these substances, especially

under conditions in which milk is not provided, are very apt to become rachitic. This disease's changes, such as increased vascularity and cartilage proliferation, are sometimes seen in normal calcified bone. In the absence of a proper supply of lime salts marked proliferation would seem impossible, as uncalcified cartilage consists largely of lime. In the absence even of dietetic rearrangement, it is possible for a patient to recover from rickets spontaneously; the lime received may be sufficient, but a defect in its assimilation in existence. Pommer says, that although enough lime is ingested, it is not absorbed properly, owing to the diminished alkalinity of the blood, despite the fact that equally competent observers have demonstrated that the alkalinity of ~~that~~ liquid is not diminished in this disease. It appears much more likely that, if lime salts are assimilated in an insufficient amount, the loss is to be accounted for by the inability to digest the casein of the milk; nevertheless this fails to explain the occurrence of the malady in the case of a child becoming rachitic on a dietary that is entirely farinaceous. Kassowitz thinks that the disease is a chronic inflammation starting in the bone-forming tissues; he compares this inflammatory process to interstitial keratitis, or to the interstitial inflammation of the liver produced by phosphorus ingestion; he does not regard the poverty of lime salts in bones peculiar to the disease, as it is to be seen in every form of inflammatory osteoporosis, and adds that of more importance is the intense vascularity of the tissues engaged in the formation of bone, which he looks upon as the initial and principal sign of the disease in question. One of his experiments was (with the object of ascertaining if artificial congestion would lead to rickets) to bandage the limb of a growing animal for several hours a day and doing so daily for several weeks. After death he noted in the bones an increased vascularity which had prevented the deposition of osseous tissue and destroyed the bone already formed; further, there was a rickets-like cartilaginous proliferation. He therefore maintains that a hyperaemia of the bone gives rise to poverty of the inorganic constituents and that an inflammatory process in the bones is the cause of the rachitic lime-salts shortage. Mirva and Stoltzner have also done some important ~~research~~ upon the effect of a diet poor in lime in this disease. They fed a six-weeks puppy for 55 days on raw horseflesh, fat and distilled water. After three or four weeks there developed typical symptoms of rickets, - viz., enlargement of the epiphyses, rickety rosary and curvature of the long bones, - but the structural alterations were more of the nature of osteoporosis than of rickets. These experiments were repeated by Reimers and Bayer (Med. Review, Vol. lx), who dieted ten-weeks puppies with horseflesh, pork, veal, horse-fat and distilled water. On the eighth day the animals were indisposed for exertion, became more and more indisposed to move and cried out in agony on sudden movement. They waddled, the fore limbs assumed ~~handiness~~ and there was a "cow-hock" anomaly of conformation behind, with enlargement of the lower ends of the radius and ulna. At the end of 31 days the animals were submitted to post-mortem examination. There was found a considerable accumulation of fat, the muscles were unaffected, rickety rosary was present, as well as long-bone epiphyseal enlargement. The long bones, when dried and macerated, proved unusually light and had a rough and porous surface. They floated in water, which is contrary to what obtains in health. On longitudinal section, the compact substance was found to be exceedingly narrow, with a corresponding ~~widthness~~ of the meshes of the spongy tissue. The growing cartilage was wider than normal and periosteal changes were observed in certain areas. The adjacent uncalcified bony layer showed no signs of proliferation and an osteoporosis was essentially the condition observed. A puppy with spontaneous rickets was used by way of control. The naked-eye and minute changes were chiefly proliferation of the osteoid tissue and were distinct from those to which deficiency of lime salts gives rise. Our authors, therefore consider that lime deficiency is not the only factor in the production of true rickets and suggest that the other one is heredity; for, if both parents have been rachitic, the offspring are specially liable to the disease. They also point out that some children would seem more predisposed to the disease than others and that these, even though they may be breast-fed, do not always escape the malady; but, as a rule, predisposed children do not, in the absence of

artificial feeding, become rachitic. It seems to me that the changes found in these young animals represented an early stage of rickets which, if the diet had been continued, might have been followed by a proliferation of osteoid ~~natural~~. This view is supported by the fact that cases of typical rickets in man have been reported, in which the changes in the uncalcified layer of osseous tissue were slight and much less marked than those in the periosteum and cartilage. I have already shown how Chalmers Watson fed young rats upon an excessive raw meat diet and produced in them softening and curvature of the bones, with white nodules in the bony parts of the ribs in 15 per cent. of the cases, - microscopic examination of the epiphyses showing, however, that the condition was not rickets, although there was a great similarity upon mere naked-eye examination. The opinion expressed by experts at a meeting of one of the London learned societies was that these experiments showed that the lesions were not so much due to the excessive meat diet as to deficiency or interference with the absorption of lime salts, especially in view of other experiments consisting of feeding young dogs on flesh, fat and distilled water - these animals remaining normal for a time, then putting on weight, but gradually becoming weaker, showing mental decay and dying; after death there were found increased vascularity of the bones, irregularity of the cartilage cells at the epiphyses, formation of imperfect osteoid tissue and shortage of mineral matter; further, under this lime-short diet the changes were less marked in small than in large animals. I very much doubt also that excess of meat in the food is always the cause of rickets in ~~rats~~; for animals that are purely carnivorous are never so affected, nor do the Eskimo, who is a pure meat-eater, suffer from this affection as seen in Watson's cases. In these animals the lime-shortage seemed to be the factor at fault. From the observer just mentioned's experiments, then, we may conclude, first, that, though animal experimentation is very unreliable for comparison with the conditions obtaining in the human infant, a diet excessive in meat and deficient in lime salts is productive of a disease ~~characteris-~~ed by bone-softening, increased vascularity of osseous tissue and debility, comparable to the condition produced by Guérin and other observers and described as rickets. Second, that, though the naked-eye appearance, - as evidenced by the bending of the bones and the hyperaemia, - is indistinguishable from rickets, the microscopic examination of the epiphyses clearly proves that the condition is something quite different from that disease. In other words, it is an osteoporosis with which, in these meat-fed animals, we have to deal.

We have now to consider for a moment the lactic acid theory of rickets, premised to which may be the statement that, if a bone be submitted to the action of an acid, it undergoes softening and loses its lime salts; from this it was formerly thought that there might be some acid, acting on the osseous tissues, which was the real excitant of the disease; furthermore, as rickets is usually associated with much digestive derangement, it was concluded that the acid in question was in all probability lactic acid liberated by the fermentation and decomposition of food in the unhealthy, stomach not infrequently seen in this disease. Heitzmann was the great advocate of this theory and contended that lactic acid stimulates excessive growth by irritating the young ossifying tissues, thereby causing the production of premature bone; but others have suggested that the acid in question combines with the lime salts, gets rid of them by a process of solution and so gives rise to a lime-famine so far as the bones are concerned. Against this idea we may urge that the presence of this acid in the bones has never been demonstrated, although it has been found in the urine. Our author fed carnivora on lactic acid and also injected it under the skin, keeping them all the time on a lime-deficient diet. The result was that rickets developed, accompanied by catarrh in the respiratory and digestive systems. These experiments were repeated by others, but with negative results. Thus, a rabbit was been injected with 10 c.c. of a 1: 200 solution of the drug for several weeks, and with the disappointing result just mentioned. Against the supposition that this acid causes deficiency of lime in the bones and resultant rickets we may urge such further facts as the following: First, some children, even when fed on starchy food, have no digestive disorders capable of favouring a production of lactic acid, but deal with

the starch well and put on weight - ultimately becoming rachitic. Further, the rachitic condition can be cured, although the child is kept on farinaceous food, if certain substances deficient in the food, - usually proteid and fat, - are supplied. Second, even if lactic ~~acid~~ does exist in the blood, it will most likely be neutralised by the alkali present: so that there would be no injury inflicted directly by the starch. Third, animals, fed on food which does not lead to the production of lactic acid, may become rachitic, as did Guérin's young animals when suddenly weaned from their mother's milk to meat. Successive litters of lion-cubs at the London Zoological Society's Gardens were, as we have seen, fed on tough fibrous meat and given bones so hard that they could not be broken, and ultimately died from rachitic exhaustion. In both these cases there would be a deficiency of proteid and fat, but nothing capable of giving rise to excessive production of lactic acid in the stomach. It seems to be beyond conception that an acid can be carried in the blood to dissolve out the lime from the bones; experiments do not confirm the theory, which may therefore be dismissed without further comment.

The hydrochloric acid theory calls for only a bried consideration. Its presence in the stomach has been regarded by some as causative of the disease under conditions of diminution. It has been found to be scanty in rickety cases, its production depending upon the amount of sodium chloride taken in with the food. It is said that in Saxony the nursing mothers ~~ingest~~ very little salt and that, in consequence, their nurslings are very often rachitic. In view of this theory, Shaw (Arch. Ped., 1903) added salt to the food of every infant which came under his observation and holds that he was thereby able to reduce the incidence of the disease. Finally, it may be noted that Nobécourt and Vilroy (Rev. d. Mal. des Enf., 1904) found that the addition of common salt to the food of infants, - especially in 5-grain dailt doses, - caused them to increase in weight, - the best results being observed in very thin and premature ~~cases~~.

A phosphate of potash theory was advocated by Delacourt, in 1899. This substance he gave to dogs and saw rickets develop. Spellmann tried it on rabbits and pigs, both by the mouth and by subcutaneous injection; in every instance negative results were forthcoming.

All the chemical hypotheses which I have considered above seem to be incapable of explaining the hyperplasia of the growing end of the rachitic bone, which would appear to be a more important matter than the absence of lime salts in the tissue. They also fail to account for the deficiency of inorganic material; and I would here urge the improbability of any such theory providing us with enlightenment as to how the disease actually occurs. There seem to be not a few observers who believe that the changes in the bone are due to some inflammatory reaction, probably the result of a direct irritation in the growing ossifying tissue, which would react readily to an injurious influence and so produce the disease under consideration. The inference from this is that there may be toxins, - perhaps of bacterial origin, - circulating in the blood which, by their irritating action cause inflammatory reaction in young bone, delay ossification and thereby pave the way favourably for the development of rickets. In short, the tendency nowadays is to regard this affection as a general one caused by poisons or toxins absorbed from the alimentary canal; and this supposition would seem to be by no means opposed to the various clinical phenomena of the disease.

Exanthemata

I have not infrequently observed rickets develop during the convalescence ~~from~~ some such exanthematous affection as measles or scarlet fever. This is not to be wondered at in view of the debilitation of these affections and the general unwholesomeness of the surroundings and social conditions in which they so often occur. By such things as insanitary houses and bad feeding the general health of the patient is perverted, with the result that the disease is at full liberty to do its worst.

H e r e d i t y

From time to time in the past not a few observers have looked to hereditary influences as a possible or probable or certain cause of the somewhat obscure disease under consideration, - the theory in question receiving apparent enhancement from the fact that it sometimes happens that parents who have suffered from rickets in childhood, or in adolescence, beget children so affected, some hereditary constitutional weakness seeming to be handed down to posterity. Nowadays, however, there are few who attach practical importance to this supposition, it being generally held that, if both the parent and the child be rachitic, the cause must be looked for in such predisposing conditions as impure air, cold, want of sunlight, or early-life dietetic anomalies. I have not infrequently experienced difficulty in securing a trustworthy history from the child's parents; for, in the absence of deformities, it is not always possible to discover a former attack of the disease; further, I cannot subscribe to the formerly alleged constancy of this factor, ~~in this case~~ of the children of healthy parents who may become rachitic and, as a rule, the parents of rickety infants do not invariably seem to have been troubled with this malady. Pfeiffer thinks that the tendency to rickets is hereditary, the affection being actually excited from this hereditary predisposition by the shortage of phosphates in the maternal milk. Vogel states that he has often come across cases of rickets in the children of previously rachitic individuals, no prophylactic or remedial measures being capable of preventing the occurrence of the affection in these children. According to Jenner, it is very doubtful that infantile rickets can be produced by paternal ill-health; and Rittershain claims to have traced the disease to tuberculosis of the father, less often to tuberculosis in the mother. But it is more generally thought that it is not the germ of constitutional disease that is inherited - rather similar unhygienic habits acting upon both parent and offspring; the term "foetal rickets" has, however, been called in question, there being doubt in the minds of many as to there being any condition to which that name can be scientifically applied. Alcoholism, syphilis, tuberculosis, or any disease producing cachectic conditions in the parents might predispose to rickets by lowering the resisting-power of the child. Again, any of these factors might also impair the milk of the mother, or her existing debility might cause inability to nurse her infant and have recourse to highly objectionable artificial feeding; therefore, though the development of rickets in the child may be attributed to the ill-health of the mother, in reality the true cause would be found perhaps in the evil conditions resulting from improper food, unhealthy surroundings or inability to give the infant the breast. If, on the other hand the father had suffered from rickets or cachectic disease, the infant might have inherited a weakened constitution, with some inability to readily assimilate its food: so that these conditions may predispose to the occurrence of the disease. Indeed, it is only in a modified way that we can look upon heredity as a factor in the production of that ailment. Pini presents an analysis of 4,176 cases of rickets; 52 of them were apparently of hereditary production and there was consanguinity of parents in 86; but other very probable causes of the malady were also discoverable. Wilks (Trans. Path. Soc., xxxii) tells of a case in which a father, who had had rickets in childhood and mollities ossium in adolescence, had also two daughters who suffered severely from rickets. Though unable to state the condition of the mother or the arrangement of the dietary, it was thought that there was nothing unreasonable in the hypothesis that a constitutional tendency to the disease had been hereditarily transmitted. Jenner regards the affections as an actual diathetic condition akin to scrofula, tuberculosis or congenital syphilis, though much has been urged against this original idea. Seigert (Jahr. f. Kinderh., viii, 129) has given long study to the etiology of rickets and holds that heredity is a more important factor even than debilitating surroundings; he maintains that when there is a pronounced family history of rickets, the affection is likely to occur in the child whilst it is still at the breast; the mother hands it on to her infant and he regards as second in importance to hereditary influences diseases of the digestive and respiratory tracts and the moral condition of the parents. It is interesting to consider the four tables which

he publishes in support of his belief in the theory of heredity: In the first tabulation he includes 31 families with rickety breast-fed infants. With the exception of one case, the mother had had rickets or showed such signs of the disease as deformity of the skull, pigeon-breast, bowlegs or difficult labour. In one case the father had had rickets, but not the mother. The children had severe and protracted rickets, despite the fact that they had been reared upon the breast. In his second table there are included 12 families with no history of rickets in the parents; the children were breast-fed without rickety signs, delayed dentition or latheness in walking. His third table comprises 12 families also; none of the parents presented rickety history, though some of the children contracted the disease through artificial feeding, respiratory affections, or the age or illegitimacy of the parents. His last table includes 14 families in which the parents had rickets and the children were artificially fed. The disease in this last set of cases developed earlier than in breast-fed infants; dentition was later and the affection ran a more chronic course, with a higher mortality. Its most severe forms were seen in the case of breast-fed infants with rickety parents. The death-rate of 121 artificially-fed rachitic infants was 87 per cent., of breast-fed rachitic infants 36 per cent., and of breast-fed nurslings only 15 per cent. Our author concludes that heredity is one of the most important etiological conditions in the causation of this disease, it being generally transmitted through the mother. Next in importance to heredity he places disorders of the digestive and respiratory tracts and the moral condition of the parents. Chance (Bodily Deformities) unreservedly accepts the hereditary theory of the disease and states that the morbid influence passes directly from the parent to the ovum to influence every stage of its development. He would also have it that ~~in~~ many cases of rickets apparently arising during infancy, are actually congenital and not recognised as such from birth owing to the disease remaining dormant for a time. Forthwith on the infant being deprived of the maternal milk and subjected to injudicious feeding, the disease, assisted by the irritation of dentition, breaks out afresh in the favourite positions, though the real excitant of the affection can be unquestionably referred to what occurs during intrauterine life. He refers to an interesting case of foetal rickets in proof of this; it is said that in it there was no doubt of the hereditary origination, though the history of the parents could not be traced. According to Cheadle (Artificial Feeding), there is no likelihood of the affection being transmitted, as it dies out during childhood. In spite of the fact that certain good observers affirm the influence of heredity in the causation of rickets, the balance of evidence and reliable opinion goes to support the theory that the disease is not so caused, but that weakly parents who have been rachitic in childhood tend to have delicate offspring, thereby favouring the ultimate production of rachitic babies. In short, the non-hereditary nature of this disease is proved by two facts: First, children of rickety parents do not always become rachitic. It is also to be noted that rickets is an affection of childhood and that the parents, although they may have suffered from it once, are often entirely free from it when they come to mature age. Anything that ~~waxes~~ bad health in the mother will predispose to rickets: hence it is a weakly constitution that is hereditary and the question of idiosyncrasy is also one that cannot be disregarded. The second point that can be urged against the disease being directly hereditary is that we constantly see cases of rickets in which the parents have never shown any signs of the disease. The predisposition, then, to the malady is hereditary; and everyone must have observed this in practices which deals largely with the science of pediatrics. Such children are more liable to the affection than others under the influence of various excitations; further, the malady is in individuals thus predisposed much more difficult to arrest than it otherwise would be.

I N S A N I T A R Y C O N D I T I O N S

Rickets is a common disease of cities and places where aggregations of people and overcrowding occur. In the great industrial parts of European and American cities the people live, as it were, herded together and the hygiene of the home seems almost ignored; the mother not infrequently has to neglect her household duties and go out every day to work in the factory or mills or workshop, in order to make up for the increasing dearness of living, or to foster pleasurable indulgences, caring little for maternal duties, putting out her child to nurse, leaving it in a crèche until she returns: so that it seldom obtains the maternal milk, but has to put up with bottles of questionable cow's milk or dangerous artificial foods. Some of these people herd together like animals, their houses are shamefully neglected and dirt and degradation abound. In other words, rickets is a disease of civilisation and it is important to note that a child kept indoors and away from the beneficent rays of the sun is very apt to suffer from it. So it happens that when rain and fogs are about and the weather is cold, there is much rickets; whereas in sunny China and Japan, for instance, the disease is comparatively seldom seen. The opposite, however, obtains in the narrow courts and alleys, and also in places where black smoke clouds hang in the air and keep out the health rays of the sun. Although the affection is sometimes seen in the homes of the well-to-do and those who keep their houses clean and sanitary, it is an affection nearly peculiar to overcrowded, jerry-built and dirty slums, especially of the larger towns and cities. Warmth and sunlight are known to be powerful vanquishers of the disease; for it is rare in tropical climates and coloured persons born therein on removal to temperate climes have children that are apt to be severely rachitic. I have already referred to the Italians and negroes of the United States of America as striking examples of this, even their breast-fed infants sometimes suffering severely from this ailment. In country districts it will usually be found that the affection attacks children who live in cold, badly ventilated, dark, damp and insanitary houses. Not a few of the latter are built with a northern aspect, without through ventilation (back-to-back houses), with windows perhaps that cannot be opened and sometimes with rooms lacking fireplaces. The front door may open directly into a stone-floored living-room, which is accordingly cold, damp and sunless. Ricketty children from these places do well under more favourable conditions; thus one not infrequently sees children, who are pallid and puny and unable to walk, pick up soon after their removal to hospital and surprise everyone by becoming possessed of a by no means inconsiderable activity. Besides the badly ventilated, overcrowded and dark habitations which such children usually have to tolerate, there is the additional evil of improper feeding. Even when the predisposition to the disease is not marked, want of cleanliness and inspiration of polluted air, &c., are amongst the most powerful excitants of rickets, which is therefore the poor child's ailment. Again, the affection is less common in the villages than in the towns, because the infants are more generally nursed at the breast and, even if the habitations are far from ideal, the surroundings are good, the air is pure and there is more out-door life: so that the growing children assume an increased power of resistance to render them less susceptible to bad influences and ward off the malign influences of artificial foods when given. Cheadle (Art. Feeding), however, does not regard such factors as the above as productive of the disease, he having observed that children brought up in good homes with excellent sanitary conditions, - warmth, light and cleanliness, for instance, - suffer from the malady. He maintains that these conditions only degrade nutrition and adds that he has never seen a child contract rickets when properly fed and kept from gastrointestinal disorders, even in the most insanitary circumstances. Morse has analysed a series of 318 cases in dispensary practice in Boston and holds that the only influence they had in common was unhygienic life in the home.

L o c a l i t y

We have seen that the location of the individual has much to do with the occurrence of rickets, it being mostly a disease of the larger cities and towns. Barlow and Bury tell us that,

leaving out of consideration the question of craniotabes and directing attention to the junction-area of the fifth and sixth ribs, there will be no difficulty in finding at least 50 per cent. of examples of distinctive rickets among children under two years attending the out-patient departments of the Hospitals in London and Manchester. It has been held that at least half of all slum children under three years of age are rachitic. We have also seen that in rural districts, though the cases are generally milder, the disease is by no means uncommon, being apt to occur in isolated hamlets composed of old, jerry-built, dark, damp and insanitary houses; there, too, fresh milk may be difficult to obtain on account of all the local supplies being sent off to the towns under contract. In other country places, however, the malady is not so often seen, the milk factor being of great importance in these cases. There is very little rickets in Greenland, Iceland, Norway and Denmark and the same is true as regards the warm countries already mentioned.

Microorganisms

It seems very doubtful that these are worthy of consideration as actual excitants of rickets; it is far more likely that toxins derived from the digestive tract, as a result of indigestion and nutritional disturbances, absorbed and circulated in the blood stream, are mainly responsible for the various expressions of the disease. It must be admitted, however, that the climatic distribution of the affection is apparently opposed to the theory of an intestinal toxæmia or microbial infection, but would add weight to the affirmation that the open-air life of the people in warm or ricket-free countries can be regarded as ricket-protecting. The germ or toxæmia theory of the malady has been given long and earnest consideration by numerous observers, the idea being suggested by such facts as the following: First, the perversion of the young osseous tissue in this disease is comparable to the process of inflammatory reaction and, because of its intensity and the manner in which it ~~is~~ rapidly affects the various parts of the body besides the bones, there would seem to be some ground for the hypothesis of microbial production. Second, this excessive hyperæmia of the bone tissue is such as would indicate the presence of a circulating toxin, probably absorbed from a diseased digestive tract. Third, there is in rickets a period which may be regarded as prodromal, during which time constipation, dyspepsia, perhaps diarrhoea and general perversion of nutrition and debilitation are apt to be observed. Fourth, according to Ashby, allied toxins are responsible for the variety of morbid phenomena observed, either the classical symptoms in the bones or constitutional effects pure and simple. ~~Wolfebyer~~, such would more readily supply a reasonable explanation for different parts of the body being affected at varying periods: for in one case gastric symptoms may predominate, in another debility with general muscular weakness, whilst other cases point to slight or profound disturbance of the nervous system as evidenced, in slight degree, by sweating of the head and restlessness at night or, in graver forms, by convulsions, tetany or laryngismus stridulus. Fifth, special symptoms, it is said, can only be explained by a bacterial cause - various toxins, with different selective actions, circulating in the blood and the nature of the toxin absorbed governing the kind of tissue attacked. In spite of such factors being suggestive of the possibility of bacterial origination, no specific microorganism has yet been discovered. Impressed by the intense affection of the bones and the rises of temperature sometimes seen in the course of the disease, Mircoli (Gaz. degli Osped., Jan. 30, 1898) conducted special observations, with the result that he reported the finding of streptococci and staphylococci constantly present in the mammary ducts of suckling women, as well as in the buccal cavities of their infants when being reared on the breast. He maintains that these microorganisms, which are quite harmless in the case of a healthy infant, become pathogenic when there exists any gastrointestinal disturbance and rickets ensues. The germs in question, having become pathogenic, attack the system generally, but select these parts which are in a state of most active growth, such as the epiphyseal ends of the bones, giving rise to the characteristic enlargements in such situations, and also the nervous system, causing reflex nervous irritability, convulsions or laryngismus stridulus. He thinks that the changes found in the osseous system are due to

chronic osteomyelitis and advances as evidence the fact that he has managed to produce enlargement of the epiphyses in young rabbits, seven days old, by injecting small quantities of cultures of the staphylococcus. In view of the unclean condition of many of the nursing mothers and the objectionable habit of using artificial teats or soothers, which are dropped on the floor or bedclothes none too clean, and then placed between the infant's lips which may be very dirty, it is not surprising that such germs as the streptococcus and staphylococcus may be found here. Murpurgo claims to have discovered a specific organism, a diplococcus, with which he injected young rats and caused typical rickets. Repetition of such experiments, however, by others has failed to effect confirmation of this opinion. Spellman has searched for a specific bacterium in the intestines of rickety infants, but did not succeed in so doing. As diarrhoea is a common symptom of the disease, he injected an extract of the diarrhoeal stools of such patients into 21 rabbits and one cat; but in one rabbit only did he produce anything like the affection in question. He tried again, but had no further success in 127 instances. Friedleben claims to have found an intestinal ferment possibly productive of the disease; no one else, however, seems to have done so. Kassowitz (Deut. Aert. Zeit., 1902, No. 31) devotes some space to an exposition of disbelief in the theory of rickets' production by an intestinal toxæmia, for the reason that the latter are more common in summer, the former in the wintertime. But this objection has been answered by the fact that the onset of rickets is so slow and insidious that it can hardly be possible to affirm that the affection did not really slowly incubate during the summer, when the various forms of gastrointestinal catarrh were about, the real symptoms of rickets developing only many months after the intestinal toxæmia had done its work. Indeed, he seems to upset his own objection; for he points out that rickets does not flare up suddenly as infectious diseases do, and further remarks that the commencement of the malady dates back much further than the winter months and must be sought for before birth or in the first months of life. He says that he has never seen cases in which an uncomplicated rickets suddenly appeared with febrile disturbances and showing fever during the evolution of the malady; in other words, he has never seen a sudden occurrence of the disease. Many others have tried to find a specific microbe, but without success; animals have been fed with the bacillus coli or were given toxin injections; the toxins of the bacillus pyocyaneus have also been used in the same way, but always without important result. It is reasonable to suppose that it is not possible to produce rickets in a healthy animal, but that some disorder of the intestinal canal must be present to ensure the commencement of the malady. Edliefesen (Deut. Aert. Zeit., Sept. 8, 1902) thinks that many of the cases of rickets are accounted for by cold and probably carbonic acid poisoning and suggests that the infection theory would account for the repeated appearance of the disease in certain dwellings - particularly in those in which other sources of infection, such as rheumatism, pneumonia and cerebrospinal meningitis, are found. Chaumier (Med. Inf., 1894) says that the disease is due to a specific virus which lurks in certain dwellings, and Burckhardt (Berl. klin. Woch., 1895) thinks that the skeletal anomalies and the splenic enlargement are expressions of a profound infection. Marfan and Marot (Rev. Mens., 1893) claim to have found the bacillus coli and the streptococcus pyogenes in the blood of rachitic infants with gastrointestinal catarrh. Czerney and Moser (Jah. f. Kinderh., xxxviii), in addition to these organisms and under similar conditions, found the bacillus lactis aerogenes. Ettore (Rev. Mens., Feb. 22, 1896) found various germs, sometimes in the bones. Charrin and Gley (Soc. Biol., Paris, Feb. 22, 1896) fed female and male rabbits on cultures of the diphtheria bacillus and the pyocyaneus one and got a rachitic litter. Lange (Verhand. d. 12, Vers. d. Ges. f. Kinderh. im Lübeck, 1895, p. 144) maintain that some infectious principle is attached to certain climates and localities, as in cretinism, because as in such ricket-free places as China and Japan, Finland, Iceland, &c., unfavourable hygienic conditions and other alleged excitants of the disease obtain.

Malaria

It not infrequently happens that the symptoms of rickets are of a vague character when occurring very early in life; thus, there may be a condition of pallor, drowsiness, general cyanosis and prostration. In short, there may be a general marasmic condition and, if this be accompanied by enlargement of the spleen, a superficial observer, under certain circumstances appertaining to locality and so forth, may make an erroneous diagnosis of malaria. But there is no doubt that the latter has no connection with the rachitic dyscrasia. Oppenheimer (Deut. Arch. f. klin. Med., xxx, 1881) thought that it had; indeed, he insisted that malaria was the principal cause of the disease, or rather his idea was that rickets is the form in which malaria makes its appearance in children of tender age. He is inclined to make light of the causes of the disease hitherto alleged and lays stress upon chronic diarrhoea and nocturnal crying in support of his theory. The perspiration, etc., accompanying the latter he takes as evidence of malaria, there being a substitution, he says of the same for the intermittent neuralgia of adults, especially in view of the enlargement of the spleen known sometimes to exist in rickets. He regards the diarrhoea of these cases as paroxysmal; it takes place, he tells us, in the morning and is, therefore, distinct from the ordinary form of intestinal catarrh. He also advances in support of his theory the serous evacuations unstained with bile, the good appetite during the daytime, the absence of emaciation, the gradual development of ~~anaemia~~ and the occasional fever. He adds that in certain instances the infants have cold feet and hands and blue lips towards evening; the skin is pale and the spleen is enlarged: otherwise there are perhaps no symptoms, but the patients endeavour to throw off the bedclothes and have a degree of more rise of temperature, with cranial perspiration in the morning; after a while the rachitic osseous and general symptoms make their appearance. His theory has found a very supporters, but my experience would seem to show that, though rickets undoubtedly occurs in malarial localities, there is no connection whatever beyond coincidence between the two diseases.

Oxygen Shortage

A lack of oxygen in the air respired is a powerful influence in the production of this malady; and it has been suggested that the real excitant is imperfect oxidation of the blood due to bronchial catarrh or collapse of the pulmonary tissue. On the other hand, excess of carbonic acid gas has been blamed for the ailment, which, in the opinion of Kassowitz (loc. cit.) may be due to inhaled organic matter, crowd-poisons and products of organic decomposition; this, he thinks, will explain the smaller number of cases and their more favourable course, if present, in the country and at high altitudes - the ammoniacal decomposition of urine furnishing such an excitant, in such an atmosphere a specially severe form of the disease being by no means infrequently observed.

Special Parental Factors

Numerous observers have affirmed that the age of the parents may have a good deal to do with the development of this disease in the children. Thus, Gelati (Hosp., Oct. 24, 1908) has made a special study of the subject during a period of four years in his hospital service. During that time he examined 3,121 children, of whom 615, - or nearly 20 per cent., - became affected with rickets. Of these children 1779 came from the town (Parma) and 307, - or a fraction over 17 per cent., - showed signs of the disease. Some 1215 were country children and 301, - or nearly 25 per cent., - were rachitic. The homes of the remaining 127 patients were not noted. With regard to the age of the parents, he says that in those under 20 the incidence of the disease was, roughly, 11 per cent.; between 20 and 30 it rose to 19 per cent.; to 22 per cent. between 30 and 40; to 22 per cent. between 40 and 50. Children born in the country always showed a higher proportion of the disease. Perverted nutrition in one or both parents may predispose to the malady, as may also anaemic or other debilitation, as well as advanced age at the time of procreation and early and improvident marriages.

S e a s o n

This subject has already received some consideration above, but it may here be specially noted that most competent observers hold that the affection is more often seen in the winter than during the summer, though it is not easy, in the case of hospital patients, to arrive at a definite conclusion as their attendance in the out-patient department is generally less frequent when the weather is cold. It is sometimes urged that rickets, in addition to being more common in winter, is at that time of a much more severe type than in summer. The disease is more common during the cold months of the year because of the long periods of inclement weather, with dark and sunless days - the result being that the children are kept as comfortable as possible in the house; this is particularly operative in the case of slum children and others of their kind, there being an awful dread of the open window and the air and ventilation being correspondingly bad. In this way the nutrition and vital resistance of the child are lowered; the infants suffer from frequent catarrhs of the respiratory or gastrointestinal tracts, the way being thereby paved for the occurrence of the disease. Cases which cannot be explained in this way have been assigned by some to the infection theory. Edlefesen (loc. cit.), from an examination of the children in the Kiel and Hamburg clinics, concludes that the fact that the number of cases reported in the first half-year increased steadily and reached its maximum between the sixth and ninth months of the year justifies the hypothesis that rickets is favoured by wintriness of weather and supports the carbonic acid theory also, because of the frequency during these months of diseases of the respiratory system.

S e x

I have never been able to convince myself that the element of sex is a factor of importance in the production of rickets, especially as personal and other statistics as to the relative incidence of the affection exhibit great variation according to coincidental and other conditions. Some writers would have it that girls are more prone to the disease than boys, whereas others affirm just the contrary or negative the idea by apparently convincing figures or show that the sexes are equally affected. The older writers on the subject, I observe, thought that the less robust frames of girls rendered them more prone to the malady. Hall (Phys. Deter. Rep., p. 89) has had a great experience of the ailments of scholars and tells us that "rickets appears to be more common among the girls than the boys. The rickety dwarf, with bowlegs, narrow pelvis and big head, is no very uncommon object among girls, but rare among boys!" In another analysis of 46 cases there were 30 boys and 16 girls, however. Goodhart and Still, in 141 cases, saw 68 boys and 73 girls; at 5 months there were 3, 7 months 1, 8 months 3, 9 months 2, 10 months 5, 11 months 6, 12 months 11, 18 months 36, - at 2 years 26, 2½ years 19, 3 years 13, 4 years 6, 5 years 5, and at 6 years 3 cases. In Gee's 635 cases there were 365 of the male sex and 270 of the female (32 under 6 months, 144 from 6 to 12 months, 183 from 12 to 18 months, 133 from 18 months to 2 years, 116 in the third year, 27 in the fourth year), and he thinks that 30 per cent. of ailing children under 2 years are rachitic.

S y p h i l i s

It is now more than thirty years since Professor Parrot, of Paris vaunted the hypothesis that rickets is nothing else than an expression of congenital syphilis in which the virus has reached an attenuated stage; he regards the osteophytic and spongoid tissue as common to the two ailments. In other words, he refuses to look upon rickets as a pathological entity, but merely as a modification of syphilitic bone disease and adds that "it is an old confusion to consider that it is rickets which absorbs the syphilitic alterations of bone, but, on the contrary, syphilis in its immense process includes rickets!" (Trans. Path. Soc., xxx). He also notes two characteristic lesions of the membranous bones, which he classifies under the categories of atrophy and production of new tissue. Though there are some who admit that syphilis may modify rickets, Parrot's theory is now regarded as untenable, the two diseases being considered

as distinct conditions having, however, certain symptoms of marked similarity. Numerous have been the objections urged against the theory, of which the following are most worthy of consideration: One daily comes in contact with cases of rickets in which there is not the slightest trace of syphilis and experiment would seem to bear this out. Thus, if puppies of, say, three weeks old are taken from the mother and fed on raw meat, bread and no milk, they develop symptoms of rickets as manifested by enlargement of the abdomen, swelling of the epiphyses, curvatures of the bones and difficulty of walking. Now, if this diet is interrupted, the morbid process is stopped and, on resumption of the diet again, the disease returns. The autopsical lesions exactly correspond with those of rickets. It is also known that animals of the kind we are considering do not contract syphilis: so that the syphilitic theory cannot obtain. Parrot, too, was mistaken in his ideas; for he states that the bone lesion was essentially characterised by spongoid tissue and that without the latter there could be no rickets. He also states that by premature feeding he could produce osteomalacia, but not spongoid tissue. But it is well known that osteomalacia is a different disease, not an arrest of development; it is a disease in which the already ossified bones have undergone subsequent softening and, moreover, it is an adult disease with a recognised and definite relation to the pregnant condition. The fundamental difference between the two maladies is very great; for rickets is a cartilage producer, whereas syphilis is the great bone producer. It does not follow that because an infant is syphilitic it will also suffer from rickets; but, owing to the debility and the cachectic condition so often produced by congenital syphilis, it often contracts rickets as a secondary complication of the specific disease. The clinical evolution of syphilis and rickets is entirely different. In congenital syphilis we have eruptions, snuffles, dental anomalies, - Hutchinson's teeth, - keratitis, scars about the angles of the mouth, and so forth; whereas in rickets there are usually none of these. Parrot replies to this that it is a case of transformation of disease, the syphilis having run itself out and made a sort of quick-change into rickets. In my practice there have been innumerable cases of congenital syphilis, but seldom or never have I seen rickets occur solely from this cause. Syphilis may, of course, accompany rickets; but before it can be decided that the former is the cause of the latter, all other causes must be excluded. Hereditary syphilis appears earlier than rickets and is often a disease of intrauterine life. In syphilis there is generally a history of previous miscarriages; as a rule, the firstborn is affected, though, if the mother is under treatment, the disease may only appear in the second child of the family. Rickets, as a rule, appears in the last child of a family when the mother's blood is poor and when she has been exhausted by pregnancies rapidly following one another or by lactation during the gestation of another child. Syphilis is common to rich and poor; whereas rickets is most often seen in the slums. Syphilis responds to treatment by mercury and some observers state that the use of this drug in rickets is dangerous. One of the points urged by Parrot in support of his theory is the fact that he could cure rickets by antisiphilitic treatment. But it is a recognised fact that rickets tends to cure itself under proper dieting and tonic medication; further, mercury is a tonic and the iodides operate beneficially in diseases other than syphilis. Again, it is possible that in not a few of Parrot's cases an error of diagnosis was made, owing to the frequent simulation of other affections by syphilis; thus, syphilitic children not infrequently suffer from periostitis, in which condition there may be enlargement of the ends of the bones. Syphilis may modify rickets, but can never create it. Non-syphilitic parents may beget rachitic children, while syphilitic parents may produce healthy infants. The skeletal changes in rickets tend to take their departure after infancy and childhood, but those of syphilis more often leave such permanent relics as can be detected in after-life. Syphilitic children seldom show the so-called spongy tissue of rickets and rachitic bones rarely present the osteophytes so characteristic of syphilis. The latter affection produces changes in the bones analogous to rickets: for instance, the cranial bone trouble known as craniotabes and the peculiar bosses on the frontal bones; but syphilitic bones do not show the spongy appearance of rachitic bones, nor do the latter exhibit the

osteophytes of syphilis. Many observers have regarded craniotabes as being invariably of syphilitic origin. Barlow and Lees (Trans. Path. Soc., xxxii) have analysed 100 cases, in which 71 showed a marked degree of craniotabes, 30 only showed it slightly and our authors concluded that syphilis was present in 47 instances. Of the latter, 12 had slight and 35 marked craniotabes; occasionally the latter condition seemed to be the last survivor of a disappearing syphilitic dyscrasia. Before the time of Parrot, Elässer noted that craniotabes was apt to recur in two or more children of the same family, it having been seen in healthy well-nursed infants and not being merely a marasmic condition. Our author regards the sign as pathognomonic of rickets, and Parrot, on the other hand, held that it was a syphilitic manifestation. Barlow and Lees think that syphilis is by far the largest factor in the causation of craniotabes. After tabulating rickety symptoms associated with the latter, they endeavour to define the relationship of one to the other by taking 53 cases between 2 and 19 months of age. Amongst ~~some~~ 11 breast-fed infants there was no rickets; there was scarcely any rickety sign in 1 case almost exclusively breast-fed; 9 had rickets in 14 partly breast- and partly bottle-fed; and 6 showed incipient rickets out of 9 cases suckled for part of a month. Our authors conclude that, if craniotabes and enlargement of the spleen are removed from the category of rickety symptoms, there is no proof that syphilis, per se, is a cause of rickets; they hold that craniotabes is very common in congenital syphilis, not common to pure rickets and not at all special thereto. It would seem, then, that syphilis is not an exciting cause of rickets, though, by lowering the general vital resistance of the system, the former may predispose to the latter.

Toxæmia

In addition to what has already been said on this subject, it may here be noted that it is now a generally-accepted fact that in such diseases as diphtheria and typhoid fever and tetanus the blood of patient manufactures antitoxins to enable the system to deal successfully with the toxins poured out into the blood stream by the particular microorganism concerned. Each disease has its own special antitoxin, which will fight against the special toxin with which it comes into contact. The reasons for all this are fully detailed in Ehrlich's and other works: so that it is unnecessary to narrate them here. But it is important to remember that it is not microbial disease alone that is able to excite such a peculiar and useful reaction of the system towards foreign invaders: for cow's milk, when injected into an animal, is able to produce in the blood-serum of the same the ability to produce a precipitate when added to fresh cow's milk; whereas this does not occur if the serum of the infected animal is mixed with the milk of a mare or woman. Similarly, if cholera bacilli, a bacteria-free filtrate of typhoid germs or spermatozoa are injected into a guinea-pig, the serum of that animal will manifest a similar phenomenon. The serum only agglutinates with the injection employed in the experiment - bacteria, bacteria-free filtrate or spermatozoa. As the injection of milk, spermatozoa, bacteria or their toxins produces a similar reaction in the serum of mammals, the inference is that the milk of the cow, the spermatozoa or the bacterial toxins can all be classed as poisons under certain conditions of the organism. Intravenous injections of cow's milk are able to lower the leucocytes in the blood and, if the injections be often repeated, death of the animal will occur. The albumin of one animal will act as a poison to another animal and all these substances, to safely support life, must first pass through the intestinal canal and be dealt with by the process of digestion if their poisonous action is to be prevented. This canal, then, has the power of protecting the system and of preventing the absorption of foreign albumins until they are first rendered harmless by being acted upon by the above-mentioned physiological process; they are acted upon by the ferments of digestion and cease to be toxic, - for if milk or egg-albumin, previously digested, are now injected into an animal, they are no longer able to cause the appearance of antitoxins in the blood stream. The proteid of the cow must be digested, or converted into human proteid, before an infant can be safely fed upon it. The epithelium of the gastrointestinal canal has the special power of being able to deal with the albumins of different

animals; but all other cells of the body can only with safety use a proteid of their own species, all others having a poisonous action. It is obvious, then, that, whereas the infant upon the breast is able to assimilate without change the human proteid which it takes in this way into its stomach, the artificially-fed infant is first obliged to transform the proteid of the cow's milk into the human proteid before it can with safety pass out of the stomach into the circulatory system. The result of this is that, in the case of artificial feeding, an alimentary canal, in a rudimentary condition and with intestinal cells poorly developed, is called upon to perform extra work at each meal which the infant takes, and with the ever-present danger added that any failure of its functions may lead to the absorption of toxic substances; for the other cell tissues will react to the albumin of the milk of the cow as if it were a poison, if unconverted into human proteid. Under ordinary conditions no toxic products are allowed to pass into the circulation; for no antibodies are found in the blood; but, if cow's milk is injected subcutaneously, antitoxins are invariably produced. There is experimental evidence to show that the mother is able to pass the antitoxins present in her milk over to the suckling, but that there is no such conversion of another animal's antitoxins. Morse (Jahr. f. Kinderh., lv, 1902) is one of the advocates of this idea. He could not discover any bactericidal properties in cow's or human milk, but noted that the blood serum of breast-fed children has much greater bactericidal and haemolytic powers than that of children reared on the bottle. He thinks that this is due to the presence of alexins in human milk and, although these cannot be demonstrated, he would have it that they are derived from the blood serum of the mother and combine with the casein by the action of mammary cells; he is therefore a great believer in the virtues of breast-feeding and maintains that no mother is fully qualified to have charge of a child unless she herself gives it the breast. With him I agree, as the advantages which accrue to a breast-fed infant are far deeper and more potent than those derived merely from the angestion of a food of synthetic arrangement. The breast-fed child is under conditions to receive a food chemically and biologically suitable for the development of its gastrointestinal tract, ready for almost immediate assimilation, non-poisonous to its tissues and containing the antitoxins of its mother's system. On the other hand, however, the bottle-fed child is reared, - or hoped to be reared, - on cow's milk or artificial food and receives a substance that may have the same chemical elements in it as human milk, but with the atoms differently arranged and possessing a biological action intended for another animal; it is not readily assimilated, for, in the case of a milk, it is intended to assist in the evolution of the digestive functions of an animal whose ratio of growth is more rapid than the human infant; it is poisonous to all cells of the human body, except the intestine, and, when acted upon by the digestive ferments, it must first be converted into human proteid before it can be absorbed with safety into the child's body; moreover, although possessing antitoxins for its own species, it is unable to transfer any of them to the human infant's organism. The consequence of all this is that an artificially-fed infant is always being nourished under gargantuan obstacles to success and, unless its food and hygienic surroundings are very carefully supervised, it is liable at any time to suffer from digestive disorders, debilitation and trophic anomalies. There is no doubt that the rickety infant very often suffers from gastric dilatation and shortage in hydrochloric acid necessary to good digestion; the result of this will be that the pepsin will not be liberated and there will be interference with the digestion of the curd. It is possible, too, that antecedent catarrhal troubles of the stomach and intestines may have weakened the epithelium, thereby allowing albuminous products, unacted upon by digestive ferments, - and therefore poisons, - to pass into the circulation. These toxins, possibly with different selective actions, would give rise to irritation of the various internal organs and, by attacking the young and growing cartilage and periosteum, interfere with the bone-producing tissues and lead to the ossification process being impaired. In short, a sort of albumin toxæmia occurs. The fact that rickets can be cured by fresh air, suitable hygienic surroundings and the addition of fat to the diet, plus digestive regulation, cannot be altogether be urged as a disproof of this

hypothesis, because under such improved conditions as the above the leucocytes of the blood could deal with the toxins present in the system, the irritation of the tissues would lessen and ultimately there would be recovery from the disease. The epithelium of the digestive tract, repaired and a healthy state, would then be able to manipulate successfully the proteid of the cow-milk and change it into human proteid, thereby preventing further toxin absorption; assimilation of the food in question now occurs, repairs the damaged tissues and allows a normal progress of the growth and development of the patient. This idea is not one that meets with invariable acceptance, but it would seem that it opens up a way along which we may ultimately arrive at the real cause of the disease.

T u b e r c u l o s i s

There have been many in the past who claim to have recognised a relationship of tuberculosis to rickets, as well as numerous others who have denied it, stating - without proof - that tubercle is rarely found in the rachitic subject of tender age. Eustace Smith refuses to regard rickets as diathetic condition and says that it never makes its appearance in children in whom the tubercular deposition is not well marked. Holt thinks that there is not sufficient reason for believing that rickets exerts any protective action against tuberculosis and adds that it is more probable that the deformity of the chest may be not without predisposing influence on the origination of the disease. Sir William Jenner holds that rickets does not negative tuberculosis and he gives statistics to show that the children of tuberculous parents exhibit a diminished incidence of the former malady. Edwards (Trans. Path. Soc., xxx) insists that the offspring of phthisical parents are less liable to become rachitic than those who are non-consumptive. Rittershain found a large number of tuberculous fathers in rachitic families and thinks that this is not without significance. Ruzf considers that it may be taken as proved that rickets and tuberculosis or scrofula are not manifestations of the same diathesis, but that, as a general rule, the one excludes the other; this law, he adds, is so absolute that there is, a priori, almost a certainty of not finding tuberculous or scrofulous affections in a rachitic subject, nor rickets in one who is scrofulous or tuberculous. ~~Rohsenhain~~, from the above and my own personal observations, it would seem that rickets does not protect the individual against tuberculosis, though the rachitical catarrhal affections of the ~~r~~espiratory organs and enfeeblement of nutrition may prepare the ground for the lodgment and destructive operations of the bacillus tuberculosis.

T h y m u s a n d T h y r o i d

Some eleven years ago it was affirmed that there is a certain association between rickets and perversion of function of the thymus gland, Mendel (Munchn. med. Woch., 1902) basing this idea upon his observations in cases of atrophy of that structure and successful treatment with its extracts. According to his school, an internal secretion of the thymus exercises some effect upon the development of bone, the enlargement of the spleen being a compensatory hypertrophy and replacement of the absent glandular function being effected thereby. Thyroid deficiency has also been alleged as a factor in causation of the disease, it being rich in phosphorus and its removal from the mother having been said to produce foetal rickets. Ausset (Gaz. Hebdom., 1901) thinks that the success of phosphorus in the treatment of rickets is due to its supplying a stimulus to the abeyant thyroid function. Further light must be shed on this question in future before it can be expected to meet with anything like general acceptance.

P a t h o l o g y

Various theories have been from time to time advanced to account for the occurrence of the osseous lesions in this disease, some of which we have already considered. In addition there -to, we may note that so good an ~~observer~~ **observer** as Kassowitz (loc. cit.) was inclined to the opinion that the malady is of an inflammatory nature; according to him, the primary anomaly is in the hyperaemia of the cartilage, periosteum, bone-marrow and bone substance, the visceral alterations being secondary to the same. In addition to this so-called inflammatory theory, Glisson referred rickets to nutritional disturbance by arterial blood; he would account for the changes in the long bones on the ground of excessive vascularity. The irritation theory of Keating is somewhat vague in its terms; it supposes that some irritation, - of obscure character, - causes an overgrowth of the sensitive osteogenetic tissue. The deprivation of lime is regarded as a secondary condition and not the actual cause of the disease; if the irritant acts suddenly or profoundly, and thus interferes with the process of assimilation, the disease produced may be not rickets, but an atrophy of tissue. No information is yet forthcoming regarding the nature of the irritant; it may be caused, it is said, by decomposing food, by microorganisms, by chemicals present adventitiously in the tissues, and so forth - it being further held that undoubtedly any injurious influence or depression of vitality, - such as dietetic errors, damp and lack of sunlight, - will give rise to a rapid output or irritant material during the susceptible age of the child. No matter how the lesions may occur, there is no doubt that in rickets there occurs a perversion of the normal processes of formation of bone tissue - instead of normal bone being laid down the final consummation of this process being arrested and inferior material being manufactured; the proliferating **cartilage** cells are far too active and, instead of calcification occurring, the parts become exceedingly vascular, with large spaces filled with a red gelatinous marrow instead of good, sound bone. Inspection shows that there is a thickening of the epiphyses of all the long bones, especially those of the lower arm; the width of the epiphyses may be increased 50 per cent., and there may be an abnormal rounding of all the angles, borders and prominences of the bones, which themselves are unusually flexible and bend under the weight which they are called upon to support. The osteogenetic perversions in question interfere with the growth of the bones concerned, both temporarily and permanently sometimes; there is interference with development, perversion of ossification and irregularity and incompleteness of calcareous deposition; finally, serious changes may be produced in the already ossified bones, that is to say, the osseous tissue loses in density, weight and firmness, with the result that the affected bones yield under the pressure of the fingers and can be cut with a knife like a soft piece of wood or turnip. Guérin has described the rachitic alterations as occurring in the four stages of effusion and rarefaction; deformation with organization of effused material; consolidation and eburnation; and consumption in exceptional cases: (1) In his first stage of effusion, rarefaction or incubation, the normal $\frac{1}{2}$ - 2 mm.-thick chondroid layer becomes from 4 - 12 mm. in thickness, swollen and spongy and bluish-grey in tint. On one side it blends with the cartilage of the epiphyses, whereas on the other it presents an irregular indented border. There is excessive activity of cartilage, the spongoid tissue pushes **itself** up in an irregular manner, there being in consequence difficulty in distinguishing spongoid and chondroid tissue; patches of one tissue are found isolated in the tissue of the other: so that calcified areas may be seen in parts which are cartilaginous and little islands of cartilage in places where complete **calcification** should normally occur. The secondary capsules do not **dissolve** but calcify, the blood-vessels enlarge and anastomose freely, and the interspaces are filled with gelatinous marrow instead of normal bone. In this spongy bone there is thickening and erosion of the bony trabeculae, thus forming large medullary spaces filled with blood-vessels and connective tissue. The medullary spaces

are continuous with the channels of the shaft and a tissue, spongy and vascular, is thus formed; the areolar tissue is dilated and full of blood-stained jelly, which has been compared to gooseberry jam. The medullary canal is also full of a highly vascular and gelatinous substance. Similar alterations occur in the periosteum, there being a faulty ossification with an excessive proliferation of material. The outer fibrous layer of the periosteum is thickened, vascular and adherent to the roughened bone; the inner layer in which bone is formed is far too active and the exudation first **protrudes** connective and afterwards osseous material. This subperiosteal bone is five or six times thicker than normal; but it is of poor quality, spongy and deficient in lime. The dried bones are porous, friable, decalcified and of very light weight; they are soft and flexible, bending under traction of the muscles or supported weight. The thickening and vascularity of the periosteum is more marked on the concave side of the bending bone, the convexity showing more adherence. (2) In the second or deformity stage the bones are markedly spongy and biscuit-like; the lime salts are absorbed and the place of normal bone is taken by fibrous tissue. (3) In the stage of consolidation and eburnation there is, in from three to fifteen months, cessation of the active proliferation of cartilage and periosteum; the bone gradually becomes less vascular and the normal process of ossification occurs - the bone being in a suitable condition for rapid ossification as soon as the rickets subsides. The new tissue underneath the periosteum and between the diaphysis and epiphysis organises and becomes calcified. It may happen, however, that this ossification occurs so rapidly that the growth of bone is cut short; but the bone now formed is very dense and as hard as ivory - hence the term, "eburnation," which is used to describe it. The bones also tend to become straight and the nodosities to disappear. (4) The stage of consumption is, as already indicated, not always present; in it consolidation of the tissue does not occur the bones become fragile, light and made up of fatty areolar material. It not infrequently happens that the active process ceases when the patient is two and a half years of age; if then examined, it will be found that the swelling at the epiphyses is undergoing diminution, the curvature, if slight, may have disappeared, the beadings of the ribs are not discernible nor palpable and the bosses of the head are not nearly so large. But, still, the deformities produced by the above changes do not always diminish, as they may last through the lifetime of the individual and convert him into a humpback or unsightly dwarf. The extreme lightness of rickety bones was well known to Trousseau, who noted that an affected femur, even after the occurrence of consolidation, is below the normal weight, perhaps only a seventh of what it should ordinarily be; he further observed that such bones were soft and easily bent, that they were easily sliced with a knife, very liable to be broken from slight cause, deficient in lime salts and yielding less gelatine when boiled - facts also noted by Jenner, who remarks (*Med. Times & Gaz.*, Mar. 17, 1860, p. 261) that in the osseous changes in rickets there occurs "an exaggeration of the conditions we find in the first stages of ossification in the healthy subject; the completion of the process only is stayed;" - in other words, great preparations are made for the process of ossification, but the performance of that process is extremely irregular and inadequate for the purpose to be achieved. Even older writers than he were familiar with the osseous changes of rickets and give good descriptions of the lesions observed. They tell us that the bones are lighter than normal and of a reddish-brown colour; they are penetrated by many enlarged blood-vessels, being porous, soft, compressible and spongy; they have a moist watery secretion, which may be pressed out of their structure as out of a sponge or piece of moist leather; the walls of the medullary cylinder of the large bones of the limbs are thin, while the bones of the skull are much increased in thickness and become spongy and reticular; all the affected bones, especially the long ones, acquire a remarkable suppleness, but if they are bent beyond a certain point, they break; instead of being filled with medulla, the medullary cavity of the long bones contains only a reddish serum, totally devoid of ~~the~~ fat, oily secretion of health. In the case of a healthy infant of four or five months, the shafts of the long bones are completely ossified, while the

epiphyses still consist of cartilage. The bones increase in length by ossification between the epiphyses and the shaft and in thickness by deposition of compact bone underneath the periosteum. The bones of the skull, except at their edges, where the fontanelles occur, do so likewise. The minute changes in rickety bone are seen, under the usual magnification to consist of a production of defective material, too much activity of the cartilage cells and in places of normal calcification an excessive vascularity with the formation of large spaces filled with a gelatinous material. The epiphyses of the long bones are thickened, especially those of the forearm; bending is comparatively easy of accomplishment. Taking a general view of the rachitic process, it would seem that there is far too active a preparation for ossification and quite as poor an accomplishment of it. The main alterations, as we have seen, are to be found in the skeletal system; but, nevertheless, it must be remembered that all the organs of the body participate, though by no means so obviously, therein. The long-bone changes comprise swelling of the epiphyses, thickening of the periosteum, shortage of lime salts, overgrowth of medulla, in the form of a gelatinous material, and bone softening. With the evolution of the disease the bones become bent and distorted and such fractures as the greenstick are very apt to occur. The affection tending towards recovery, there occur consolidation and eburnation of the implicated bones, which ultimately become much harder than normal. Not infrequently the patient is permanently dwarfed; and a former attack is sometimes betokened throughout life by bossed head and enlarged articulations, though perhaps almost as often as not the adult shows no relic of the disease.

It is a well-known fact that the long bones grow longer owing to increasing bony deposition between the epiphyses and shaft; they thicken by a similar happening underneath the inner periosteal layer; whereas absorption of the inner bony layers gives rise to the medullary tunnel. Now in rickets there occurs a perversion of these normal processes; there is far too much cartilage produced at the epiphysis, there is too much subperiosteal deposition and there is imperfection or arrest of ossification in general; the inner bony layers are absorbed too quickly, with the result that the medullary canal assumes too large dimensions and fills with a spongy material; finally, there is imperfect calcification of the fully-formed bone. In the process of long-bone growth the active element is the diaphyseal cartilage, which passes through the two intermediate stages of chondroid and spongioid prior to the accomplishment of normal bone manufacture. The tissue in question is a layer of hyaline cartilage separating the body of the bone from the head of the latter. It undergoes thickening, proliferates and a spongy layer is formed presenting delicate reticular trabeculae in its substance. The chondroid layer measures from a half to two millimetres in diameter and lies between the diaphysis and the intermediate cartilage. It consists of primary cartilaginous capsules in which lie parallel rows of secondary capsules separated by layers of granular substance - the rivulations of Broca. In these intermediate rivulets the deposition of lime salts first takes place, then in the primary capsules and, finally, the secondary capsules dissolve and break down: so that their contents mingle in the primary capsules, the calcified partitions being absorbed and the spongioid layer formed. This undergoes proliferation and ossified cartilage vessels pierce the layer, little canals are formed and the final stage in the manufacture of bone is accomplished. In the case of ordinary healthy bone inorganic material constitutes a third, this proportions, however, being reversed in rickets: so that we have a third inorganic to two-thirds organic substance. Absorption or removal of lime phosphate is mainly responsible for the deficiency in question, though so good an observer as Kassowitz holds that it is a defective deposition of that salt, not its absorption, which must be incriminated.

Blood

Repeated examination of the blood in rickety individuals not infrequently shows that there is nothing wrong with it, which is somewhat remarkable in view of the rôle of the bone-marrow in this disease. In not a few of my cases, however, I have found anaemic indications, the red blood-corpuscles being diminished, together with the haemoglobin, and a few nucleated red cells here and there. I have found the haemoglobin to stand at from 50 to 75 per cent., the results therefore pointing to a general weakness of the parts which are concerned in the manufacture of blood. Complications will, of course, give rise to the usual secondary anaemias and blood changes and in many instances I have observed a varying degree of leucocytosis.

Bones

Long Bones.

We have already seen that the older writers were in the habit of vaunting all sorts of fanciful theories to account for the osseous changes in this disease, Glisson being the first to give a correct account of the softening and subsequent consolidation processes. Duverney, in 1751, devoted a treatise to the consideration of osseous affections and described certain bone lesions of rickets, particularly rarefaction, lightness and surface roughening from the pressure of layers of osseous material formed by what he termed nutritive-juice extravasation. Equally competent observers referred the changes to a disturbance of the growth of bones and muscles from unequal distribution of the juices which fed the body; whereas others at this time looked upon rickets as an expression of such diseases as syphilis, scrofula, scurvy and rheumatism, the scrofula and syphilis theories being perhaps the most popular. Guérin's four stages have already been described; he states that the stage of consumption, admittedly uncommon, is observed in those who have long suffered from the disease and in whom the so-called rachitic cachexia has occurred - in this stage there being no attempt at restitution. The bone tissue remains modified; the matter effused into the areolae and into the interlamellar spaces underneath the periosteum and the medullary membrane is not transformed into that cartilaginous tissue which ought itself to be transformed into new bone, the old osseous material being generally absorbed. Our author also remarks that in the recent state the long bones are soft and friable; they are reduced to a very thin shell filled with fatty matter; in some places they are whitish, or of a more or less reddish tinge, and contain the débris of osseous plates; in the dry state these bones are remarkably light, friable and brittle; the lightness and friability are just as conspicuous in the short as in the flat bones, the tables of which become very porous, whilst their dioplœ, the cellules of which enlarge, assumes an aspect like a paste that has risen and looks like a macaroon. He tells us that the first stage, - incubation or effusion, - lasts for some months, when gastrointestinal irritation, nocturnal perspirations, mental depression and nervous irritability, as well as weakness of the muscles and ligaments, are much in evidence, thereafter appearing the curvatures and growth stunting of the implicated bones; the deformity is said to commence in the leg bones and work upwards. He has published analytical tables to show that the retardation of growth in the lower extremities is much greater than in the upper ones - other observers, however, not confirming this from actual **phætic**-ice; The reason for this discrepancy would seem to be the time of onset of rickets, whether before or after the art of walking has been acquired by the **philiant**. The periosteum of a rickety bone is more vascular than in health and the outer fibrous layer, as well as the inner proliferating one, is thickened. To the naked eye it presents a bright pinkish tinge, due to the excessive blood supply of the part obtaining. The periosteum is easily stripped from the bone, which is also hyperaemic and not exhibiting the smoothness of the normal, but roughened from irregular osteoid deposits from the internal layer of this membrane. A longitudinal section of an affected long bone shows a large area with bluish semitransparent tint between the epiphysis and diaphysis. Normally a thin, straight, transverse line, it is now much wider and of irregular outline. The yellow zone of

cartilaginous calcification is even more irregular, with isolated patches in the bluish part. Blood-vessels may be detected entering the cartilage at different levels, there being also considerable swelling of the blue proliferating zone. The shaft of the bone appears to consist of very soft, spongy bone containing a large amount of reddish-brown gelatinous material. In the third stage, - that of consolidation and eburation, - there occurs a deposition of healthy osseous material, the blood-vessels become involuted and bone is formed around the former blood spaces in lamellar systems, with small or obliterated canaliculi occasioning the ivory-like appearance of the characteristic rickety bone. It would appear that the deposition of osseous material is most actively effected in those parts which most require it, that is to say, on the inner or concave surface of the curved aspect, and that the thickness of the bone at the most curved part bears an exact ratio to the degree of curvature that the bone has undergone. Nevertheless, the deposition of osseous material is not always adjusted to the mechanical conditions of the parts of the body as means to ends, for the cranial bones are far thicker than they need to be. Development of bone occurs either by the intracartilaginous or the intramembranous method. The intracartilaginous method is to be found in the case of the flat bones of the cranium and face, as well as in periosteal ossification. We have seen that the long bones lengthen by the deposition of osseous material in the cartilage between the epiphysis and shaft; also that the latter thickens by the production of bone underneath the inner layer of the periosteum, absorption of the inner layers of bone leading to increase of the lumen of the medullary canal. Under normal conditions the lengthening of bone is more rapid than its thickening, the most obvious changes in this disease being seen at the ends of the long bones. If we cut down the length of the femur, for instance, the thing which first catches the eye is the large size of the piece of cartilage which lies between the ossifying centres of the epiphysis and the shaft, it appearing also to be unduly active. There are larger absorption spaces than in health and hyperaemia is present, with an epiphyseal line of abnormal width. The osteoblasts are more abundant and they arrange themselves around the absorption spaces in the cartilage as in normal ossification. They go the length of providing the fibrous homogeneous matrix, but they go no further and the formation of true bone is accordingly arrested in its occurrence; in short, as we have seen, there is an extensive preparation for ossification, but imperfect performance of the same. The patient, because of the cessation of the normal lengthening process, becomes stunted in appearance. The crude fibrous tissue accumulates at the epiphyseal line and gives rise, by so doing, to the well-known rickety tuberosities. In the same way the inner layer of the periosteum shows undue activity and cell proliferation; the fibrous tissue is laid down in successive lamellae, but calcification is restrained, an undue flexibility of the implicated bones resulting. All the long bones, however, are not affected to the same degree. Sometimes those of the lower limbs are most implicated, sometimes those of the forearms, sometimes those of the thoracic wall. The deformities occurring may be due to an exaggeration of the normal curves or exaggeration of the same by swelling of the epiphyses; further, muscular action, atmospheric pressure, posture, and so forth, may be responsible, aided by the shortage of lime salts obtaining. It was long ago observed that, in the beading of the ribs, the cartilage fits into the bone like the acorn into its capsule. There is an interruption of the union of the flat bones of the skull, which fail to unite and occasion patency of fontanelles. Large bosses or prominences, due to bone thickening, usually make their appearance about the centres of ossification; they are soft and spongy, come mostly from the outer table and are seen to be of excessive vascularity. Greenstick fractures may occur in connection with such bones as the radius, ulna, clavicle and ribs, leading ultimately to more or less impaction and being followed by callus-like condition and simulating a false joint. In from three to fifteen months there is a vigorous initiation of the stage of restitution in both the cartilage and periosteum. The new periosteal bone is thrown down underneath that membrane, this deposit being most plentiful on the convexity of the bone and very dense and ivory-like. By the time that the patient is two years old, or thereabouts, the osseous alterations have run their course. At

this time the swellings at the end of the long bones undergo gradual diminution, as also the curvatures sometimes when permanent deformity is not going to occur. The so-called consumption stage has already been described; it is by no means commonly encountered. If we submit a longitudinal section of, for instance a rib, to microscopical examination, we shall note irregular proliferation and calcification of cartilage, as well as irregular formation of osseous tissue, with increased absorption of this bone-like material. The swollen end corresponds to the lower zone of the cartilage, in which the cartilage cells have undergone proliferation and arrangement in rows. Next to this we have the epiphyseal layer, normally narrow and sharply defined, now irregular and badly defined and composed of areas of uncalcified cartilage and osteoid tissue, with large medullary spaces and blood-vessels. There is a marrow rich in cells, but possessed of a shortage of fat cells, the bone, in general, showing very few of the normal histological characters. Lying in the recesses of the osteoid tissue may be seen large cells with two or three nuclei; they are termed the osteoblasts and assist the blood-vessels in the production of true bone. Underneath the periosteum, which is thicker and more vascular than normal, there is an excessive production of cells, though calcification is arrested and a spongy appearance of the part results. The increased vascularity of the tissue in which the bone is to be formed, the marked hyperplasia and the increased osteoporosis of both compact and spongy bone are readily appreciated under this form of examination. It is interesting to compare the changes noted in the bones of young animals fed on foods poor in lime salts, and in which a sort of pseudo-rickets occurs, with what occurs in rickets. Thus, if we feed a six weeks' puppy on raw horse flesh, bacon and distilled water, - a lime-short diet, - the animal will gain in size and weight. If we kill the animal at, say, the tenth day of the experiment, the bones will present the appearance of tenderness; there will be beading of the ribs and some swelling of the epiphyses, with bowing of the limbs. Cutting through the part of the end of a rib corresponding to the columnar zone of cartilage we shall find some thickening of the periosteum and such high degree of osteoporosis as to simulate a rachitic condition. But a careful examination of this osteoporosis will show that it is very different actually to the rachitic analogue, because in rickets an excessive preparation for ossification takes place in the form of a rich deposition of osteoid tissue which, however, remains almost uncalcified; whereas in osteoporosis there is but slight preparation much below the normal and, despite the scarcity of lime salts, this little is calcified; further, in osteoporosis in contradistinction to rickets, the cartilage matrix, notwithstanding the lime shortage, becomes calcified also. The bones, if examined when the disease is at its height, will be found to be remarkably light in weight; their water and inorganic matter and fat are much increased. Faulty secretive activity of the osteoblasts, deficiency of the earthy salts in the food, and a failure on the part of the digestive tract to absorb the essential salts are some of the theories advanced to account for this - reversion of the osteoblasts to fibroblastic form being, according to Hamilton, the most feasible explanation. The lactic acid theory has already been considered; and we may finally note that Wachsmuth (Jahr. f. Kinderh., Bd. 39) has stated that the conditions for the precipitation of lime salts in normal growing bones are the presence of fully developed cartilage cells and the existence of carbon dioxide in the tissues of the cartilage and bone in in sufficient quantity to hold the lime salts in solution or to redissolve them when precipitated, in rickets both of these essential conditions being incompletely fulfilled and in inverse proportion to the gravity of the disease; there is a plentiful development of the small-cell elements of the cartilage and scarcity of fully developed cells and at the same time the free carbon dioxide of the blood is increased, giving rise to poisoning by that compound or asphyxia of the bone in the course of its growth.

Pelvis.

A condition well known to and dreaded by experienced obstetricians is the so-called "rickety pelvis," which is one of the most troublesome of abnormal labour conditions, particularly in first confinements. It is the outcome of the softening of the general skeletal system which occurs in this disease, the mechanical forces bearing upon the pelvic girdle having certain untoward effects. The weight of the body above on this part is resisted by the limbs below, the pelvic bones are acted upon by the muscles attached to them and the imperfectly ossified bones can oppose only a perverted resistance thereto. The most typical rachitic condition is the flat pelvis with contracted diameters, especially the narrowed anterior-posterior one. But there may be a simple flat pelvis with normal transverse diameters, or the so-called pseudoosteomalacic pelvis, in which there is a uniform narrowing from mechanical pressure upon the softened bones. In the common form of rickety pelvis (the flat or generally contracted) there is forward and downward displacement of the sacrum between the iliac bones, with rotation on its transverse axis from the pressure of the body and the downward pull of the psoas muscles upon the spinal column and the upward pull of the spinal erectors on its posterior aspect. The tip of the sacrum and coccyx is thrown backwards and the bone itself lies more horizontally than normal. The sacrum is also bent, owing to the resistance of the sacrosciatic ligaments and muscles. The ~~iliac~~ bones assume an abnormal prominence and the innominate ones are too curved: so that the transverse diameter of the pelvis is relatively increased; but, as the entire pelvis is generally undersized, this diameter seldom exceeds or equals the normal transverse one. The bending of the innominate bones also throws the acetabula forwards: so that the counterpressure of the lower limbs acts more anteroposteriorly than under normal conditions. There is here enlargement of the cartilage and the inlet is shaped like an 8. The ischial tuberosities are enlarged also and pulled outwards and forwards, with widening of the arch of the pubis and increase of the pelvic outlet in transverse measurement. The entire pelvic girdle is generally tilted forwards and its bones are smaller and more brittle than they should be, though sometimes thicker and increased in weight. It may happen also that the pelvic canal is generally contracted in all directions from an attack of rickets; the outlook as regards future labour will then be far from favourable.

Skull.

This part shares in the general rickety condition of perverted ossification, with delay, atrophy and hyperplasia as in other flat bones obtaining. In the case of the occipito-parietal region especially, the delay in ossification may lead to a membranous substitute. Atrophy of the previously formed bone may lead to the possibility of the part yielding to the pressure of the fingers and the simulation of parchment and, on removal of the dura mater, small pit-like depressions may be found on the inner table. When hyperplasia occurs there will be the formation of hyperostoses or cranial bosses, which elevations are formed from the outer table and consist of red and vascular and spongy material readily yielding to the pressure of the examining fingers. On section it is seen that the trabecular spaces are much increased. These bosses are large and spongy and soft and when pressed upon by the finger give free vent to a sanio-serous discharge; they usually remain permanently and are characteristic features of the rickety skull, though they tend to organise and more rarely are completely absorbed to the exclusion of any adult tell-tale trace. Another important ricket indicator by no means infrequently found is the patchy thinning of the membranous bones first described by Elsässer and termed by him craniotabes. The occurrence of these patchy abnormalities has been ascribed to the pressure of the enlarged brain on the top of the skull covering it, as well as to the pressure of the head of the patient upon the pillow, though it seems more likely that they are the outcome of imperfect ossification of the bones concerned. To the touch they give the impression of parchment, the condition being most often seen in the parietals, occipitals and sometimes the temporal bones. The most characteristic patches are usually in the centre of the bone, which in some places consists only of a membranous tissue. The rickety child has a square, broad or prominent forehead, with bosses or thickenings on either side. There is a well-defined groove

between the two halves of the frontal bone; this is continued backwards to the anterior fontanelle, - which remains patent for two or three or even five or six years, instead of closing at from the eighteenth to the twentieth month, - and then between the parietal bones to the occiput. Another distinct groove may be discovered ~~upon the~~ temples and between the sutures in other parts. These grooves are due to the membranous condition of the cranial bones and the pressure of the veins underneath. The entire cranium may be so soft that it can be easily crushed by the fingers; it is considerably larger than normal and may even measure more around than the patient's thorax. But, in contrast to the hydrocephalic head, - which is almost circular in outline, - the skull of rickets is oblong. There is sometimes a marked asymmetry of the cranium, one side of the frontal or occipital region being much larger than the other. The face, when compared to the large skull, seems small; the upper jaw tends to become beak-like and the lower one squared, with some inversion of the anterior aspect. The base of the skull usually escapes, though sometimes softening may be found.

Spine.

This part, owing to the lax condition of the muscles and ligaments, plus the general debilitation existing, is curved and a characteristic deformity results; this is a kyphosis, most often seen in the mid-dorsal region, the curvature being most often towards the left side. It should be noted, however, that this humpback condition is not the angular bend seen in spinal caries, but a more or less regular convexity of the spine backwards, with slight lateral curvature sometimes, and exaggeration of the forward lumbar curve. In mild cases the implicated components of the spine are not markedly affected; but in a severe case, on section, there will be found a proliferating ~~zone~~ zone between the body of the vertebra and disc, the former being unduly vascular, spongy and soft.

Thorax.

The shape of the chest is usually markedly affected in rickets, particularly if the patient is subject to respiratory trouble. The beading of the ribs, - the so-called "rickety rosary"; - is not infrequently the first conspicuous sign of the changes occurring in the osseous system of the rickety child. These nodules or beads are to be found at the junction of the ribs with their cartilages and such enlargements may be present in the child at birth, - it is said also perhaps in the foetus, - and are very noticeable sometimes in the lower ribs, especially on their inner aspect. But the beadings never persist to adolescence, though very important early indications of the disease. There is also bending or kinking of the angles of the ribs and the shape of the chest is modified into the so-called "pigeon-breast" by the softening of the ribs and their consequent yielding to both atmospheric and mechanical pressure. A large groove, called after Harrison, runs on either side of the chest from the lower end of the sternum across the thorax to the posterior border of the axilla, just above the line of attachment of the diaphragm. It is said by some to be due to a yielding of the softened ribs at their point of least support; whereas others maintain that it does not correspond to the attachment of the midriff, but follows the upper border of the stomach, liver and spleen, it being not infrequently asymmetrical. One may also note another groove running vertically down the front of the chest internal to the junction of the ribs with their cartilages, the rickety rosary being found just outside this depression. Such grooves give rise to ~~top~~ prominence of the sternum, most appreciable under conditions of respiratory abnormality. The alteration in the shape of the chest causes a change in the position of the apex of the heart and there may be a white patch, due to friction, on the front of the left ventricle. Partial or greenstick fractures may give rise to enlargement of the angles of the ribs behind. We have seen that the beadings are more marked on the inner than the outer aspect of the ribs, which latter can be readily bent or kinked. The periosteum is thickened and, when the rib is broken, the two ends are held together by a superabundance of connective tissue. A longitudinal section through the cartilaginous end of such a rib shows that the beading mainly involves the columnar zone of the cartilage and that the epiphyseal line separating the bone from the cartilage is more irregular and wider than in health. A transverse section shows an almost entire absence of compact tissue and

that the bone is composed of a soft, spongy and gelatinous red-tinged material. The clavicles are thickened and twisted and sometimes the shoulder blades are distorted from their normal shape.

Brain

Long ago it was held that the brain of the rickety child is much larger than in health. I have applied this affirmation to my own patients, - young and adult, - and find that it is not the case. It is true that the head is increased in size, but this is due to the thickening of the bones, the brain itself remaining unchanged as regards dimensions. I have seen it stated that the brain is actually smaller than normal or, if large, due to fibrosis or associated hydrocephalus with ventricular effusion. Jenner speaks of a so-called hypertrophy of the white matter of the brain, a fibrosis consequent on hyperplasia in other organs; albuminoid infiltration is also described, and an imperfect development of the cerebral tissue has been alleged and held accountable for the mental dulness of rickety individuals sometimes observed, though often denied. Actual inflammation of the brain has been reported, but it seems to have no essential connection with rickets, there being some affection elsewhere responsible. Gee would have it that the brain is dwarfed in rickets, but this observation seems to be unusual and lacks confirmation.

Heart

We have seen that rickety deformity of the chest may have an influence upon this organ. Even in the absence of cardiac disease, it may be displaced and the apex forced outwards and downwards to the left. A white patch of thickened tissue may be produced on the pericardium where it rubs against a beaded rib, a friction sound being sometimes discoverable therefrom. It has been held that the favourite position for this white patch is the left ventricle just above the apex, not the right ventricle as in soldiers wearing constricting knapsacks daily. Though the heart may share in the general perversion of nutrition obtaining elsewhere, it is quite exceptional to find it the seat of organic disease not of secondary origination.

Ligaments and Muscles

These structures participate in the general body weakness resulting from malnutrition; they yield under the weight and pressure of the body and produce such well-known deformities as knock-knee, flat-foot, humpback, &c. Tension of the distended intestines may give rise to separation of the recti abdominis and "potbelly". Though there may be some obscuration of the precise outlines of the muscular fibres, there is nothing that can be regarded as characteristic upon microscopical examination. In general, the muscles lack the firmness and good development seen in health and are flabby and weak, so that perhaps the child is unable to walk, rise from bed or raise the head.

Liver

It used to be held that rickets led to enlargement of the liver to the extent of actual hypertrophy; but it is now known that this increase in size of the organ is only apparent and due to displacement of the organ into the huge abdomen, owing to contraction of the chest forcing the diaphragm downwards. There are several cases on record in which the liver dulness has descended to an inch below the navel, the chest being contracted and deformed from repeated bronchitic attacks. In rare instances there has been observed a diffuse fibrosis, with increase in the hepatic cells and a deficiency of lime salts - this hyperplasia being ascribed to a passive hyperaemia from pulmonary obstruction in the presence of an enfeebled heart and thoracic constriction resulting from some form of respiratory insufficiency. Again, this enlargement of the organ may be the outcome of existing syphilis or irritation of a circulating toxin. I have never seen a case of waxy degeneration of the liver in rickets, but have read of one somewhere. A fatty change may occur, but is not peculiar to the disease.

Lungs

Respiratory troubles are well known to be by no means infrequent occurrence in rickets, the subject of the latter

being very liable to suffer from various catarrhal conditions of the mucous membrane. Owing to the pressure of the sunken thoracic walls, those portions of the lungs lying beneath the lateral grooves or furrows described above are not infrequently in a state of collapse, while those part of the lung substance adjacent to the collapsed areas suffer from compensatory emphysema. The collapsed parts are always much larger if the patient suffers from whooping-cough or bronchopneumonia, which affections in themselves are by no means infrequently the cause of death.

Skin

There is a peculiar pallor noticeable in the subjects of this disease, though some of the patients have a certain amount of sallowness or actual pigmentation of the skin. There is not often much emaciation, obesity - or something approaching there-to - being the general rule with occasional exceptions. The patient, however, will lose flesh usually when there is some general trophic disturbance or such weakening condition as congenital syphilis. In convulsive cases there may be haemorrhages under the skin and it may happen that some part of the body, such as the lobe of the ear or the external genitals, fail to effect their full development. Some nervous or toxic influence seems to be responsible for the characteristic head sweating of rachitic children.

Spleen

The enlargement of the spleen which occurs in rickets is a well recognised condition. It is the seat of a general hyperplasia or fibrosis, the cells are enormously increased and the earthy salts undergo diminution. The cause of all this has been, and still is indeed, in dispute. The enlargement in question has by some observers been referred to the complications arising in the course of the affection, particularly bronchitis, gastrointestinal catarrh and anaemia. Sasuchin (Jahr. f. Kinderh., 1900, p. 297) thinks that it is the direct result of the disease and characteristic of it; he found that the enlargement existed in 15 per cent. or so of his 66 cases, the changes observed consisting of capsular thickening, connective tissue cell proliferation, arteritis, thickening of the walls of the arteries, atrophy and obliteration of the Malpighian bodies, with anaemia of the organ, all these changes being indicative of a chronic inflammatory process. In his control examinations of healthy children he could discover nothing wrong with the organ and he seems of the opinion that here again the presence of a toxin or toxins in the blood is responsible for a chronic irritation of the spleen culminating in hyperplasia. Syphilis or passive hyperaemia may, of course, be responsible for the condition, the increase in some instances being nearly three times the normal size. On the other hand, Hutchinson (Dis. of Childr.) found that in only 5 per cent. of his cases was the spleen enlarged during the lifetime of the patient, 4 per cent. being the observation of Cowan and McClure in a Glasgow institution, their conclusion being that the abnormality in question is referable to splenic anaemia or syphilis (Brit. Jour. Childr. Dis., Aug., 1906).

Stomach and Intestines

The stomach is by no means infrequently dilated to a considerable size and there is generally some evidence of gastrointestinal catarrh, which latter, indeed, is usually the anomaly for which treatment is solicited. The gastric and intestinal distension is due partly to insufficient support given by the flabby belly muscles and part, usually more frequently, to the evolution of gas occurring in the course of fermentative indigestion; in not a few of my cases, however, the affection seems to have been due to actual overfeeding.

Teeth

Rickety children are usually late in cutting their temporary teeth; when cut they are of poor constitution and liable to speedy decay; it is a fairly common thing to come across an infant of twelve months without a tooth in his head and when they do appear, they often do so very irregularly. The permanent teeth are usually just as unsatisfactory, though not always.

C L I N I C A L C O U R S E

It is a well-known fact that rickets undergoes its clinical evolution very slowly and is not easy to detect during the early days of its existence, though the time of its commencement may be suggested by certain **suspicious** occurrences. Trousseau, for instance amongst other observers, holds that the mental condition of the child is particularly suggestive of the early stage of the disease; he would have it that there is "a certain kind of **sadness** analogous to that observed in cerebral affections or, still better to indicate **its** nature, analogous to that **gloom** which takes possession of children" who are incubating the disease known as "cerebral fever. This mental gloom of rachitic patients depends, according to all appearances, upon their exquisite sensibility in every part of the body, which shows itself by eliciting distressing cries of pain when an attempt is made to raise up the young patient. The unfortunate little creature, who up to then may have been enchanted by the caresses lavished upon it, appears now to be afraid of them; even an approach to the bed upon which it lies, made as if with the object of changing its position, causes its countenance to anxiety and fear. This change in the child's character, this fear which it shows of having pain raised up by the pressure of the hand, this habitual stamp of **sadness** upon **its** countenance differ from anything seen at the commencement of serious maladies, particularly from the prodromata of cerebral fever. Indeed, in a child stricken by that cruel affection, we can still produce a transient cheerfulness, causing it for the moment to emerge from its habitual melancholy languor. In the rachitic child this is impossible. The more we try to excite it, or induce it to move, the more will it manifest impatience. It is heedless as to the games which formerly it was so fond of. This repugnance to the amusements of its age, this habitual **sadness** in a child which, with an appetite increased rather than diminished, loses flesh visibly, which has always an acceleration of the pulse coincident with profuse sweats, are symptoms, I say, which have a certain meaning; for the child does not cough and presents no signs which can give rise to a suspicion of the existence of pulmonary tuberculosis. These phenomena, in proportion **as** the child begins to walk, become more and more evident; the fever, at all events the acceleration of the pulse, continues; the skin is constantly covered with profuse sweat, whether the patient be sleeping or waking, or whether more or less covered than usual. The excessive perspiration is greatest in the head, and it is on that account necessary to change the child's pillowcase several times a day, so quickly do they become soaked! The newborn babe is at first in a very helpless condition and remains so for some weeks, without voluntary movement power and without will. Any movements which it makes are purely reflex and, until it has attained the age of some months, it cannot hold up its head nor walk nor take food with its hands. It should be able to hold up its head between three and four months, to sit up between the ninth and twelfth months or even earlier, to stand and walk between the twelfth and eighteenth months and to **imitate** baby-talk by the end of the second year. Each week there should be observed a definite progressive increase in weight, the birth-weight being doubled at the fifth month and trebled at the fifteenth. The child should cut its first tooth between the sixth and seventh month and, by the end of the third year, all the temporary teeth should be through the gums. Further, there is something wrong with a child which has not its anterior fontanelle closed between the eighteenth and twenty-fourth month. To such signs as these, Hutchinson has given the name "milestones," which does well for fixing the same upon the memory. He recognises three milestones - first, the age at which the child cuts its teeth; second, the time of closure of the anterior fontanelle; third, the age at which the erect posture is assumed. During the first years of life the child's organism undergoes rapid development, which, however, may be hindered or set back by comparatively trivial agencies, many additional months being afterwards spent in covering lost ground - for instance, a febrile seizure, a

chill or a day's intestinal catarrh. Scould rickets make its appearance, there is a retardation of all development processes; the teeth appear late and in irregular order, the anterior fontanelle may not close for months or years after the normal and much alarm or suspicion of paralytic or other serious disease may be occasioned by the late acquirement of articulation, erect posture or walking. In short, the patient is very late in passing the above-mentioned milestones. There is therefore an important indication for watchfulness during the first year of the infant's age, with a view to the prompt detection of any departure from the normal, particularly as regards weight, muscular condition, mental stability, appearance of the teeth and relation to the milestones. If such investigations be done regularly, it becomes possible to detect retardation of development or other rachitic anomalies perhaps long before the incipience of the disease would otherwise have been suspected; for it is an affection which is slow to affect the infant's nutrition, with consequent slight or misleading symptoms, the definite osseous anomalies being perhaps indefinitely postponed. Malnutrition and development retardation are easily observed during infancy when rickets is actually present; when the milestones are not passed at the proper time, there should be a careful examination for suspicious indications of the dyscrasia, no matter how slight. Mere wasting, however, should not be put down to rickets, for these so-called atrophic cases by no means invariably have that disease. It should also be borne in mind that rickety patients are apt to be large and fat, of excessive weight as patient food advertisements on the hoardings would seem to suggest. But it will be found that the muscles are soft and flabby, anaemia is present, there may be constipation or diarrhoea, there is a marked tendency to take cold and that respiratory or other catarrhs are by no means infrequently observed. Yet none of these signs are absolutely conclusive - merely suggestive under certain conditions. The insidious nature of the onset would seem almost peculiar to the malady. The child appears at first to be out of sorts, fretful and restless, particularly from bedtime to early morning. There is often a certain amount of dyspepsia, with flatulence, and not infrequently some diarrhoea, together with the passage of undigested milk in the stools, which are often of a greenish hue and of horrid odour. An early sign of the disease, to which I attach some importance, is an anaemia occurring in an otherwise robust infant; in such cases there is often general muscular weakness. The child is unable to sit up or hold its head erect at the proper age; there may be a tendency towards slight curvature of the vertebral column and, though there seems plenty of flesh upon the lower extremities, it does not seem able to stand up or, if it stands up, it soon falls exhausted to the floor. The general muscular weakness invades the abdominal wall, with the result that there is weakness of the muscular coating of the intestines, feeble peristalsis, belly inflation and constipation, which latter during the early days of infancy, even in breast-fed infants, is worthy of note as being possibly rachitic. As stated, some of these cases have much diarrhoea with stinking motions; they may suffer from gastritis and vomiting, with frequent attacks of colic or flatulence. Carmichael holds that frequency of micturition is an important early sign of rickets; others take a cue to diagnosis from the peculiar odour of the urine; whereas still others deny the same and considerable importance to acidity of reaction in the absence of increase of ammonia. Such signs as these, however, cannot be regarded as anything more than indications of nutritional disorder from faulty diet, cold, unsuitable hygiene, or the like; nevertheless, they should always be borne in mind for the reason stated above. It may also happen that bronchial catarrh is one of the earliest suggestive signs of the disease; such attacks may draw attention to the general condition of bad health obtaining and make one careful to avoid worse possibilities by not suffering them to undergo neglect. In some cases the first signs of early rickets seem to concentrate about the nervous system, though, in ignorance, perhaps put down by the parents to dentition or intestinal parasites. The child appears perhaps to be generally ailing, irritable, peevish and always tearful, nothing seeming to please it. It may suffer from spells of anger and may even seem to go into a kink, though attacks of laryngismus stridulus are not seen usually before the end of the twenty-fourth month. It may be extremely restless,

though this is not infrequently the outcome of flatulence and the general discomfort of indigestion or bony or muscular pains or perhaps the local tenderness of scorbutus. During the time that the child is in bed it becomes heated and therefore tries to throw off the bedclothes; it sweats profusely, especially about the head and neck, the head being tossed about so much in discomfort that the hair becomes thin through the boring action upon the pillow. This head-sweating has for ages been looked upon as a classical early indication of rickets; and such losses of sweat can be distinguished from those occurring in general debilitation by the fact that they are localised, being confined to special parts of the body, leaving the trunk and limbs unaffected; whereas, in the confusing condition just named, the perspiration breaks out all over the body. A rachitic infant has a very tender skin; it is therefore subject to irritations, erythema, intertrigo or perhaps outbreaks of mattery pimples. The child resents being handled and, even on the coldest night, the bed coverings are apt to become displaced. It is an uncommonly bad sleeper and keeps awake and howling with an exasperating perversity. It sometimes sleeps in extraordinary positions and may awake screaming; for, owing to its faulty digestion and irritable nervous system, it is the frequent subject of night-terrors. The bald patch which results from head-rolling during nocturnal restlessness is looked upon by many as one of the earliest signs of the disease. The child may also bang its head about and so bore the same into the pillow that a suspicion of meningitis may be raised. Such abnormalities as the above are generally observed to occur during the fourth to the sixth month, that is to say, long before the softening of the bones or the deformities give warning that rickets is actually present. In addition to a dislike for being handled, the child is apt to stare at its mother with a solemn and owl-like expression, which changes to one of alarm and despair when she comes closer to it. The teeth are late in cutting the gums; they often do so irregularly and with considerable constitutional disturbance. Therefore, between the sixth and seventh months the mouth should be daily examined and a failure of the lower incisors to come through the gums at that time should give warning of developmental irregularity and possible rickets. If during the next few months no teeth appear, or are few in number or cut in an irregular manner, the suspicion of rickets is practically always confirmed. But I have seen several cases in which the two lower central incisors were cut at the proper time, with a toothless condition of the rest of the gums until the end of the twelfth month. The occurrence of craniotabes is important. About the age of six months areas of softened bone may develop on the occipital, frontal and parietal bones which, however, are by no means pathognomonic signs of the disease as we shall presently see. During the first year of life other osseous changes are rib beading, lateral sinking in of the chest wall and epiphyseal enlargement; they make their appearance after the above-described early changes have lasted for some months and may now be briefly noted. It usually happens that the first-mentioned of these osseous anomalies closely follows the prodromal symptoms resulting from the digestive, respiratory or nervous affections; this rib-beading is the first definite sign discoverable at the stage of deformation. It consists of the occurrence of thickened nodules on each side of the chest forming, when seen collectively, the characteristic rickety rosary. The beading can at first only be detected by careful palpation with the fingers; but, with the slow progress of the disease, the thickened nodes become more prominent and detectable with macroscopic ease. These beadings are the outcome of a hyperplasia occurring at the junction of the ribs with their cartilages, and are best appreciable at the lower part of the chest between the fourth and ninth ribs. The thickening is even greater on the under surface of the ribs, where small patches of collapse of the lung substance may be produced by pressure thereon. I have often seen rib-beading present at the age of three months, though at that time they have seldom been large; I have also been able to detect the commencement of their future ripe presence as early as one month and dissection has revealed their presence in the infant at birth. These beads increase in size up to the end of the second year and then slowly diminish and take their departure. I have seldom seen them after the age of five years and I have never been able to detect them in the adult.

The thorax of the rachitic infant, being soft and deficient in elasticity, is sucked in on either side during inspiration, aided by atmospheric pressure. Vertical and lateral grooves are so formed and later become characteristic of the disease. The underlying lungs may show a read of collapse and compensatory emphysema, particularly in the presence of bronchial catarrh. The sternum projects forwards and there may be transverse sulci just below the nipple line on either side; in contrast to these transverse constrictions the lower ribs are apt to be everted above the large belly, which is so prominent a feature of the disease. Enlargement of the epiphyseal cartilage, especially at the wrists and ankles, is another important early indication of rickets and due to thickening at the line of ossification; it is usually of later occurrence than beading of the ribs, though both conditions are of early onset as rickets makes for the parts of the osseous system in which development is in most active progress and interferes with the formation of normal bone. There is something that is remarkably striking in this swollen appearance of the affected epiphyses, the associated articulations being peculiarly loose and flail-like through weakening of the controlling ligaments allowing of yielding to weight or sustained movement. Regarding the changes in the skeletal components occurring after the first year of life, it may be noted that the deformities become more conspicuous as will presently be seen. The disease does not spare a single bone in the body; for its effects may be seen in the head, the trunk and the extremities, i. e., in every part of the skeleton. The parts of the osseous system which suffer most, and the usual order of their frequency, are the head, ribs, spine and pelvis. The thickening of the limbs is usually observed before deformities appear in other parts of the body. Guérin says that it is an incontrovertible law, almost without exception, that rickets proceeds in the deformation of the skeleton from below upwards and that the deformity of the spine is the last to make its appearance. He gives the following as the order in which the various parts of the skeleton undergo deformity: First, swelling of the epiphyses of the lower limbs. Second, deviations of the knee; third, curvature of the tibia and fibula. Fourth, curvature of the femora. Fifth, swelling of the wrist and deviation of the pelvis. Sixth, swelling and deformity of the ribs, scapulae, clavicles and spine. But this law of Guérin's cannot be accepted nowadays; for, as a rule, deformities do not appear first in the lower extremities. Indeed, if craniotabes be taken as a symptom of rickets, the affection shows itself quite early in the cranial bones and the thickening of the epiphyses of the wrists, as well as the beading of the ribs, can usually be detected, - especially if the child is able to crawl, - some time before any swelling or curvature can be detected in the lower limbs. Chance, in his work on "Bodily Deformities," (211) remarks that he kept a record of six hundred cases and observed how many parts of the body were affected when the child was first brought for treatment, according to the mother. There was mention of the leg in 420, the arm in 202, the spine in 28, the chest in 20, the head in the same number. He says that the mother doubtless overlooked the enlargement of the wrists, which were always enlarged when the child first came under his observation. In 90 cases two parts of the skeleton were found affected at the same time - so that the number 600 should be increased. It has been my experience that mothers almost invariably fail to detect the early signs of the disease in the head, chest and ribs. Prominence of the forehead, slight flattening of the thorax, or a faint beading of the ribs, would pass unobserved; but, as soon as the child begins to walk, the soft bones bend and the presence of the disease is suspected. Then the mother, fearful lest the child should grow up with bowed or bandy-legs, brings it, perhaps in great haste, for medical attention. It is possible that this failure to detect the early deformities accounts for the large number of cases that have bending of the lower limbs when first observed. The same author also gives a table to show the frequency and the various deformities of the upper and lower limbs. In the latter the curvatures usually appear before those in the former are appreciable. But this only holds good if the child has been sitting up much in the pagoda attitude or has tried to walk early: otherwise deformities of the upper extremities may be the first to come under observation. In his 600 cases there was knock-knee in 396, curvatures of the tibia and fibula in 368, enlargement

of the ankles in 300, curvature of the femora in 142. Out of these cases 216 were knock-knees and curvatures and 170 curvatures without any affection of the knees. The pelvis was affected in only 16 cases. I have often seen rickets attack two or more parts of the skeleton at the same time - for instance, ~~the~~ the wrist and the tibia, the wrist and the ribs, or even the wrist and the tibia and thorax or the wrist and the knee and the head. In some cases, if a more minute examination of the bones is practised, signs of the disease will be found in nearly all parts of the body. The skeletal components affected by rickets ultimately become eburnated and acquire a condition of extreme hardness, massiveness and strength much in excess of the normal. The increased deposition of bone may be so great that the medullary canal may be partially obliterated. The disease seems to have a predilection for filling up the concavity of the bone with an osseous outgrowth, just as if nature were trying to splint up the weakest part. The eburnation and hyperostosis, when once commenced, does not cease for some time after the disappearance of the rachitic attack and until the earthy salts have been renewed and deposited. This activity may lead to the production of exostoses and bony spicules, often of great size, which are especially apt to occur on the spine of the tibia. Their presence not infrequently is the cause of considerable inconvenience, as in later life they may lead to stiffness or ankylosis of the articulations; indeed, comfortable movement may be impeded by a tendency to interlock. In the worst cases the deformities last for life, but in milder ones improvement occurs when the patient is from four to seven years of age; reports of school inspectors show that many children have the disease relics. Rickets injuriously affects the entire physique of the child; in bad cases the bones remain stunted, curved and bent; the joints are "knocked" and the muscles are poorly developed. In the worst cases the evils produced in the early years never pass away, but leave the individual in some respect physically unsound, incapacity for hard work being perhaps due to debility or engrafted tuberculosis or bowlegs and the like; flat-foot would also disqualify for the army, post-office and so forth. If the rachitic child is able to walk, its gait is often waddling, duck-like or stumbling. If fat and heavy with prominent calves and some spinal lordosis from muscular weakness, the presence of pseudohypertrophic paralysis may be suggested; but in rickets pure and simple the knee-jerks are present - not absent as in the condition just named; and the differentiation between the two affections is aided by the fact that in pseudohypertrophic paralysis the child levers itself up by placing its hands upon its knees and finds the locomatory acts of considerable difficulty. Although in severe attacks of rickets the child seems to be very ill, the danger to life is not in proportion to the intensity of the phenomena observed. The disease in itself is seldom, - some writers say never, - fatal; for it is the complications which are so apt to carry off the patient, particularly diseases of the bronchi, lungs, intestines and nervous system.

VARIETIES

There are so many expressions clinically of the rachitic dyscrasia that it has been considered advisable by some to consider the disease under the above heading; whereas others have devoted considerable ingenuity to the invention of classifications according to the predominance of certain symptoms in certain cases. There seems utility in considering the various types of the affection in appropriate detail:

Acute Form

This variety of rickets, which is also termed multiple epiphysitis or multiple periostitis of the long-bone joints, - is characterised by a rapid onset of illness culminating in a condition of marasmus and cachexia; but, as it is now generally recognised as scurvy, not rickets, it is scarcely deserving of inclusion in rachitic classifications. In it, the changes, which in the ordinary form of rickets are of very slow evolution, occur rapidly, the patient perhaps seeming quite well prior to its initial manifestations. There has been a considerable amount of disputation in the past regarding its nature, some insisting that it is a disease by itself and an expression of a constitutional predisposition, others maintaining that it is an

acute form of rickets or an actual inflammation of the bones. As stated, it seems probable and is usually held to be not pure rickets, but rickets in association with infantile scurvy, which was formerly known as haemorrhagic scurvy or scurvy rickets. Glisson spoke of the occurrence of scurvy in connection with rickets, but the disease seems to have been lost sight of until two centuries later when Müller described some cases under the title of "acute rickets" - a term which was adopted by various continental writers of note. In 1873, Jalland reported a case, a child of 10 months, which he regarded as identical with the scurvy of adults. Another one was reported three years later by Thomas Smith, who described it as an instance of haemorrhagic periostitis of several of the long bones with separation of the epiphyses, but failed to recognise its scorbutic nature. Cheadle was the first to point out the identity of such cases with the scurvy of adults, which he did in 1878. He says that the condition may be produced by a diet consisting solely of preserved foods, such as dried milk or peptonised milk, dried milk or so-called humanised milk. In older children the affection occasionally occurs when they are kept on a diet lacking vegetables. The principal lesions are to be found in the bones. The periosteum of an affected bone is thickened, vascular and separated from the bone by blood-clot. There are no inflammatory signs; the muscles of the affected region may be infiltrated with serum or blood. There is considerable rarefaction of the bones, the cancellous tissue being very porous and the normal marrow replaced by a highly vascular connective tissue. This rarefaction is said to be due to delayed ossification. The serous cavities, joints, etc., may show haemorrhages. The occurrence of periosteal haemorrhage and tenderness, haemorrhagic stomatitis, and sometimes of haematuria, characterises this disease. Infantile scurvy usually occurs between the age of seven months and two years. The child cries whenever it is handled or disturbed. There is marked tenderness of one or more of the limbs, especially the lower ones; there is perhaps local swelling or oedema of the feet or thighs; and there not infrequently is also a conspicuous redness under the nails. The legs hang down as if paralysed, the infant not moving them on account of pain and tenderness. When a tooth is out, the surrounding gum is seen to be purple and swollen. There is often more or less bright blood in the urine or faeces or contusion-like marks on the surface of the body. Bleedings may take place into the orbital cavity or viscera and the patient may appear to be very anaemic at times. It is easy to recognise severe instances of the disease, but the mild or early ones may be overlooked; the kind of diet and the result of treatment are of assistance in differentiation. Infantile paralysis, which may be mistaken for the condition, shows no swelling of the affected limb and there is an absence of the other signs of scurvy. Below the age of one year rheumatism is seldom seen. The swelling in infantile scurvy is not limited to the locality of the epiphyses, which fact distinguishes it from epiphysitis. In the case of periostitis there may be some difficulty in the diagnosis, if there are no haemorrhages in the gums and if other distinctive signs of scurvy are absent. A high temperature would probably indicate periostitis, a normal one being the usual happening in scurvy. The prognosis is usually good, as under proper feeding there is a comparatively rapid recovery.

Catarrhal Form

Cases of this type are much troubled by attacks of bronchitis, with appearance of the characteristic deformities at the tenth month or later.

Congenital Form

It may happen that an infant is born rachitic, though there is some uncertainty as to the frequency of this. The lesions are similar to those seen in the ordinary form of the disease, the special feature, however, being the frequency of fractures, often complete, of the long bones. Such fractures have been known to occur within the uterus; Ashby tells of an instance in which deformity of the chest was present at the age of fourteen days, together with several fractures of the long bones. Sometimes, however, the bones are merely bent. The cranium may be incompletely ossified. Sometimes the fractures unite; but in not a few instances they do not or are very tardy in their healing.

Foetal Form

This variety of rickets has also been designated "achondroplasia," "chondrodystrophia foetalis" and "foetal cretinism". But in considering it one should bear in mind the fact that the latest researches would apparently tend to negative the alleged occurrence of rickets whilst the child is in the uterus, as well as to warrant the classification of such alleged instances ~~under~~ the heading of osteoporosis or osteogenesis imperfecta. Comby thinks that no one is born rachitic, rather one becomes rachitic after birth; he would also have it that foetal rickets, - with its deformities, its multiple fractures and its anomalies of ossification, - presents a number of unrelated phenomena which do not belong to rickets as it is ordinarily known. It is likewise held that many of the children born under-sized and deformed, and described as foetal rickets, are probably instances of achondroplasia foetalis, which is disease described by Parrot, who maintained that a defect in the development of cartilaginous bone was responsible for the same. Even before this observation the malady was regarded as foetal rickets and, as far back as 1856, Virchow drew attention to the large head and short limbs of some of the infants which he studied at the time of birth. Fifteen years later, Winkler differentiated the disease from ordinary rickets and proposed for it the name rachitis micromiela; Kauffmann called the disease chondrodystrophia foetalis and Depaul insisted that achondroplasia and rickets are quite distinct maladies, Pierre Marie supplying contrast measurements of the body in health and the former disease. Some twenty years ago a case was reported of a newborn child, apparently rachitic, which presented a marked rarefaction of the bones, with a thin layer of osseous material surrounding a dilated medullary canal full of marrow. There is extensive resorption of bone, which condition is not present in ordinary rickets. Histologists have demonstrated that the defective growth is due to a rudimentary and irregular arrangement of the proliferating cartilage cells, with ingrowth of connective tissue from the periosteum leading to separation of the epiphysis and shaft; there may be wide separation of the cranial sutures. In the so-called foetal rickets the limbs are short, stunted and bent; the head is large, but its bones, as well as those of the thorax, present no deformities; the fingers are equally long and spread out at their distal extremities into what the French term the "trident hand". It would seem, then, that a proportion of the cases of foetal rickets should be classified as achondroplasia, which also must be regarded as a disease of foetal cartilage preceding the period of bone formation and without relation to the true rachitic disease. When a child born with the so-called foetal rickets is submitted to examination, it is found that the bones of the skull, ribs and limbs are imperfectly calcified, with enlargement of the epiphyses, and that they sustain fractures from the most trivial causes, no matter how carefully handled they may be. I have knowledge of case in which an infant, apparently well-nourished at birth, during the first fortnight of life sustained fracture of the right humerus and radius, left humerus, ulna and femur and, when six weeks old, during examination had its right thigh-bone broken; when a fortnight old signs of rickets were observed, as well as craniotabes of the parietal and occipital bones, softened ribs, broad vertical grooving of the thorax, beaded ribs, but no epiphyseal enlargement. Chaussin reports the interesting case of a foetus in which there were no less than 43 fractures; and apparently such instances as these should be regarded as osteogenesis imperfecta, not true rachitis foetalis. Whilst upon the subject of animal experimentation I showed that parent rats, when fed with excessive meat, developed symptoms identical with those of rickets; though the changes seemed to be those present in human rickets to the naked-eye, the microscope showed that there was a great difference, the epiphyseal lines no presenting the irregular dentate appearance so characteristic of this affection. Similar experiments have taken the form of feeding a puppy on horseflesh, bacon and distilled-water, the result being that the animal increased in weight, had epiphyseal enlargement and bending of the bones; the disease produced resembled rickets, though the bones were very spongy and the production of true rickets very doubtful. The conclusion forthcoming from a careful study of the literature of the disease is that the evidence is against the occurrence

of rickets whilst the child is in the uterus, the essential characters of osteogenesis imperfecta being, first, a process of bone formation everywhere checked and of an abnormal kind; second, deposition of cartilage greater than normal and much less of bone; third, formation of periosteal bone, but of an abnormal and incomplete character; fourth, deformities due to imperfectly united fractures and curving and bending of the bones from the disease itself simulating rickets at first sight. In rickets, on the other hand, there is a very extensive preparation for ossification, a rich formation of osteoid tissue, - which with no lack of lime salts remains uncalcified; whereas in osteoporosis there is very little preparation for ossification, but a lack of lime salts, calcification of the osteoid tissue and of the cartilaginous matrix. Foetal rickets is of uncertain etiology, though there would seem to be a close connection between the health of the mother during pregnancy and the condition of the child at birth; in short an hereditary influence has been affirmed; for the child may be born with this malady if the mother has suffered from any acute illness or been subjected to want or starvation. Experiments on rats would seem to support this idea and the theory that causes are in operation in the mother ready to operate adversely at the conception or birth of the foetus is strongly suggested by cases in which a child, born of a tuberculous mother fed on excessive meat-juice for months before marriage, developed a similar disease. Charrin and Gley claim to have produced congenital rickets in a rabbit by injecting its parents with the toxins of diphtheria and blue pus containing the bacillus pyocyaneus. According to Lovett and Nicholls, special attention should be directed to the condition of the thyroid, adrenal and other glands, because children may show a cretinoid condition of the face and have small adrenal glands - the further suggestion being made that, in addition to examination of their structure, due attention should be paid to the weight of these organs in relation to what is normal. The disease is admittedly rare and not amenable to treatment, the patient seldom exceeding three feet in height when grown up to an adult, and in childhood resembling a basset hound. To sum up: this so-called variety of rickets is characterised by defective growth of the bones and dwarfing and deformity of the extremities of perhaps lifelong duration; the term has in the past been used to designate various conditions seen in the foetus and newborn babe, resembling rickets, though many of them have been shown to have nothing in common with that disease; the affection is of obscure causation, though it is supposed to have some hereditary tendency; and is observed in animals and man; it occurs during early intrauterine life and runs its course mainly between the third and sixth months of foetal existence; the consequent deformities consist of the arms and legs being half their normal length, the bones being thick and short and their normal curves exaggerated; the fingers diverge and form the so-called "main en trident"; with a basset-hound appearance of the child; the head is large and the nose is tilted up through the shortening of the base of the skull.

Gastrointestinal Form

Cases which show a tendency to stomach troubles, intestinal catarrh or constipation, with ultimate osseous anomalies, have by some been described under this heading.

Late Form

Rachitis tarda is a synonym for this variety of rickets, which affection is almost always seen during infancy or early childhood - the former being an uncommon form in which there occurs a late appearance and a lengthy persistence of the characteristic anomalies observed. The affection may be seen in its incipience at the time of puberty or even in adolescence. At the former period both growth and ossification attain an intensity only equalled or surpassed in the first two years of life. The condition must not be confused with what has been termed by some "prolonged rickets," in which cases of the infantile disorder persist, perhaps with remission intervals, up to or beyond puberty. Clutton holds that adolescent rickets is pathologically the same disease as infantile rickets, modified by the age of the patient, other observers not being agreed as to whether the adult form is a continuation of the ordinary early disease or a new development thereof. Against the view that it is a recrudescence of early rickets is the fact that the bones after recovery from this disease are usually eburnated and

sclerosed, a condition in which it is not easy to suppose that a relapse would occur later on. If it be a continuation of infantile rickets, then the first attack must be very mild, unnoticed and with no tendency to recovery; and if it is an entirely new development, then the origin of rickets at any age from infancy to adolescence must be assumed. Rickets is an affection associated with growth and development and it is somewhat rare for it to occur after the ossification of the epiphyses. As a rule, late rickets is confined to the growing limbs; the head is scarcely affected, because there ~~is~~ ^{is} a completion of growth and expansion in that disease. In some cases there is a history of rickets during early years, followed by a long period of relapseless health and the appearance of late rickets at the puberty age. Some writers have shown a liking for a classification of cases into those in which rickets appears in adolescence, - rachitis tarda, - and those in which there is a relapse or a recrudescence of the disease at puberty, - secondary rachitis tarda, - after complete recovery from an attack in infancy. Other -s, however, favour a classification into three varieties, viz., first, cases identical with ordinary rickets; second, cases with atrophy and fragility of the bones like osteomalacia, such cases constituting the majority; and, third, cases unlike the above, but possessed of peculiar features of their own. Trousseau regarded rickets and osteomalacia and rickets as one and the same disease. The chief complaint in a case of late rickets occurring before puberty is of a condition of fatigue induced in the child, even on very slight exertion, which may be accompanied by excessive perspirations. The muscles and ligaments are lax, the epiphyses slowly enlarge at the wrists and ankles, there is stiffness of the lower extremities and some muscular and osseous pain is experienced. Later on the characteristic rachitic deformities make their appearance, particularly lateral curvature of the spine, knock-knee, flat-foot, beading of the ribs, a waddling gait, or perhaps a mode of walking like that seen in disease of the hip. There is flatness of the pelvis as in early rickets and the muscles may be hypertrophied, with an undersized stature. The nervous system is does not escape; the child is irritable, restless whilst in bed, unfit for school or other occupation, and may become hypochondriac or much depressed. Roos says that the cardinal symptom of late rickets is enlargement of the epiphyses and that one is not justified in diagnosing the presence of the disease merely from the presence of such simple deformities as knock-knee; whereas Mickulicz maintains that genu valgum adolescentium so-called is usually only a characteristic of late rickets; he could find no constant relation between knock-knee, the second decade of life and long periods of standing or the carrying of heavy weights which some have blamed for the origination of the malady. In the few cases which I have seen the invasion of the affection was usually accompanied by pain in the limbs, sometimes very severe, the weakness of the legs being so pronounced in some that the patient from the first could not stand without assistance, and enlargement of the epiphyses, rib beading and chest deformity being also present, with a waddling, awkward gait. During the active stage of the disease an irregular rise of temperature may be seen and the urine may show an excess of lime salts. There is some uncertainty regarding its causation and it is not always possible to diagnose the presence of rickets clinically, though the Röntgen rays may be useful to that end. Overexertion and fatigue have been blamed for its origination and would seem to explain its localisations to some extent. Bad feeding and defects in hygiene may unfavourably influence the malady, but cannot be regarded as such powerful factors in the etiology as in the case of the infantile disease. Late rickets not infrequently appears at from twelve to fourteen years, which is admittedly the time of great intellectual stress, owing to the demands of educationalists upon the child; hence it has been suggested that the higher nerve centres unemployed during infancy become inordinately developed during puberty and interfere with metabolism, the intelligence of the patients being quite up to or above the normal standard of their age. My own opinion is that prolonged mental strain may be a factor of by no means inconsiderable importance. Some of these cases show premature sexual development: so that the element of sexual condition may have to be taken into consideration. Marsden (Lancet, 1904, p. 1835) tells of a case of late rickets occurring in a young woman of

of nineteen years of age. The use of the X-rays revealed a striking increase in the breadth of the epiphyseal cartilages and our observer's suggestion was that ~~this~~ might be a more trustworthy indication of late rickets than the enlargement of the ends of the long bones. He classified late rickets as primary and secondary disseminated and primary and secondary localised, stating that the affection was due to an absence of the conditions necessary for healthy growth and that faulty nutrition or bad hygienic surroundings, by their influence upon the blood, so altered its characters that it was prevented from fulfilling its proper function and thus giving rise to rachitic troubles. Pollosson and Broea in a case of cured rickets observed the persistence of pearls of cartilage in the middle of the epiphyses and suggest that these granules may explain the late deformities of the disease. Some writers have affirmed that late rickets is allied to osteomalacia; whereas others regard it as relapsed rickets or an associate of masturbation and albuminuria. It has even been described as osteomalacia and it is therefore important that no confusion should occur on that account. Goodhardt and Still affirm the identity of the two affections, stating that different processes are in the ascendant in each; whereas Ashby and Wright (Dis. of Childr., p. 216) insist upon a separation of the two, as the patients never succumb to rickets pure and simple, the processes become arrested and it does not occur under the conditions met with, nor attack the parts affected in osteomalacia; they hold that it is just as certain that masturbation and albuminuria are not invariable etiological conditions and maintain that it is due simply to poor health, bad air, long standing, poor food, - in short, to bad hygienic conditions at the time when growth is active in the limbs, - that is to say, mainly to those causes which produce rickets in earlier life. Osteomalacia is an affection of adolescence, it is seen in women more than men, - ten times as often, - and is usually encountered between twenty-five and thirty years of age. It breaks out more often in pregnant women than in the non-pregnant, more especially in multiparae, the first indications of the malady appearing in the pelvis, though all the bones of the body, - particularly the limbs, - may be affected. The sacral promontory is pushed forwards and the pressure of the femora approximates the pubic bones, with conversion of the pelvis into a sort of beak. The bone becomes soft and decalcified, as if it had been immersed in hydrochloric acid, and there is subsequent absorption of the animal basis by the increasing marrow. The result of the absorption of the lime salts is that the bones are merely thin-walled, brittle shells, full of red marrow, or the bone may disappear completely and leave merely a periosteal covering for the marrow. In this disease there is an even greater decalcification than in rickets and an excessive amount of fat is also present. The urine always contains an excess of lime salts. Fractures are common, but they heal readily so long as bone is present. The malady may be distinguished from late rickets by its frequent occurrence in pregnant women, by the fragile shells of bone filled with plenteous marrow and excessive fat, by the characteristic beak-like pelvis not simulating the peculiar rachitic one, and by the normal appearance of the epiphyses, whereas in late rickets the epiphyseal line is broadened. It is also necessary to distinguish late rickets from persistent rickets, which has been said to be common in orthopaedic hospitals in children from eight years of age up to about fourteen; this distinction is affected by a careful inquiry into the history of the case. The treatment is that of ordinary rickets, such as by rest in bed, dietetic correction, fresh air, salt baths, phosphorus and cod-liver oil; even in the absence of syphilis, iodide of potassium may do good or it may not; thyroid extract is a failure; surgical correction of the deformities should be instituted after the disappearance of the active signs. On the whole, the treatment is not satisfactory, though much may be done by palliative means.

Osseous Form

In this so-called type there is an absence of the usual early symptoms, the child, though perhaps late in walking, remaining healthy and robust. But on examination of the skeletal parts it is seen that the brunt of the attack has fallen upon the bones, characteristic deformities of the thigh and leg-bones being seen

Pseudoparalytic Form

In this variety of the affection the patient seem to be in tolerably good health, but cannot stand up; the muscles are flabby and toneless and the joints give one the impression of being as loose as a flail. It is sometimes called the "acrobatic form" of rickets. No signs of true paralysis are present in these cases.

COMPLICATIONS

Though the various complicating conditions which may be encountered in the course of a rachitic attack will receive detailed consideration in the analytical section, a due regard to orderliness of narration demands that at least some of them should meet with brief mention at this point. Such adventitious affections, we shall see, prejudice adversely the otherwise normal evolution of the disease and perhaps prove positively dangerous, particularly if the respiratory or gastrointestinal systems be involved. It should always be borne in mind that the tissues of the patient are remarkably vulnerable and constitute a favourable nidus for the development of infections and other intercurrent maladies and that they are apt to be fatal in a large proportion of the cases. It is very necessary to guard the child against contracting summer diarrhoea, which is a very weakening disease often ending in death. When influenza is about, these patients suffer severely and present a high mortality. In the course of recovery from rickets, as well as during the actual attack, one or other of the acute diseases of early life, - such as scarlatina or measles or whooping-cough, - may occur and prove serious complications. Indeed, actual observation has convinced me that rickety children are peculiarly predisposed to infectious diseases; they seem to catch all the ailments that are about and in nearly all my cases of measles, scarlatina, whooping-cough and diphtheria in young patients I have been able to elicit a history or presence of rachitic disease. I have seen it stated in the literature, as the opinion of a competent observer, that rickets is not so much a disease of the bones as a perversion of metabolism and that, as renal disease is associated with gout, in a similar way respiratory and gastrointestinal catarrh may be associated with rickets - hence the urgent importance of extreme watchfulness, the more so as so many rachitic children die annually from intercurrent diseases. Convulsions, too, are of very grave import when manifesting themselves in a rickety child. The tonsils of a rickety patient may act as a trap for the diphtheria bacillus and the toxin of the same, acting upon the sympathetic and motor nerves, renders this complication extremely dangerous in the presence of circulatory or respiratory insufficiencies due to rickets, as is the case in influenza. Scarlet fever also is apt to be of severe evolution when rickets is already present; convulsions, early or late in the disease, would be very likely to ensue and a lethal issue would be still further favoured by any condition of gastrointestinal catarrh existing. In the case of a rickety child measles, - which is characterised usually by much catarrh of the conjunctiva and nose, with sometimes bronchitis or bronchopneumonia, - is comparatively seldom fatal in most cases if the disease is trivial; but when pronounced rickets is existing, capillary bronchitis or lung disease, - acute or chronic, - may put an end to the patient's debilitated existence, finishing up perhaps with convulsions, which are more likely to prove dangerous in themselves at such times. The little patient has little or no chance of recovery if pulmonary tuberculosis be engrafted on the rachitic condition; nevertheless it is a fortunate circumstance that one rarely finds tuberculosis occur at the same time as this disease, it being alleged that the circulating toxin has some germicidal effect upon the B. tuberculosis; but, should this complication or sequel occur, the badly nourished infant would, of course, have little chance of surviving the conjoint machinations of the two factors. Pertussis is another very dangerous complication or sequela of rickets, largely because of the softening of the ribs and enfeebled respiratory mechanism, the patient being unable to cope with the sudden spasmodic coughing fits (or ward off the threatening pulmonary collapse) and perhaps die in the middle of one of them. It may here also be

mentioned that there is a possibility many of the deaths, now classified as due to delayed chloroform poisoning, have rickets as their predisposing factor, to which the chloroform has given the finishing-stroke. Several such cases have been reported, - mostly operations on the bones for the correction of rachitic deformities, - in which symptoms pointing to delayed poisoning by that drug occurred. It is possible that the toxæmia of rickets produces some change in the tissues or that there may be some faulty condition of the liver which renders the child peculiarly sensitive to the narcosis of the chloroform inhalation, though it may only be that rickety children, in common with all others of lymphatic temperament, take the drug very unsatisfactorily. Therefore, whenever a rickety child has to be submitted to operation, the above-mentioned possibility should not be overlooked.

ANALYSIS OF PHENOMENA

Abdominal Enlargement

The so-called "pot-belly" of rickets is usually supposed to be very characteristic of the disease. The debilitation and flabbiness of the muscular system throughout the body generally obtaining has its effects very conspicuous in the case of the abdominal wall, as well as internally in the case of the intestines. In common with the other muscles of the body, the recti abdominis are weak, flabby and ill-fitted for their work and, at the same time, are still further strained and rendered incompetent by the pressure of the protuberant stomach and intestines. They not infrequently become unduly stretched, with the result that umbilical hernia occurs or there is diastasis of the muscles in the middle line of the belly. Abdominal enlargement may pass unnoticed in infants, but may be appreciated if the child is raised by the head and shoulders in such a way as to put the recti muscles on the stretch. Atony of muscles is an early sign of rickets and so this diastasis, - which may be a half to one inch in width, - may be found as early as the fifth month of life, long before the presence of rickets is suggested by the implication of the osseous system. It is said that this diastasis has been detected in cases which have shown rachitic symptoms at birth, though it is very doubtful that any hereditary element therein obtains. Although one of the earliest signs of rickets, it is slow to take its departure; it may persist for years, as the muscles are very slow in recovering their normal tonicity and during adult life this abdominal weakness may be the cause partially of various herniæ. The primary cause of the diastasis is arrest of development and nutrition of the muscles, the secondary cause the pressure of the stomach and intestines, inflated with gas, upon the weak and flabby muscles of the belly wall; the condition, however, may occur in healthy children and disappear about the age of puberty. In the normal child closure takes place from below upwards and, as a rule, the diastasis is only seen above the umbilicus; whereas it involves the whole extent of the abdominal wall in the rachitic infant. In itself I do not regard it as pathognomonic of rickets, as it has been shown to be present in a large proportion of healthy children. The large size of the belly, - alleged by some to be almost always a symptom of rickets, - early attracts the attention of the parents, the mother bringing her infant for treatment perhaps solely because it has a "pot-belly" with some slight digestive disorder. Such an abdomen is usually tense and tympanitic to percussion and, as we have seen, is the result of manifold condition acting together - its production being contributed to by the atony of the abdominal muscles, the depression of the diaphragm, the contraction of the thorax, the enlargement of the liver and spleen, the dilatation of the stomach and intestines and the shallowness of the pelvis. According to one of the older writers, the large belly of rickets is not due to deformity of the chest, diaphragm depression or enlarged viscera, but to dilatation of the stomach - the probability, however, being that the atonic condition of the belly muscles is an important factor in maintaining the abnormality in question. The enlargement is uniform and chronic indigestion, - gastric or intestinal or both, - may indefinitely prolong it as a troublesome condition

Adenopathy

The glands of the body generally in rickety cases may take on a tendency to enlargement, the hampering of free inspiration and, it may be, the impurity of the atmosphere of the surroundings, causing not a few of these cases to develop adenoid growths in the nasopharynx, with hypertrophy of the tonsils and swollen cervical lymph nodes. There may also be considerable tumefaction and enlargement of the glands of the trachea, bronchi and mediastinum, particularly if bronchial catarrh is much in evidence. Percussion may reveal the presence of a dull area behind the manubrium sterni and, if the thymus is increased in size, - as is often the case in rickets, - it can be mapped out upwards into the neck.

Blood

Owing to the frequent presence of anaemia in rickety cases, it is very common to observe the blood changes peculiar to that complication, the patients presenting the waxy, greenish-yellow tint so often seen in anaemic females. In such cases enlargement of the spleen is common. There is a diminution in the number of the red blood-corpuscles, nucleated red ones may be seen and there is sometimes a slight degree of leucocytosis. The blood conditions may be complicated by the so-called splenic anaemia or pseudoleukaemia infantum, which has been held by some to be the result of syphilis, though occasionally quite independent of it. Jacobi says that many more blood cells are required to fill the arteries when wide than when narrow and adds that these vessels in rickets are wide and the blood pressure low. If, therefore, the formation of blood cells is hampered by any disease of the digestive or blood-making organs, the tissues will then show a relative increase in the percentage of water. I have had many opportunities of showing this fact to be true in the case of rickety children. The anaemia which so often troubles the rachitic man may be set up by an excess of bad food resulting in auto-intoxication, by any chilling of the body surface causing destruction of the blood-corpuscles and, in the later stages, by any overtaking of the strength of the ill-nourished and debilitated patient. Such an affection as rickets, which interferes with nutrition to so great an extent, may cause changes in the blood that makes it revert to the more infantile form, in which there is a high proportion of lymphocytes and only a small number of polynuclear cells. Hutchinson refers to a case in which the examination of the blood seemed to indicate the presence of a chlorosis. In general, the blood has no special characteristics, there being by no means always a slight increase in the polymorphs.

Blood Vessels

In rickets the various arteries throughout the body are apt to undergo dilatation; the blood pressure is low; there is stasis in the principal viscera and at the periphery, the circulation being generally retarded and sluggish. Beneke says that he has found the pulmonary artery in rickets abnormally large, this fact being advanced towards explanation of the pulmonary complications arising from disturbance of the circulation in the lungs. Under normal conditions the circulation of blood and lymph is carried on mainly by the cardiac muscular contractions and pressure; in rickets the muscles of the abdominal wall is weak and atonic, as also is the muscular coating of the intestines. Owing to this loss of muscular power, there is capillary stagnation, the tissues becoming bathed in an excess of blood and lymph. Such congestion is seen in all the viscera drained by the branches of the portal vein, that is to say, the inferior and superior vena cava, the splenic and gastric trunks. That formed by their union, the vena portae, enters the liver and ramifies through its substance; and its branches emerge from the organ as the hepatic veins to join the inferior vena cava. The excess of liquid in the tissues gives rise to hyperplasia, with the result that the spleen becomes enlarged and the liver also when heart anomalies exist. The congestion of the viscera will be in proportion to the weakness and atony of the affected muscles, there being a catarrhal condition of the stomach and bowels when such are involved. The occasional occurrence of piles can thus be explained, the same being less common than it is owing to the patient lying down so much from sheer necessity through weakness almost always.

Chest

In estimating the extent of the thoracic deformity it is necessary to bear in mind certain anatomical considerations. The chest of the adult is of an oval outline, whereas during infancy it is cylindrical or almost circular, the ribs and cartilages being more horizontal, the collar-bones elevated and the neck short. The upper part of the chest contains the heart, lungs and thymus, while in the lower portion the abdominal contents crowd into the space under diaphragm, which has a higher dome than in adults. The characteristic rickety "pot-belly" produces a broadening of the base of the thorax, which becomes still further exaggerated as the disease becomes worse. The adult thorax is a bony case, but in the child soft and flexible and therefore easily affected by the atmospheric pressure or any obstruction to respiration. The infantile chest is very light and pliable and there is great mobility of the ribs and cartilages; it yields readily to atmospheric pressure, but, unlike the chest of the adult, has no resiliency and little elastic recoil. The act of respiration is due to muscular action and during inspiration the intrapulmonary pressure is reduced and air enters the lungs. The capacity of the chest is increased, its vertical diameter by descent and elevation of the diaphragm. Elevation and outward rotation of the ribs increase the transverse diameter, while the anteroposterior one is increased by a movement upwards and outwards of the ribs, costal cartilages and sternum. The muscles of inspiration are the diaphragm, the external intercostals, - which elevate all the ribs except the first, - the intercartilaginous portions of the internal intercostals, the scalenus anticus, the serratus posticus superior and the levatores costarum. Thoracic expansion in infants is mostly diaphragmatic and abdominal and the act of expiration passive. On cessation of inspiration the distended lung partially ceases on account of its elasticity and in so doing draws in the chest wall which, owing to its elasticity, tends to return to the condition obtaining prior to inspiration. The action of gravity on the chest wall and retraction of the muscles of the belly assist. The pressure of the inspired air is not sufficient normally to bring about equilibrium to the pressure of the outside air on the chest wall. This is evident in such affections as pneumothorax, in which air enters the pleural cavity and causes collapse of pulmonary tissue or prevents the complete expansion of the lung, as in cases in which adhesions have prevented complete collapse. Normally, however, the chest wall resists the atmospheric pressure and establishes an equilibrium. A child's lungs are relatively smaller than an adult's, in proportion to the size of the heart and also because the other viscera require more space; the pulmonary tissue is exceedingly delicate and elastic, it is easily affected by obstructive catarrh or pressure from without, so that very little causes a condition of emphysema or collapse. In the early weeks of life the chest measures slightly less, - that is, about half an inch, - than the head, but its circumference should be greater by the end of the first year. In rickets it is seen that the head is larger than the thorax, which is narrow, flattened and deformed, its stunted condition being markedly emphasised by the "pot-belly" below. The chest loses its cylindrical shape and approximates more closely to the quadripedal type. A cyrtometer tracing of the chest, through the articulation of the sternum and xiphoid, gives a figure resembling the periphery of a violin, the broadest portion being posterior and the narrow rounded portion anterior, while the constriction corresponds to the lateral grooves. In rickets both bones and muscles are weakened and the thoracic wall does not offer resistance; it therefore falls in at its weakest and least supported parts. At the junction of the ribs with the costal cartilages there is a falling-in of the ribs; the cartilages and sternum are thus thrust forwards, forming a rounded protuberance. Broad and shallow grooves thus formed occur on either side of the sternum and run from the second or third rib to the hypochondrium. The floor of the depression is constituted by the ribs outside their junction with the cartilages; and along the inner side of the groove the swollen ends of the ribs can be seen - the so-called beading which constitutes the "rickety rosary"; the latter being perhaps the most constant feature of the disease. These bead-like swellings are, at the chondral junction, most marked on the fifth, sixth

and seventh ribs. The beading is sometimes obscured by the fact that the ribs are displaced backwards at their junction with the cartilage. No trace of this rosary is to be seen when the rachitic patient is grown-up, it seldom also being observed in a child of five years of age. The ribs are softer than the cartilages in rickets; in the healthy, - and even more so in the tubercular, it may be noted, - the cartilages are softer than the ribs. Owing to the softness of the bones and cartilages, the chest loses its firmness and is easily depressed by atmospheric pressure or sucked inwards during the act of inspiration - a condition still further assisted by the flexibility of the thoracic wall; if there is obstruction, as from bronchitis, to the air entering the lungs, this indrawing is aggravated through the lungs being less supported and less distended. Some of the older writers dwell on the fact that the deformity of the chest is produced by atmospheric pressure, aided by the position of the upper margin of the liver, stomach and spleen, but not by the diaphragm's contractions. The chest wall does not recede so much as that of the opposite where it covers the heart: so that the left side appears larger than the right, an impression of precordial fulness being thereby produced. Another well-marked sign of rickets is what is known as Harrison's groove; when the chest wall is sucked in and there is obstruction to inspiration, the costal margins will be drawn inwards and, if interference is more pronounced, a groove appears on either side of the chest, running backwards and downwards round the anterior portion of the chest. It vanishes when the obstruction is removed; but permanent deformity may remain, if the ribs are soft and weakened by the rachitic disease. This groove is sometimes called the "rachitic girdle" and is about two inches in width; it lies at the level of the attachment of the diaphragm, it encircles each side of the chest from the lower end of the sternum backwards to the posterior edge of the axilla and becomes more marked if any disease of the lungs causes increased inward suction of the thorax. This lateral furrow serves to mark the upper limit of the abdominal viscera, which press out the chest wall below the groove, giving a dome-shaped appearance to the lower part of the chest. The lower costal arches are moulded upon the abdominal contents and there is thickening and eversion of the lower edges of the ribs and cartilages - the same having been compared to the brim of a hat. Jenner insists that the groove is not due to traction of the midriff, but to atmospheric pressure aided by the position of the upper edge of the liver, stomach and spleen; enfeeblement of inspiration in the presence of incomplete expansion, softening of the ribs and atony of the abdominal muscles contribute to its occurrence. One may sometimes observe another groove, of vertical position and outside the nipple line, on each side of the chest; it may be alongside or behind the rickety rosary where the ribs and cartilages unite and is present on both sides. It runs outwards and obliquely downwards on the front face of the chest; its position is determined, according to Jenner, not by the loss of power of the respiratory muscles attached to the outside of the ribs, but by the softness and want of resistance of the ribs themselves. Still another groove is sometimes observed immediately in front of the rickety rosary; it is a very bright one, as a rule. From the third to the ninth rib there may be flattening of the chest. The upper ribs are stronger and better supported and covered by thick muscles; consequently, there is no appreciable flattening in that position. The chest in the upper part appears contracted because the clavicles are shortened and the shoulders approximate; but in reality its capacity is not diminished. The lateral pressure upon the softened ribs may cause the sternum to bulge forwards, there perhaps being a vertical groove running down its centre. The projecting sternum of rickets has a rounded outline and gives an appearance called "pigeon-breast"; it must, however, be differentiated from the sharp pigeon-breast caused by pertussis, bronchitis or other severe respiratory disease in childhood. The condition in question is much more angular and has a sharp projection resembling the breast of a bird; it may be associated with the rickety pigeon-breast, when the deformity will be much increased, and modifies its appearance. In rickets we sometimes also see what is termed the "funnel-chest", in which there is a deep hollow, from one to two inches in depth, over the middle of the ensiform cartilage. We have

already seen that the rickety rosary is usually observed some months before the presence of rickets is suggested by the occurrence of the thoracic anomalies. With the growth of the child the various chest deformities described above tend to vanish, the thorax becoming normal in appearance. The rickety chest can often be detected up to five or six years of age, but with age the viscera sink still further into the abdomen and the chest walls expand more freely as the lungs receive freer play. The ribs from the second to the eighth are usually most affected; they may fail to attain a proper length and the angle becomes less obtuse than normal. Instead of projecting, the breast-bone may sink in bodily.

Clavicles

The collar-bones may show increase of their ordinary curvations, or they may actually undergo twisting or distortion. There is not infrequently enlargement of the articular ends, most often the sternal extremity. In severe cases of rickets there may be observed, at the junction of the inner and middle third, an acute forward and upward kink or bend, causing a peculiar alteration in the shape of the bone. There may also be evidences of greenstick fracture appreciable to the inner end or the middle third. Chance presents an analysis of 600 cases; in 120 there was curvature of the clavicle, in 60 curvature of the radius, with the like anomaly of the humerus in 36. The occurrence of the deformity is appreciated when we recollect that the collar-bone is an important structure whose function is to hold up the arm away from the body, as well as to act as a fulcrum on which it performs its movements. Later, when the child begins to crawl, the weight of the body is thrown through the clavicles on to the outspread hands. These bones are therefore obliged to withstand considerable pressure and if, in the softened rachitic condition, they are unable to do so, they yield under the continuous strain and become bent and twisted more often than other bones with less exacting strain to bear.

Constipation and Diarrhoea

The "pot-belly" weakness or atony of the external abdominal muscles has its counterpart in the general enfeeblement of the muscular coats of the intestines excited by rickets, leading to defective peristaltic action of the fibres, culminating in constipation, flatulence, fermentation of the retained food in the stomach and intestines and distension of the belly. Constipation is an early symptom of rickets and may be present even in breast-fed infants. It is partly due, as stated, to the weakness of the muscular layer of the intestines, but is aggravated by laxity and enfeeblement of the abdominal walls. It has been known to begin as early as the second or third months of life; it must be distinguished from congenital constipation, which is invariably present from birth. Though most of the cases are at times markedly constipated, it may happen that severe diarrhoea and straining may dominate the clinical picture of the disease. The stools are not infrequently of a greenish colour and mixed with a whitish and curdy material, consisting mainly of undigested milk and possessed of a very offensive odour. The food apparently passes through the alimentary canal mostly unchanged, being propelled too quickly for digestion, owing to intestinal peristalsis being too active. In every case in which the bowels participate in the general catarrh of the mucous membranes, diarrhoea will become a marked feature of the disease, and some of the cases show a tendency to piles. The respiratory troubles so often observed in rickets may be subordinate to the gastrointestinal catarrh, whereas sometimes a child suffers alternately from these conditions. In many of my cases I have noticed that, long before the appearance of the classical rachitic signs the child suffers from considerable disturbance of the gastrointestinal canal. Constipation, diarrhoea, gastric catarrh, colic and flatulence I have often seen to follow faulty feeding, with culmination in malnutrition or malassimilation and rachitic disease at last. On the other hand, I have noticed these vague symptoms of digestive disorders as the actual prodromata of rickets, the first one to arouse my suspicion being obstinate constipation, which Jacobi regards, even in breast-fed infants as pathognomonic of the disease; this resultant of abdominal muscular atony, which is sometimes associated with acholia and dry and pale stools, may later change into acute gastrointestinal catarrh, with or without diarrhoea and vomiting.

Haemorrhoids

I have seen several rickety children in whom piles gave rise to much suffering and inconvenience, as the result of congestion of the lowermost abdominal parts.

Head

In rickets, as we shall presently observe, the normal ratio between the head, the chest and the abdomen undergoes alteration, but only for the period during which the disease is in evidence; for later these parts attain almost equal measurements, the chest gradually gaining on the head and belly, until at length it has the greatest circumference. But it is very apparent, on inspection of a rickety child, that the head and belly are larger than normal and greater around than the chest, which is narrow and contracted and marked by the sulci already described. The head of the rachitic infant - markedly large in contrast to the small face - is of an enlargement that is often more apparent than real; for it may not measure more than that of a healthy child, though, owing to the narrow and contracted chest, it seems on comparison out of proportion to the rest of the body. The enlargement appears to be due mainly to the thickening of the cranial bones, an increase of one or two inches being sometimes observed in circumference of the skull. Trousseau says that the large head indicates precocity; this theory, however, no one nowadays seems to subscribe. Jenner thought that it was often hypertrophic, whereas Gee maintained that it was small. Others have contended that it may be enlarged, as are the ends of the long bones, - a sort of fibrosis, - or that it may suffer like the muscles and be atrophied, while the ventricles contain much liquid. It is also important to note that the shape of the head is latered in this disease. The skull is long on the antero-posterior diameter, that is to say, it is dolichocephalic, and is square and box-like or quadrate. The forehead is broad and prominent, from the bulging forwards of the thickened frontal bosses, - the so-called "Olympian forehead" being formed, - with the like condition of the parietals and perhaps development of cellular cavities in the bone. As stated, in contrast to the square head, the face appears to be small and narrow; but there is also some arrest of bony development. The top of the head is flattened, owing to separation of the parietal from the temporal bone and the retroversion of the occipital. The temples and occiput are also flattened from pressure, Parrot's bosses being well marked in these situations. Carpentier (Brit. Jour. Chil. Dis., Vol. i, 216) regards these nodes as definite signs of syphilis and states that the spleen will be found enlarged in such cases; according to him it is essentially a syphilitic manifestation and present in 50 per cent. of such cases, having a marked tendency to occur in rickety children; various observers, however, have disproved this theory. Still, for instance has made a careful study of the symptoms and histories of numerous cases, but could not satisfy himself that these bosses, whether close to the fontanelle or farther away, really indicate the presence of the syphilitic dyscrasia. Though undoubtedly they occur in syphilitic infants and often are the results of this disease, yet he was fully assured that bosses occur on the infant's skull which, though impossible to distinguish clinically from syphilitic bosses, result only from rachitic disease. He uses the word "clinically, because syphilitic and rachitic bosses differ in their morbid anatomy; and it may be possible that the clinical manifestations represent two varieties of bony outgrowth, one syphilitic the other rachitic, which differ anatomically in their method of formation. The occipital bone is flattened by pressure upon the pillow and the brain is pressed forwards against the frontal bone; but the centres of ossification have usually been active for some time prior to the onset of rickets; the frontal and parietal eminences offer resistance and the direction of least resistance is backwards: so that the brain will follow this and push out the skull at the sides. The disease supervenes perhaps at one year and then the interfrontal suture will be ossified and thus the brain again will go backwards and produce a long posterior segment. It will also widen out the parietal eminences and occasion a small and square forehead. If rickets commences early, the posterior fontanelle may remain open, as well as the anterior and, if the brain is large or contains excess of fluid, the sutures may be widely open and the bones forming their walls greatly thickened: so

that furrowing is produced along the line of the sagittal and frontal sutures, as well as another groove at right angles along the coronal suture. These furrows, together with the protuberant bosses, give the skull the peculiar "hot-cross-bun" appearance, "the natiform skull" of some writers, which is only seen in severe cases of the disease and regarded also by some as a syphilitic sign. In rickets the closure of the anterior fontanelle is delayed, and this is of importance in diagnosis. It ought to close at about eighteen months, but in rickets it may be found open to the age of two and a half years, and I have knowledge of instances in which that period was prolonged to four years. A systolic murmur is frequently heard on auscultating over it; it originates in the cerebral vessels and is conducted better by the unossified membrane than by the bones of the head; it was formerly regarded as an important symptom, until Osler (Prac. Med., p. 43) showed that it is also present in healthy children, though seldom heard after the age of five years. When rickets occurs very early, there may be abnormal patency of the posterior fontanelle as well. The above-mentioned grooves must not be mistaken for open sutures; they are the result of pressure from enlarged veins coursing in the scalp and are sometimes very noticeable. Elsässer was the first to describe the occurrence of small patches of thinned bone, - a quarter to an inch in diameter, - upon the occipital and sometimes the parietal bones, resulting from wasting of the inner table of the bone. They are most often seen from the second to the sixth month of the infant's age and take from four to five weeks for full development; they are seldom seen after the end of the first half-year of life, though in rare instances they have been noted up to the eleventh month in cases in which rickets was of late onset. On pressing on these patches with the tips of the fingers, a bulging or crackling sensation is felt, just as if the part were made of thick parchment. The frequency of the condition may be estimated at about 30 per cent., and is apt to be associated with laryngismus stridulus and tetany. Several authors have argued for an association of craniotabes with syphilis or a manifestation thereof. Lees and Barlow found that congenital syphilis was present in 47 per cent. of their cases of craniotabes in rickets, they therefore contending that syphilis is by far the largest factor in the causation of the anomaly in question. Carpentier (Rep. Soc. St. Dis. Child., Vol. iii) finds that the condition is usually present in the parietal bones, - 60 per cent., and occipitals, - 30 per cent., - and least often in the frontal bones and then only if the other cranial bones are attacked. He thinks that it is usually a syphilitic manifestation; he has studied 238 instances and noted it most often during the second, third and fourth months of life, very rarely about the ninth month - the conclusion being that during the prerachitic period craniotabes is most evident, it being found with difficulty upon the appearance of rickets. Cautley's observations are much to the same effect, he finding most cases between the second and fourth months, but most often in infants fed on condensed milk or other maternal milk substitutes. He would have it that the boiling of such infant foods renders the salts less soluble and refers its occurrence to deficiency of salts in the infant's artificial diet; he cannot believe that craniotabes of this type is associated with syphilis, but is due to dietetic errors which, later on, lead to rickets or other nutritional perversions. Comby also does not recognise syphilis in its production, but attaches most etiological importance to improper feeding upon the bottle or deficiency of breast milk. Still (loc. cit) also doubts whether it should always be regarded as of syphilitic origin, especially if the definition of craniotabes be taken as a small patch of thinning of the skull bones, at some distance from the sutures, and not a diffuse thinning of the edges of the bone adjoining them; he thinks that it is much more a manifestation of rickets than syphilis and, if further proof were needed, he refers the reader to the frequent association of the condition with laryngismus stridulus, which is a symptom practically always of rickets and one which rapidly disappears under suitable treatment for that disease. Rasum (Rous. Vrach, iii, 5) has conducted extensive histological examinations and concludes that there are two kinds of craniotabes in children - the rachitic and the non-rachitic. The former, he tells us, is

atrophy or undeveloped structure of bone, the rachitic form to lack of lime salts plus pressure of the brain on the delicate bone. The rachitic changes are usually seen after the third month, while atrophic changes may occur even in intrauterine life. During life the softening due to rickets can only be detected if the rosary on the chest is also in existence. The histological appearances in these two forms are not alike. E. Spietschka (Jahr. f. Kinder., lix, H. 3) submits an extensive study of newborn infants and points out that they often exhibited softening of the occiput, defects in ossification, yielding and gaping sutures and a disproportion between the size of the head and chest, which may develop into true rickets in the absence of suitable treatment; the frequent and early occurrence of this softening convinces him that rickets may be and often is a congenital condition, which from birth he successfully treats with phosphorus and cod-liver oil. From the above observations it would seem, then, that craniotabes can be considered a symptom of rickets, even apart from syphilis, though it is not always possible to regard it as a sign of this disease alone; further, its presence can be interpreted as indicative of the existence of some malnutritional disorder, possibly syphilitic or rachitic and associated with defective absorption of lime salts, there being more certainly about a diagnosis of rickets if the beading be present at the same time. In the craniotabetic condition there is marked patency of the anterior fontanelle, which instead of closing at the eighteenth month or up to two years, in the rickety child remains open up to two or three years, and even as long as five years a slight opening perhaps being discoverable. The veins of the head are unusually prominent and often, even before other symptoms have appeared the bald patch on the occiput, due to friction upon the pillow, is distinctly suggestive of the rachitic state. The face of the rachitic child appears to be much smaller than normal in contrast to the big head, and it may even have a wizened appearance. The palatine vault is deformed, the nasal fossae may be narrowed and the tonsils pushed towards the middle line, with production of various catarrhs in this locality and perhaps adenoid vegetations. The alveolar border of the upper jaw is somewhat beak-like or more spherical than normal, with undue prominence of the cheek bones. The lower jaw is inclined to be polygonal; it is short, softened in front and there everted, with thickening of its rami. The incisors may be in a straight line, the bone exhibiting backward bending at the situation of the canine teeth, this condition, according to Fleischmann being due to imperfect growth of the middle portion of the bone. In certain cases one may observe that there is a want of symmetry between the two halves of the bone, so that one side is higher than the other. Eruption of the permanent teeth may occur in irregular order or be delayed by the rotation of the lower jaw horizontally, which leads to a polygonal shape.

Heart

The complications here arising are largely of mechanical nature, produced by the contraction and distortion of the thoracic walls, as well as by the pressure from below of the abdominal organs. In children the normal position of the apex beat of the heart is in the fourth interspace; but in rickets, owing to the above factors, its position undergoes a certain amount of change. The apex of the heart is displaced a little outwards towards the left; here, at each beat, the heart comes in contact with the nodule on the rib, there being in consequence produced on the thickened pericardium a whitish friction patch, which is on the left ventricle of the organ a little above the apex. In the heart itself there are no definite microscopical changes, though the organ, of course, shares in the general malnutrition of the system and leads to enfeeblement of the circulation. Palpitation, tachycardia, or slow and irregular pulsations may be produced by the hampering of the cardiac action from abdominal distension. The muscular substance of the heart is apt to be badly nourished and its muscle correspondingly weak. When there have been bronchial and other catarrhal thoracic disorders, the right ventricle has more work to do and, if, as in bronchopneumonia, there is considerable pulmonary obstruction, the right heart may be unable to cope with its task and sudden death occur. The right ventricle first dilates and then hypertrophies, with the result that the venous system becomes engorged. There may result also engorgement of the right auricle, -

which itself dilates and hypertrophies, -and of the inferior and superior vena cava; the liver becomes congested and, later on, there will be found development of oedema of the feet, legs and trunk, together with albuminuria, from congestion of the renal organs. Tricuspid incompetency is a rare finding.

Intercurrent Affections

In view of the malnutrition and constitutional enfeeblement obtaining in the case of the rickety child, it is not surprising to find that it readily becomes a prey to diseases that are going about and that such ones as bronchopneumonia and diarrhoea are particularly apt to cause death. There is also a great liability to the contraction of infectious ailments, such liability being more considerable than in the case of healthy children, the vital powers of resistance being so feeble. Statisticians long ago proved that the mortality of rickety infants from such affections as measles, diphtheria, scarlet fever, enteric fever and influenza is enormous; figures are, however, not easy to obtain, as rickets is comparatively seldom mentioned on death certificates. I always regard the occurrence of pertussis in a rickety child as of very unfavourable prognosis, as the patient is unable to cope with the sudden spasmodic attacks of that disease, hampered as it is by the contracted chest and muscular weakness, as well as handicapped by the tendency to collapse of the lungs, syncopal fatality being apt to occur. The remarkable predisposition of rachitic children towards infectious diseases has been strongly urged by Hansemann (Berl. klin. Woch., Feb. 26, 1906), who states that he has seldom had a case die from diphtheria, scarlatina or measles, unless there was at the same time rickets present, the same being true as regards whooping-cough. He looks upon rickets not as a bone disease, but as a perversion of nutrition and adds that, as renal affections are associated with gout, in a similar manner pulmonary and intestinal catarrh manifest an association with rickets. This theory has found a few adherents, but does not seem to have enjoyed general acceptance.

Limbs

The various anomalies observed in connection with the extremities make their appearance when the infant tries to walk; but, as this act is not infrequently delayed by rickets, attention may not be drawn to the curving or distortion of the bones until the age of two years or even later. The thoracic deformity may be lessening when the legs begin to bend, as the disease is now on the decline and muscular power has increased; nevertheless, the bones of the legs are soft and give way under the weight of the trunk. So long as the child is at rest, lying down, little curvature may be detected; but when it adopts the erect posture and the body weight is pressing on the lower limbs, the deformities and twistings of the extremities begin to make their appearance. For a similar reason the epiphyseal thickening at the ends of the long bones is usually more marked at the wrists in the early stages of rickets and at a later date in the ankles, because the child first attempts to crawl on all fours and throws the bulk of its weight forwards to the greatest extent on its hands, fractures at the wrists being common at this time. When the child stands up and attempts to walk, the thickening of the epiphyses at the ankles becomes more evident, of course. But, in addition to the body weight, muscular traction acts upon the soft and pliable bone and produces exaggerations of normal curvatures and distortions of the extremities. The deformities may last for life, but usually under proper treatment they can be corrected at an early age. In other cases the rachitic dyscrasia affects unfavourably the whole life of the subject and renders him unfit for certain kinds of work, or a condition of bad health may be produced which may or may not pave the way for various serious affections. Pain in the limbs has been regarded as an important sign of rickets by some, whereas others have assigned it to the presence of scurvy at the same time as that disease. Some go the length of affirming that all rachitic children suffer pain, the same perhaps being so severe that the patient lies in a flaccid condition, the slightest touch occasioning intense suffering; the pain has been also held to be confined to the bones and not to vary in proportion to the severity of the rachitic disease. We have already seen that one of the commonest signs of rickets is enlargement of the epiphyses, especially at the lower end of the radius, the same being present, it would seem, in some 60 per cent. of

the cases. It is not macroscopically apparent before the third month; by the tenth or twelfth month it is usually at its maximum, while it may often be detected much later, perhaps at puberty also; further, it is not seldom observed at the lower end of the thigh and shin-bones. The shafts of the long bones become bent, the actual degree of curvature being in relation to the age of the patient, that is to say, the time it commences to walk about. The characteristic attitude of a rickety child is a squatting one, the lower limbs being crossed like a working tailor, the body inclined forwards and part of the weight of the head and trunk supported by the palms of the hands placed upon the floor; thus there occurs a bending outwards of the bones of the arms. The gait of the child is usually waddling or duck-like. The presence of pseudohypertrophic paralysis may be suggested, in the case of fat and heavy patients with big calves and lordosis. But in rickets there is an abolition of the knee-jerks and there is no pathognomonic method of getting up from the floor as in the case of the former disease. It has been said that all rachitic bones are shorter than normal, especially those of the lower extremities and pelvis. The line of pressure will determine largely the nature of the deformities produced. In the case of the legs there is an outward and forward curve in the case of the femur, which is exaggerated if the patient has commenced to walk before the onset of the disease. The tibia shows a similar deformity, which may be unequal in the two limbs, or there may be knock-knee on one side and bowleg on the other. One sometimes observes that the shin bone is bent backwards at the junction of the upper epiphysis and the shaft, while a sabre-blade tibia is sometimes encountered. Bowleg is much more common in young children than knock-knee, owing to the natural roll of the gait at this age; the lower third of the tibia may sometimes show an actual kink. The inner condyle of the femur may exhibit a bony outgrowth or tubercle. Rachitic children not infrequently grow up into dwarfs, owing to the permanent shortening of the bones; Guérin carefully studied the question of dwarfism in rickets and stated that, owing to the constancy of the diminution in length of the bones, given the dimensions of a rachitic bone, he could tell the size of the others. The frequency of fracture of rachitic bones is well recognised; Guersant affirms that no less than a third of the fractures in his pediatric practice were due to rickets; sometimes more than one bone breaks, a large callus being usually formed on the concavity of the broken bone. As regards detailed particulars of the anomalies of the bones of the extremities observed in this disease, we may note the following: The humerus is apt to bend outwards, its greatest convexity corresponding to the insertion of the deltoid muscle; this is due to the weight of the forearm and hand when raised. It may also have a concavity forwards and inwards, or there may be a concavity looking backwards and outwards. The radius and ulna sometimes curve outwards, the convexity looking towards the back of the hand and the concavity towards the palm; these bones are also apt to be twisted. If the child, through inability to walk, has crawled very much on all fours, the curvatures will be most marked in the lower third of the bones, owing to the direction of the pressure on the pronated forearm and expanded hand. We have already seen that the epiphyses at the elbow and the lower epiphyses of the radius are characteristically enlarged and constitute very early signs of the disease; this thickening is particularly noticeable at the wrists and forms a bulging or bracelet at the radiocarpal articulation; further, greenstick fractures are of common occurrence in the neighbourhood of this joint. As regards the fingers, note must be made of the anomaly known as "Koplik's hand," which may be mistaken for syphilitic disease by the casual observer in the absence of definite signs of rickets. He describes it (Arch. Ped., 1904, p. 770) as consisting of a bowing of the hand with thickening of the phalanges, which latter are longer and more tapering than usual. A skiagram shows that the shaft of the finger is thickened and that its peculiar shape is due to changes in the bone, not to any alteration of the softer tissues, the increased length and tapering of the phalanges being the outcome of laxity of the ligaments of the joints in this locality. The distance between the extremities of the fingers which make up the joint is greater in the rachitic than in the normal hand and may be the cause of the inward curving

of the fingers at the articulations. Most of the infants studied by this author, in whom the rachitic hand could be demonstrated, suffered from marked rachitis accompanied by such acute pain in the bones that it almost suggested a syphilitic condition; but the clinical evidence was opposed to that idea and showed that the real nature of the disease was rickets. Another peculiar anomaly of this part is what is termed "Neurath's rachitic fingers" or "spindle hand." That author claims (Wien. klin. Woch., 1903) that the presence of rickets can be diagnosed by the mere detection of the presence of this anomaly of the fingers, which consists of a peculiar change in this shape from apparent sinking of the articulations, giving a spindle appearance to the proximal, middle and terminal phalanges. It is said to be best appreciated during the first year and, to detect it, the hand should be examined in profile against the light, when its outline will be seen to resemble a string of pearls on the back of the fingers stretching the overlying skin; in short, there is hypertrophy of the second phalanx, the other two being normal and in marked contrast thereto, the stretched skin being only with difficulty thrown into its normal wrinkles. The same author mentions a rarer condition, peculiar to rickets, in which the proximal and middle phalanges are conical in shape, the terminal one being enlarged like a drumstick at the level of the nail. The use of the X-rays will show that the thickening in question is due to an infiltration of the periosteum, which occurs at the middle of the shaft of the bone, not at the ends of the articulations, each finger being really of a spindle form. It would seem, however, that such digital anomalies occur only in the most severe cases of rickets and are very rarely observed in general practice. Tuberculous dactylitis must be eliminated from the diagnosis in the ordinary way; and it is particularly necessary that there be no confusion with the similar deformity seen in congenital syphilis, in which disease the end of the finger is like a truncated cone, whereas in rickets the whole finger is affected by a fusiform swelling and the therapeutic test is conclusive. Thickening of the toes is of very rare occurrence, in this case also its true nature being determined by the presence of other rachitic signs, the age of the patient being about the first year and the severity of the rachitic disease when it occurs in older children; if hereditary syphilis be the cause, it affects the unguis phalanx, giving it the appearance of a truncated cone, whereas in rickets the whole member is affected by a fusiform deformity, which vanishes under antirachitic treatment. Deformities of the thigh bone and others of the lower extremity depend on whether or not the infant has commenced to walk or make locomotory attempts. If the art of progression has been initiated, the natural curve of the femur is increased forwards and outwards. The head of the bone may be at an angle with the shaft; if the child cannot walk, the shaft will be directed forwards and, for the same reason that, as the patient sits on its mother's knee, the weight of the leg drags on the lower part of the thigh. The femoral deformity may be on one side, but usually it is on both. This condition of bending of the neck of the femur without disease of the hip joint - coxa vara as it is called - is by no means uncommon in rickets and consists of a downward bending of the part in question, with elevation of the great trochanter, such severe expression of rickets being due to the weight of the body acting on the softened bone. There is an undue mobility of the articulation in certain directions, eversion, abduction and adduction are freer than normal, but it is almost impossible to produce inversion. It is more often than not of congenital origin and, when such, may be distinguished from the rachitic variety by the history of the onset without previous disease, the absence of other rachitic signs, the abnormally short neck of the femur and the vertical direction of the epiphyseal line; whereas in rickets the latter runs diagonally downwards and inwards at a sharpish angle with the lower border of the longer neck of the bone; the use of the X-rays assists the diagnosis materially. The opposite condition to the above is what is known as coxa valga; it is sometimes a rachitic expression and in it also there is a change in the angle formed by the neck and shaft of the thigh bone, the average size of this angle being 125 degrees, in vara it being lessened, in valga increased. The symptoms of coxa valga are increased abduction and outward rotation of the lower limb; the

body tilts forwards and there may be slight lordosis of the vertebral column; the patient walks with a rocking movement, swinging the body from side to side; the hips are slightly bent and there is marked extension of the knees. Pressure on the outside of the ankle, owing to inability of the child to walk leading to a cross-legged attitude, results in the concavity of the curvature of the legs being directed forwards and inwards and the knees far apart. If the child can walk, the concavity of the tibia and fibula is from without backwards, so that the knees are approximated and an abrupt curve, with the convexity directed forwards and outwards, exists in the lower third of the leg. Sometimes the legs are variously bent, the one inwards and the other outwards perhaps. An infant not yet commenced to walk may have the lower extremities straight, though small; while the upper ones may show deformity. The old theory that rachitic deformities always proceed from below upwards is not now entertained. Bowlegs is perhaps the commonest deformity seen in the lower extremities. If extreme, there may be a greenstick fracture of the part and arrest of development obtains here as well as elsewhere. There may occur an outgrowth of bone on the inner side of the head of the tibia. The principal changes, then, to be observed in the long bones are thickened epiphyses, exaggeration of the normal curves, development of other curves from pressure, the appearance of tubercles or irregular outgrowths of bone and, in the later stages of the disease, deficiency in length and stunting of growth. Jenner maintains that all the bones of the adult, previously affected by rickets, are diminished in length; but the lower limbs, including the pelvis, are disproportionately diminished in size and the face small in comparison to the cranium. We have also seen that the character of the deformity produced in the bones of the limbs is largely governed by the line of pressure produced by the position or attitude which is usually assumed by the patient. The femur bends outwards and forwards in one large curve, the angle of the neck may be reduced and coxa vara formed. The curvatures may be symmetrical or unequal and frequently the two thighs together form an imperfect oval. If knock-knee is present, there may be an overgrowth at the inner segment of the lower epiphyseal line. The tibia and fibula take on a forward and outward curvature, with usually a sharp kink in the lower third. The former bone not infrequently presents, as we have seen, a peculiar formation called the "sabre-tibia" and subperiosteal fractures may still further deform it. There may be an overgrowth at the inner segment of the upper tibial epiphysis, while both that bone and the fibula may be curved sharply backwards. Such curvatures are due to the child's position in bed with the knees flexed and the legs crossed. The development of knock-knee or bandy-leg is governed by the age of the patient and its capability of walking or making progressive attempts. The latter deformity is much more common in young children; for at an early age the gait is more of a roll or waddle than a straightforward walking. It often happens that the child throws a sudden strain on the lower third of the leg in its endeavours to balance or prevent falling forwards and so produces anterior curvature of the bone with the above-mentioned marked kink in this locality; the lateral efforts made to preserve its centre of gravity cause the outward bend of the bone and consequent bowing of the legs. In older children, when able to stand or walk about, genu valgum is more common, because in the erect posture the weight of the body is transmitted mainly through the outer condyle of the femur and in rickets there is an arrest of growth on the outer side of the epiphyses of the femur and tibia, which, however, is unchecked on the inner segment. It would seem that these deformities of the legs are not infrequently of a compound nature and that a knock-knee may be due to a deformity of the femur or the tibia alone, or to a deformity of both these bones. In some cases there may be an outward bending of the femur and an inward bending of the tibia. If one bone only is affected, the limb may show deformity; but with curvature of more than one bone the limb, as a whole, may seem to be straight.

Liver

It sometimes happens in rickets that the liver is displaced downwards or enlarged, or both displaced and enlarged. The displacement in question is due to thoracic deformity; the enlargement results from chronic hyperaemic fibrosis resulting

from the pulmonary obstruction and cardiac enfeeblement, or perhaps from congenital syphilis or a circulating toxin, in which case there is usually also a considerable enlargement of the spleen and possibly of the lymphatic glands generally. The increase in size of the organ may be merely the result of a passive congestion, with perhaps culmination in actual fibrosis or an ultimate fatty change. Concomitant hyperplastic enlargement may be seen sometimes in the mesenteric glands, while it may happen that jaundice occurs.

Mesenteric Glands

As stated, these sometimes participate in the general adenopathy obtaining, or they may undergo enlargement without similar anomalies elsewhere.

Muscles and Ligaments

The muscular and ligamentous structures are profoundly affected in rickets, the child being very weak and with muscles so flabby perhaps that it cannot hold up its head and, from spinal muscular and ligamentous debilitation presenting arching of the back. The ligamentous structures are lax, elongated and wanting in normal tonicity, particularly those about the larger articulations. Thus there occurs a marked ~~assistance~~ in the production of knock-knee, a peculiar hyperextension at that joint, - the so-called genu recurvatum, weak ankles, flat-foot, flail-like joints and spinal and hip deformities. The ligaments participating in the general malnutritional asthenia are so weak that they yield to both traction and pressure, especially in the case of the spine; and the same is also well instanced in the muscles of the arms and legs, for, if the child has been able to walk, it loses this art on the occurrence of rickets and the muscles waste from malnutrition and disuse and find aggravation of their abnormalities in the softening of the bones obtaining. All the muscles exhibit this loss of tone; they are also poorly developed and small and flabby; under the microscope their tissue may appear pale, with their striae colourless, blurred and difficult of definition. The child is unable to hold up its head at the proper age, - which should be at about the thirteenth week if the back is supported, - it is late in trying to sit up, - fifth to the sixth month, - and can seldom walk at the normal period, - the twelfth to the eighteenth month. So it may happen that the patient cannot effect locomotion until two and a half or three years of age and the presence of rickets may be suggested by this backwardness, together with the presence of weak ankles or deformities of the legs. The weight of the body, acting on the weak ligaments of the ankles, often causes the feet to splay downwards and outwards - this abnormality being often encountered in fat and heavy children, while knock-knee and flat-foot are also met with, as a rule. Indeed, the presence of flat-foot in children over two years of age should prompt a careful examination for knock-knee, the co-existence of these two conditions being dependent upon the presence of rickets at the time or anteriorly. In these cases the mother is very liable to fear that her child has become paralytic and brings it for treatment of that disease, stating that for some time her offspring has been unable to walk or run about, having gradually ceased to do so, becoming lethargic, inclined to sit still and apt to cry when interfered with. Great care must be taken not to confuse such a condition with infantile or diphtheritic paralysis. But the flabby state of the muscles, the normal electrical reactions, the ability to move the limbs and to walk, though disinclination for the attempt, should constitute sufficient evidence of the presence of rickets, which may or may not be aggravated by the co-existence of some scorbutic taint. There may also be a history of prolonged lactation by the mother, of improper artificial feeding of the child, or unfavourable hygienic rearing. Such cases have been classified as rachitic pseudoparalysis and regarded as excited by the weakness of the muscles and ligaments, plus the softening of the bones, the weight of the body proving too much for the flabby limbs; which fail to support it, so that the patient, though still possessed of the power of movement, is unable or disinclined for exertion. The general muscular asthenia existing is markedly shared in by the abdominal muscles, which fact is apparent externally in the muscles of the belly wall and internally in those of the intestines. In common with the other muscles of the body, the recti abdominis are weak, flabby and badly fitted

for their normal function and, at the same time, they are still further adversely prejudiced by the pressure of the protuberant stomach and bowels; from repeated attacks of indigestion the intestines become distended, which still further favours local atony of a progressive sort. In the case of a healthy infant the respiration is mostly abdominal, the abdomen being protruded with each inspiration. In the case of a child with well-marked atony of the abdominal muscles, very little protrusion of the abdomen takes place. The lower ribs in normal inspiration are also expanded; but in rickets, instead of expansion, a retraction of the thoracic walls is seen when the diaphragm contracts. This is because the weakened ribs and cartilages yield more readily to atmospheric pressure in the imperfectly expanded lung. Owing to this want of a fixed fulcrum, the diaphragm, already weakened, is hampered in its action on the enlarged abdomen. Expiration is also interfered with, on account of the weakened condition of the belly and is simply a passive recoil; even the latter is weak through imperfectly performed inspiration. The recti abdominis muscles not infrequently become unduly stretched, with the result that umbilical hernia occurs, or there is middle-line diastasis of the muscles here. Such a condition is apt to escape attention in infants, but may be readily detected if the child is raised by the head and shoulders in such a way that the recti are put on the stretch. In older children there may even be a diastasis of a half to one inch in width. Atony of the muscles being an early rachitic sign, this separation of the recti may be observed as early as the fifth month of life, long before the presence of osseous anomalies suggests the existence of the parent disease. The condition does not seem to be hereditary, though rachitis has been detected at birth. Though one of the earliest symptoms of the disease, it is slow to take its departure and may, indeed, persist for years, as the muscles are very slow in recovering their normal tonicity; in adult visceral ptosis this fact should be borne in mind. The diastasis is primarily produced by the arrest of the development and nutrition of the muscles, secondarily by the pressure of the stomach and bowels, inflated with gas, acting on the weak and flabby muscles of the belly wall. It may occur in healthy children, in whom it disappears about puberty. In the normal child closure takes place from below upwards and, as a rule, the diastasis is only seen above the umbilicus; but in the rickety infant the separation involves the whole extent of the belly wall; though said to be present in many cases of rickets, it is not a pathognomonic sign, as it so often occurs in the absence of that disease. Owing to the abdominal distension, there is found a dome-like appearance of the lower part of the chest; and the loss of proportion between the enlarged belly and the narrowed base of the chest causes the midriff to work at a disadvantage; as it lacks power to project the belly in the normal way, the mechanical effect of the shortening of its fibres is a direct pull on its attachments, which may partially account for the inspiratory recession in the region of the lower ribs.

Nervous System

The rachitic child has a profoundly affected nervous system, as may be expected from the fact that it is naturally somewhat unstable during this period of its development. It is still further enfeebled by the existing malnutrition and it is apt to be prejudiced unfavourably by toxins circulating in the blood stream and by reflex irritations from various parts of the body, particularly the gastrointestinal tract. So it comes to pass that one not infrequently observes the development of nervous anomalies during the course of the disease, the milder forms being represented by facial irritability, head-nodding, nocturnal restlessness and sweating, the graver conditions being such alarming conditions as tetany, laryngismus and convulsions. It has been stated that the nervous system is deprived of the necessary healthy fatty matter, with the result that, in rickets, the neurons cannot undergo proper development. Money states that rickets is essentially a neurosis producer and the great creator of infantile affections. A peculiar rachitic anomaly is that known as "body-rocking", in which the child is in the habit of swaying its body to and fro whilst sitting up, as some adult persons do when in great pain in a melodious pendulum fashion. As it occurs in the sitting position, very young children, under

nine months of age do not, of course, present the symptom. The movement is usually from side to side, though it may perhaps be from before backwards from the hips as a fulcrum. Further, there may be a combination of movements, rhythmic in character, which are even occasionally continued during light sleep. But it should be remembered that some normal children have the habit of swaying the body from the hips when sitting up, though it is most often seen in rickety children, who are lethargic and disinclined for exertion and prefer being left alone to their monotonous movements. Such patients not infrequently adopt the so-called "pagoda position" seen in the statue of Buddha; for they sit on the floor with the legs crossed in front or under the body, with perhaps the hands folded; in such cases body-rocking is often observed. It is a well-known fact, well recognised, that convulsive disorders are very common in rickety children and some, moreover, would have it that rachitic disease is responsible for all children's ailments occurring during the first year of life. At this tender age the nervous system being in an unstable condition, some trivial cause, - such as dentition, overloading of the stomach or intestines and dietetic errors, - may suffice to excite a convulsive seizure, which very often proves fatal, particularly in the absence of prompt treatment. In short, infants suffering from rickets are more prone to such attacks than healthy ones of the same age and it would also seem that rickets is by no means infrequently the most potent cause of the fatal or non-fatal or dangerous spasmodic affections of childhood. It is also worth bearing in mind that many cases of convulsions ascribed to teething are possibly rachitic expressions; for in that dyscrasia dentition is delayed and rendered more difficult, owing to the frequent presence of gastrointestinal disturbance. In a healthy infant cutting the teeth is a normal physiological process, with little disturbance of the nervous system; whereas in the rachitic child the dental penetration of the gum may be as the match that lights a fire and occasion a convulsive explosion. According to Jacobi, a normal dentition never produces a convulsion and difficult dentition as a cause of convulsions is among the affections which are as rare as they are too readily diagnosed. Much has been written on the subject of convulsions in rickets, but despite the differences of opinion on certain points, there seems little doubt that fits occur most usually from the age of three to six months, during which time it is more fatal than otherwise, as has been my experience. But rickets usually occurs between the seventh and eighteenth months: so that, if it is a predisposing cause, it is not the only one. Nevertheless, rickets is a powerful predisposing cause of the spasms of early life, a large proportion of the rachitic infants being affected in this way. There seems a great tendency on the part of medical men to assign convulsions during the dentition period to teething, which is a mistaken idea for the reason that so many attacks occur before the teeth commence to cut the gums, when rickets is present to some extent and obviously responsible for the fits. To dentition, gastrointestinal disorders, and other things which predispose to the attack, should always be added a suspicion of rickets - in which case the seizures will be much more severe and dangerous. There is no doubt that in rickets the nervous system is in a badly nourished condition. In addition to the increased reflex excitability induced, there must be taken into consideration various changes occurring in the body leading to delayed development of the the proper functions of the inhibitory or controlling centres of the central nervous system. Then trivial causes, such as constipation and diarrhoea or dentition, as well as various rachitic simulations, may excite violent convulsions - the latter being also predisposed to by the compression of the cranial contents by the softened skull bones and the imperfectly oxygenated blood supply of the brain. Convulsions occurring in rachitic children are always, apart from possible fatality, are to be taken seriously, as they may predispose to epilepsy in adolescence or thereabouts. Cautley (Clin. Jour., xxvi), whilst allowing that rickets is a very common apparent cause of convulsions, insists that too much importance has been attached to this association, for the reason that, in his opinion, the fits cannot be explained by the osseous or craniotabetic changes which that disease induces. It is, however, the dietetic errors responsible for rickets in which he believes, especially in view of the fact that he has found that

comparatively few rachitic infants become convulsed and that rickets is practically universal. It is justifiable to regard rickets only as a predisposing cause, mainly on account of its complications, the bad feeding, the gastrointestinal disturbance, bronchitis, dentition and partly through malnutrition with consequent diminished control of the nervous system. Divine (Brit. Jour. Chil. Dis., iii, 448) has given close study to the mortality figures ascribed to convulsions and various rachitic conditions, as well as dentition, scurbutus and laryngismus stridulus, during the year 1903. He found that convulsions caused 62 per cent. of deaths from birth to 3 months, 20.9 per cent. from 3 to 6 months, 17.1 per cent. from 6 to 12 months; whereas the figures for rickets were respectively 3.7, 17.2, 79.1. During the first three months of life rickets does not enter into the calculations, during that period hereditary influences being the principal cause of the fits - to which Divine would add the use of unsuitable foods. Between three and six months rickets is in the incipient stage; but, during the last six months of the first year it is easy to detect its malign influence: so that, between the ages of six months and three years Ashby finds that the large majority of children who suffer from convulsions manifest some sort of rachitic taint. In the above-mentioned year there were 2255 deaths of children between six and twelve months of age registered as due to convulsions; but of these only 444 were held as being due to rickets alone. Divine maintains that the mortality due to rickets is understated; that due to probable rickets is increased to 2283 if teething and laryngismus stridulus are added, but even then deaths from bronchitis and diarrhoea of rachitic origination are not included; further, it is impossible, unless the true rachitic origin of the terminal fatal affection is appreciated and noted in the death certificate, to arrive at the true death-rate of rickets. Holt looks upon rickets as the most important predisposing cause of the disturbance of the nutrition of the brain; he concludes that the instability of infancy is most frequently produced by rickets and that, in the presence of convulsions without ~~discernable~~ cause, search should always be made for the existence of that affection. Taylor is of the opinion that most convulsive attacks in infancy are due to rickets, owing to the lack of co-ordination between the development of the nervous ~~system~~ and the enveloping connective material, irritability of the cells being thereby produced. He would have it that under certain circumstances the higher centres of the nervous system govern the action of the lower ones, which latter, if uncontrolled, have a certain inherent automatic action leading to discharge, when unbridled, in the form of convulsive seizures. Again, incomplete development of the higher centres deprives the lower ones of their salutary control, with resulting discharges of the kind just named. He therefore recognises two kinds of convulsions, the higher-level and the lower-level ones, which may be produced by irritation of any kind, particularly such as exists in rickets, which latter, too, may have an etiological bearing upon ~~epilepsy~~ when the child has grown up; the fits may assume various forms, such as laryngismus stridulus, tetany and general convulsions, there being, however, some difference of opinion regarding the frequency of these conditions and for the reason that the onset of the rachitic disease cannot always be determined with accuracy. Thomson also thinks that rickets is the most prolific cause of convulsive seizures in infants and remarks that proper prophylactic treatment will prevent their occurrence. He finds the condition most common from six months to two years, especially during the cold and windy months of the spring. In view of the history of the case and the special rachitic symptoms, he concludes that there should be no difficulty in diagnosis and that it is unnecessary to administer sedatives for its relief. The convulsive seizures of rachitic children generally affect the entire body and are very seldom unilateral in distribution. As a rule, they are not dangerous to life, though they tend to weaken the nervous system and may be the starting-point for true epilepsy in later life; therefore, a convulsion should not be lightly disregarded and every effort should be made to prevent any recurrence. If the presence of toxins is suspected, the digestive system should receive attention of the indicated sort and all possible done to create a sound nervous mechanism in a healthy frame. Some rachitic infants are in the habit of banging the head against

the bed or floor, such phenomenon being usually observed between the age of six months and two years, seldom after the fifth year. The most characteristic sign is a constant beating of the head by the child with its fists when the cot or floor are not attacked by that part. Rickets is discovered in a considerable proportion of these cases, though it is not certain that this disease is the real cause of such nervous ailment, it being probably also associated with dentition and other reflex irritations. Carpentier describes various cerebral lesions in connection with it, showing that sometimes there may be a deep-seated cause at work. Osler holds that it should be classified, together with head-nodding, as a coordinated tic. Still has found rickets present in some of his cases of head-banging, but ascribes it most frequently to an irritation proceeding from cutting the teeth or a middle-ear disease. In some cases of rickets the condition known as "spasmus nutans" or "head-nodding" is observed; in it there occurs a rhythmic movement of the head or nodding of the same backwards and forwards like an animal. Most often the motion of the head is in the antero-posterior direction of the body, though it may be combined with lateral rotation. It may be associated with nystagmus, which, as a rule, is more marked in one eye than the other and sometimes is limited to a single eye, the direction of the nystagmus being rotary, vertical or horizontal. It is an indication of the unstable condition of the nervous system and is apt to occur early in the course of the disease; it ceases when the patient wakes from sleep, is seen from six to twelve months and, if it be of long duration, may give rise to the suspicion of profound cerebral affection or otorrhoea. It is seldom observed after the second year and appears to be more frequent in the winter months, rarely commencing in the summer. Head-rolling is also sometimes seen in the early stages of rickets; the child rolls its head monotonously from side to side and not infrequently, in these cases it is seen that the back of the head has the hair thinned by friction on the pillow. The rolling may continue during light sleep, but always ceases when the patient sits up. It is not peculiar to rickets, but a by no means infrequent associate of the same. Still (Clin. Jour., xxix, 88) found rickets present in 15 out of 19 cases examined and in 4 cases other nervous disorders dependent upon rickets - laryngismus stridulus with facial irritability in 3 and facial irritability alone in one of them. The affection is almost entirely one of infant life, most common between six and twelve months and seldom seen after the second year. If very pronounced or persistent, the suspicion of meningitis is likely to arise and receive the usual diagnostic confirmation, while Burnet thinks that it may have some connection with otorrhoea. Still doubts if rickets should be considered an essential factor; in one of his cases head-rolling began at four weeks and in some other he observed that rickets was absent - there being therefore some other factor present, probably some form of peripheral irritation; the latter most often assumed the form of disease of the middle ear, the rolling of the head, however, appearing long before the aural affection could be suspected or diagnosed, though dental irritation could be made out. The fact that the time of onset is apt to be confined to the winter months serves to distinguish head-rolling from all forms of infantile rotatory spasm of the head, which commence at any time of the year. Owing to its invariable appearance during the cold months of the year, it was assumed that head-rolling was set up by darkness and it was sought to compare with the nystagmus of miners. From this view Still dissents and compares the seasonal incidence of spasmus nutans with that of laryngismus stridulus, which is also seen most often from November to March. Further, there would seem to be a close connection between the laryngeal affection named and rickets and not a few observers say that this disease is most common during the colder months and that this affords strong grounds for the assumption that many cases of spasmus nutans are due to rickets. Henoeh attaches special importance to dentition as a cause; whereas Hadden has come across many cases in which cutting the teeth neither caused nor aggravated the movements, in the majority of these instances there being an absence of the usual local and general signs of teething. Jacobi refers the affection to rickets as a central cause, or intestinal disorders as a local one, and points out

that there is often a considerable amount of anaemia present at the same time. Ashby (Rep. Soc. Dis. of Chil., v, 256) regards head-shaking and head-nodding as coordinated neuroses associated with purposive or instinctive movements which become confirmed into a habit; the head-shaking movements may first be noticed at the third or fourth months when the infant first attempts to lift up its head or to look about; it is generally seen in infants and children of low vitality, many of them rachitic to some extent, the movements being absent in severe cases of rickets. We have seen that the enlargement of the head in rickets is almost entirely associated with and due to thickening of the cranial bones, there being, as a rule, little or no increase in the cranial capacity and the bones undergoing thickening where ossification is most active. It has been supposed that the increase in the size of the head is caused by hydrocephalus, but it would seem that this is rarely so, the lesion being the least frequent cause of augmentation of the dimensions of the cranium; Holt maintains that rickets and hydrocephalus are only rarely associated and that it is doubtful if there is any etiological connection between the two affections; on the other hand, Jenner insists that the condition is in many cases the outcome of rickets, whereas others have strongly denied this idea. It is not infrequently associated with nystagmus. In rickets the circulation of the brain may be sluggish and there may be hyperaemia of the cerebral meninges. In health the cerebrospinal liquid is controlled by the pressure of the cranial bones and a uniform pressure on the various structures is maintained. In rickets, when ossification is at fault, there is on removal of the pressure excess of cerebrospinal exudation. Fluid distends the cerebral ventricles, the brain substance is compressed, the convolutions are flattened out and the bones themselves yield to intracranial pressure, hydrocephalus being produced. The rachitic hydrocephalic head may be of great size, the face in comparison to the skull being remarkably small. The sutures and fontanelles are widely open and the skin of the head is thin and stretched. The cranial bones are thin and diaphanous. Fluid fills the lateral and third ventricles. The corpus callosum and fornix form an incomplete partition between these, their intercommunication being by the foramen of Munro. The liquid pushes up the fornix and corpus callosum and drives the septum lucidum forwards, with the result that the three cavities are converted into a single one. The basal ganglia are also compressed, the velum interpositum and choroid plexus being free in the effusion. The grey and white matter are attenuated, there is very little fluid in the subarachnoid space, the veins on the surface of the brain are compressed and contain very little blood. The liquid may also distend the fourth ventricle by the usual communication of this with the brain above. The child's head is rounded and globular and in some cases it becomes so large that it cannot be raised from the pillow. On its surface numerous distended veins are observed. The condition is, when it occurs in rickets, usually observed before the fifth month or sometimes up to the eighteenth; it may be congenital and, of course, should the head be ossified before its commencement, there will be no enlargement of the skull. The gradual enlargement of the head may be the first thing observed, or the affection may commence with symptoms of irritability and fretfulness, or of strabismus and convulsions, before any increase in the size of the head is observed. The orbital plates of the frontal bones may be displaced, with consequent depression of the eyeballs. Pressure upon the optic nerve may give rise to neuritis, atrophy or blindness. There may ultimately occur increasing mental enfeeblement, loss of memory, convulsions, weakness or paralysis of the extremities, as well as various special-sense disorders: so that the patient has to keep constantly in bed. The appetite in these cases is sometimes unimpaired; sometimes, however, it is lost and great emaciation occurs. Convulsions, coma, marasmus, or some such intercurrent affection as pneumonia or bronchitis may carry off the patient in a few months or years. Sometimes the patient recovers and lives to grow up, though with some outcome of the affection in the form of muscular or mental enfeeblement. In general, hydrocephalus is rarely associated with rickets, though the production of the disease may be favoured by a combination of the dilated arteries, the low arterial tension, the sluggishness of the arterial circulation, the intense hyperaemia of the cranial

bones and cerebral meninges, and diminished removal of the cerebrospinal fluid from stasis of blood at the periphery. Tumours and posterior basal meningitis are the most common causes of hydrocephalus and it is possible that some syphilitic taint may be present in certain cases. Apart from the actual disease, it is important not to confound hydrocephalus with the enlarged head of rickets. In the latter affection the head is long, square and sometimes asymmetrical; in hydrocephalus it is bulging, globular in outline, often pyramidal and the enlargement, as a rule, is symmetrical in every direction. In rickets the actual increase in circumference may be little above the normal; but in hydrocephalus there may be an increase of many inches and the head, as age increases, grows larger and larger. The forehead in rickets goes up more or less vertically in front and at the sides; in hydrocephalus it is high and projecting, with prominent bulging at the temples, overhanging at the eyes and a characteristic prominence at the root of the nose. The top of a rachitic head is flat; that of a hydrocephalic one is convex and globular, with widely dilated sutures and tense anterior fontanelle, the opening up of the cranial bones as the head enlarges being compared by Trousseau to the falling back of the petals of an opening flower. With recovery the thickening of the rachitic head may diminish gradually and no trace be left behind, though sometimes it persists for life. Laryngismus stridulus is one of the commonest forms of convulsive disorder seen in rachitic children, it being the opinion of most that it seldom or never occurs in the absence of that dyscrasia. As indicated, it consists of a spasmodic affection of the larynx leading to sudden arrest of respiration, such reflex occurrence being excited by such slight irritation as that of a draught of cold air, crying, laughing, fright, anger, pharyngeal tickling, excitement, emotion and the like, and lasting for a few seconds and terminating by a crowing inspiration from the air being forcibly sucked in through the narrowed glottis. The attacks are usually seen at nighttime, though not a few cases, in my experience, have occurred during the day when the child has perhaps been somewhat indisposed, had a tendency to cough or manifested some slight catarrh of the throat or bronchial tubes. At midnight, or later, the child awakes with a crowing or whistling inspiration. It starts up in bed and seems to have great difficulty in breathing; this trouble is obviously in inspiration, expiration being easily and freely performed. The eyes are prominent, the lips blue, the skin not infrequently bathed in perspiration; the pulse is accelerated, small, at times irregular; there is, if the child is old enough to reason in the matter, great alarm; often there is cough of a more or less characteristic kind, it being hoarse, metallic, barking and croupy. If the attack is limited to the larynx, the other muscles not being affected, the child clutches at whatever it can reach and it often grabs at the throat as if there were something there to tear away. The skin becomes cyanotic and all the symptoms of suffocation are present. The voice, though not usually abolished, if altered: it becomes hoarse or husky. In a few minutes the severity of the attack is passed and the child sinks into a more or less disturbed slumber. A second attack may occur the same night, or there may be peace until the following evening. The second attack, if it occurs as it generally does the next night, is usually less severe than the first; the third is still more mild and this generally ends the case for the time being. During the intervals, that is during the day, the child in the vast majority of instances seems little the worse for the night's experience. There will perhaps be a slight cough, with some indisposition and less inclination for its usual amusements. But in some cases there is more marked disturbance of the general health. The spasms are more severe, the cramp is not confined to the laryngeal muscles but involves other parts, such as the muscles of the chest and limbs. During the intervals of the attack there is perhaps a little fever, the digestive tract is somewhat disordered, the cough may be more marked during the day and there may be an increase of the bronchial catarrh present in these cases. Attacks may take place during the day for several days, the cough may continue to be croupy and a hoarseness of voice may be observed. According to my experience, the laryngeal spasms are mostly of a transient nature; it has lasted for from a few seconds in the milder cases to several minutes in the severe forms of the disease. The spasms are intermittent and

they are relieved ~~at~~ intervals by comparative relaxation of the muscles involved. Even in the intervals there is, however, a certain amount of contraction of the constrictors of the larynx, so that relief is not absolute. Two or three days elapse before the attack may be regarded as quite over. In the severer forms one may see the effects of the malady for a still longer period, though definite sequels are not often seen. When the patient has recovered there is no further disease of the kind, only perhaps a tendency to recurrence, another attack being excited by the factors above named. The outlook is, on the whole favourable, the vast majority of cases recovering; the child seldom dies from the affection unless there is some complicating or coincident malady. Pulmonary collapse, so often present during the course of rickets, is increased in probability by laryngismus stridulus and also may be another dangerous factor if the paroxysms are frequent or prolonged. Elsässer says that the affection is perhaps the most common form of convulsions seen in a rickety child and this seems to be the general opinion in many quarters; he thinks that craniotabes is associated with it, though this is usually denied, though it may be associated with general convulsions and tetany; he accounts for its frequent occurrence during the night by pressure of the thinned occiput on the pillow and adds that it can be produced by pressing on a craniotabic patch on this bone. Gee has studied fifty cases and, in all but two, found rickets present; the attacks occurred mostly in males from the sixth to the eighteenth month, Jacobi maintains that the affection is in the vast majority of instances a rachitic expression of meningeal or cerebral irritation. Eustace Smith denies that there is an exciting cerebral irritation, but prefers to regard it as a reflex neurosis consequent upon peripheral irritation existing in rickets and stimulating the irritable nervous tissue. The brain in rickets not infrequently is affected by an excessive supply of blood, which in time may give rise to hydrocephalus from serous effusion; in other instances it is markedly anaemic, or there may be an actual hypertrophy of the cerebral tissue. The mental condition in this disease has been the subject of much discussion, some stating that it is brightened, others just the reverse. It is possible that rickety children, being quiet and indisposed to play with children of their own age, mix more with adult persons and are therefore apt to become very knowing, though they are really usually dull and of defective intelligence. The more frequent tendency of the disease is to induce a condition of lethargy; for the brain and blood-vessels suffer deterioration from the general systemic malnutrition obtaining. The patient prefers to sit quietly by itself, often with its legs crossed like a working tailor, in the so-called pagoda attitude, which same markedly contrasts with the normal activity of a healthy child that is ever on the move. The fact that backward children have suffered from rickets in earlier life has been proved by numerous writers. Bourneville (*Arch. de Neurol.*, 1903) has shown that, in combination with liveliness of spirits and some precocity, a certain amount of mental enfeeblement exists. In 435 feeble-minded children he, with Lemaire, found certain signs of rickets present in 34; in the latter the defects were not congenital, but were first appreciable from the eighteenth to the twenty-fourth month, the exciting cause not infrequently being an attack of gastroenteritis or bronchopneumonia. In a few cases so complicated, deformities of bones alone resulted instead of mental enfeeblement, suggesting that the same toxic or infectious agent was at work, though productive of different results. No stigmata of degeneration were found in the rachitic children, any congenital element of etiology being therefore out of the question. Thackeray, whose head was of peculiar shape, has been cited as an instance of rachitic mental precocity; Jenner has opposed this view and says that extremely rachitic children are almost always deficient in intellectual capacity and power; they are not idiots; they show no signs of idiocy; they resemble rather children of low intellectual capacity and power much younger than themselves. He ascribed the error to the fact that rickety children, removed from the companionship of other children by reason of their physical defects, would spend a lot of their time in the company of adults and would catch their peculiarities of expression, their moods and even some perhaps of their ideas. The same author described the brain in rickets as affected with albuminoid infiltration in common with the liver, spleen, thymus

and lymphatic glands. Lucas (Brit. Jour. Chil. Dis., i, 341) thinks that rickety children are more clever than normal ones, though he seems to be at a loss how to account for this; influenced by this view, he suggests that cases of microcephaly should be so treated that they will develop rickets, in order to enlarge the head and give the brain more room to grow; he adds that persons recovered from rickets, and who have afterwards proper diet, not infrequently show greater capacity for learning; he thinks that rickets and syphilis are the two great causes, however, of physical deterioration in the metropolis of England. The rickety infant is not infrequently very backward in learning to talk, which fact would point to lethargy or impairment of the mental faculties and perhaps justify a positive diagnosis in the presence of other indicative signs of the disease; talking efforts should normally be made about the sixteenth month and markedly progress up to the end of the third year. Renault (Th. de Paris, 1903) presents a careful study of the mental and cerebral conditions in rachitic infants and points out that the large brachycephalic head, with protuberant forehead, differs from the degenerate skull, which is often asymmetrical in shape and of oblique formation; he warns against the theory that rickets is the cause of enfeeblement of the mind, but allows that the latter and the former may be caused by various toxæmic processes of infancy. Ritter v. Rittershain has measured many children's heads and finds that, as a rule, the skull in rickets is no larger than in normal children of the same age. Shuttleworth (Brit. Med. Jour., Oct. 3, 1903) does not believe in the mental precocity of rickety children and states that he has seen the poorer children suffer from much hebetude from general malnutrition, some of the cases being even weakminded, though it would seem that amongst the children of the well-to-do classes abnormal mental development may occur. Smith comes to the conclusion that, apart from all physical changes, "the behaviour of the rickety child is very characteristic and of itself almost sufficient to warrant a diagnosis of the disease. He goes on to remark that the "quiet, the repose about him strike the observer at once. Such a child, if able to support himself, will sit for hours, his legs stretched out straight before him on the floor, perfectly contented if only allowed to remain unnoticed. All that he wants is to be left alone. A healthy child delights in movements; a rickety child is only happy when at rest; his greatest pleasure consists in inaction. To look at him we are irresistibly reminded of the other term of life, for he appears to have anticipated at least one consequence of the weight of years and to have combined the patient endurance of old age with the face and figure of a child." Trousseau regards rickety children as possessing precocity and intelligence beyond their years and suggests that this is due to the fact that the softness of the cranium favours more easy development of the nervous centres, which results in the intelligence being in advance of the age of the patient. He adds that "these little suffering creatures, who are unable to walk about without assistance, generally possess a greater intelligence" than others and that their physiognomy, so often stamped with sadness and suffering, their expression of countenance, their way of speaking all denote an advanced development of the intellectual faculties." On the other hand, Taylor thinks that such individuals are dull-witted, deficient in intelligence and weakminded. Warner (Study of Child., 233) fails to come across any evidence of precocity in the rickety child and notes that these children are delicate, grow up stunted and that about a third of them make dull pupils. The conclusion which I have formed from actual experience of these cases is that in all probability the mental power is lowered by such a profound disturbance of nutrition as obtains in rickets; also that any precocity occasionally observed cannot be regarded as real or permanent. A nervous accident, particularly of tender age, which has for its special feature a spasmodic contraction of the flexor muscles of the forearm and back of the leg, is that known as tetany or carpopedal spasm and common in rickets with or without the association of laryngismus. There occurs a tonic spasm of the muscles of the extremities; the calf muscles seem hard, the foot is pointed, the wrist is pronated and flexed and there is spasmodic contraction of the interosseous muscles. This functional disorder may appear quite suddenly with spasmodic twitchings of the limbs, though usually

one observes certain premonitory symptoms in the form of prickling, numbness and sensations of heaviness in the arms and legs, after which the muscular trouble commences. The thumb is bent into the palm, the fingers are flexed at their junction with the **carpus** and extended at other joints, producing an appearance of the accoucheur's hand. This form of contraction is, however, not always present, the fingers being sometimes, with the thumbs, tightly flexed. The wrist is bent upon the forearm and the latter flexed sometimes upon the arm, which is generally held tightly against the trunk. The thighs and knees are extended, the toes flexed and the foot inverted. The contractions are tonic and the muscles are hard. Some pain may be experienced during the muscular contractions, though not always. In mild cases the spasms affect only the arms and possibly the lower extremities. It is usually associated with laryngismus stridulus. In severe cases the body muscles may be implicated and a condition of opisthotonos develops. In the most serious cases the entire body may appear as stiff as in tetanus, even the muscles of the face becoming contracted and the child seeming tetanised; nevertheless tetanus is rare and occurs only late in the course of the disease. The attacks last for from a few minutes to several hours, or even for many days. They are not accompanied by a rise of temperature, or any serious effects upon the system, and may subside for several weeks or months and again return; in rare cases the affection may manifest itself for months or years, though the usual duration does not exceed a few weeks. It is possible that epilepsy may be induced and oedema of the hands and feet, due to venous obstruction, is occasionally observed. Many of the cases have been referred to dilatation of the stomach from fermenting food. Burnett (Med. Press, 1905, p. 54) says that tetany, not due to gastric disturbances, is invariably originated by rickets and both Bourneville and Devine conclude that dilatation of the stomach and hyperacidity favour the occurrence of the affection. Holt considers that tetany and laryngismus stridulus are very rare in the absence of rickets; and Jacobi thinks that the majority of cases are connected with this disease, especially when there are marked cranial symptoms, such as craniotabes, laryngismus or other rachitic expressions. The diagnosis of the malady is of importance and there are useful signs which can be employed in its accomplishment. Chvostek's symptom, or facial irritability, is also not infrequently present, even, it is said, when there are no other nervous peculiarities present. It consists of a peculiar irritability of the motor nerves. This is so marked that striking a motor point gives rise to a muscular contraction which may remain for many seconds. When the motor point of the facial nerve is struck, a spasm of the muscles of that side of the face is produced, this phenomenon constituting the symptom in question. The masseter muscle is a convenient one for the detection of the sign, which is a symptom of considerable moment, being a clear indication of the nervous irritability of the child. It is, we have seen, not infrequently associated with laryngismus; but often it exists alone and is then strongly suggestive of convulsive tendencies. A similar nervous irregularity may sometimes be noted in connection with the muscles of the limbs and other parts. Another peculiar sign is that called after Erb, consisting of an increased electrical irritability of the muscles and nerves, especially to the continuous current. A negative polar contraction is occasioned by a very weak current of a fraction of a milliampère and, if this current is made stronger, the contraction becomes tonic. The positive pole opening may also be tonic and there may be an opening tetanic contraction with the negative pole. Hoffmann's symptom consists of a peculiar irritability of the sensory nerves, which is appreciable when they are pressed upon, sensations of prickling and of formication being experienced along their course. There is an increase also in the electrical sensibility, as can be shown by the application of a very weak galvanic current. The sensibility of the auditory nerve is also abnormally increased. Thomas's sign is a peculiar electrical condition in which the kathode, when placed over the nerve, produces first fibrillary, and then tonic, spasms in the muscles supplied by the nerve; but when the anode is substituted for the kathode, no contraction is seen with even so strong a current as five or seven milliampères. Trousseau's symptom is a condition in which, when the upper extremity is grasped so that the vessels and nerves on the inner side of the limb are firmly compressed,

after an interval of from a half to two or three minutes the hand presents the characteristic appearance already described. The careless observer may mistake the condition for tetanus, but not when he bears in mind the history of the case and the fact that it has not the grave import and exact expressions of that disease. The spasms are usually restricted to the limbs and are of comparatively short duration; there is, besides, no affection of the jaw or bending of the back. The spasms in tetany begin in the extremities and extend up the trunk and the muscles of the face and neck are seldom implicated; there is, further, no suggestive wound or injury. The outlook is, on the whole favourable, despite the fact that it is a distressing and alarming complication at first sight. Finally, it may be noted that rickets may leave a lasting impression on the nervous system, particularly if the child is the offspring of neurotic parents; though the rachitis has disappeared, the patient may grow up unduly excitable and emotional, possibly with only slight nervous disorders, such as twitching of the muscles of the hands and face and more or less constant restlessness. Disordered digestion, overpressure at school and excitement easily produce abnormal irritability, sleeplessness, as well as night-terrors, leading to more serious disease; and the convulsive affections of the parent affection may be the precursors of true epilepsy. The patient may readily fall a prey to neurasthenia or hysteria when grown up, unless carefully supervised when young. The possibility of paralytic simulation should be borne in mind; the inability or unwillingness to move the limbs and the tenderness of the body when handled may even lead to the suspicion of the presence of infantile paralysis, though closer investigation will show that there is no real loss of power in the limbs and that the knee-jerks and the electrical reactions are normal. Starr, dealing with this possible confusion, states that rickets may lead to a sudden febrile onset, with much pain and tenderness in the extremities and inability to move. The child, he remarks, is not really paralysed and the tenderness of its bones, the appearance of the gums (he is evidently referring to scurvy or scurvy rickets) and the sweating, as well as the lack of limitation of the pain and immobility in one or two limbs, should prevent this disease being mistaken for infantile paralysis. The true character of the nervous disorder in a child is apt to be overlooked and, unless it is realised that the origin of the mischief is rickets, - as evidenced by tetany, convulsions or laryngismus, - no treatment will result in permanent cure.

Pelvis

The various rachitic deformities of the pelvis are of great importance because of their effect upon childbirth. Chalmers (Phys. Deter. Rep., 240) says that the number of operations for contracted pelvis have increased in recent years in Glasgow, these ~~concerning~~ concerning the women who required osteotomies as children a generation ago. The number of such operations, however, is, in general on the decrease, so that the outlook for the obstetrician of the future is not unpromising. Owing to the softness of the pelvis bones in this disease, the improper use of tight napkins may give rise to deformities of these structures and pads, if firmly applied between the thighs, may wedge out the legs and produce curvature of the femora. The risk of this should be recognised even in the case of non-rachitic infants. It is by no means invariably a simple matter to discover the pelvis deformities due to rickets, as the bones are not easily felt in very young children. In common with the rest of the osseous system, the bones are soft and there may be thickening of the ilia about their edges. The pressure of the spine and of the abdominal organs downwards, acting conjointly with upward pressure from the legs, crushes in the softened bones, with the result that the whole pelvis has a distorted and stunted appearance. The alterations induced will, in general depend upon the age of the patient at the commencement of the disease and the degree to which ossification has advanced, arrest of development and distortion being commonly observed; the position of the child and its ability to walk will also have an important influence. One of the commonest deformities is that known as the flat pelvis, in which the iliac fossae are flatter than normal and of forward inclination; it is not infrequently contracted as well as flat, because of the arrested development, though softening of the bones is not a marked feature.

What is termed the infantile pelvis is sometimes produced by rickets through arrest of development, though also without much softening of the bones. Bone softening is best seen in the sacrum, where it gives rise to the so-called reniform or kidney-shaped pelvis or brim. The sacrum sinks between the ilia and its promontory is rotated forwards. The pubic arch is widened and, in comparison with the normal pelvis, the relative size of the transverse diameter at the brim and of the distance between the ischial tuberosities is increased, the general effect of all this being to occasion a shallow passage. This form of pelvis is produced before the child can walk: hence the relative widening of the cavity, especially at the outlet, counterpressure at the acetabula having little influence. The weight of the body in the sitting position, plus the counterpressure of the tuber ischii, occasions the widening. There is a diminution in all the diameters of the generally contracted pelvis, though there is no change in their relative proportions. Occasionally the conjugate diameter is contracted more than the others. The iliac fossae look forwards, but the pelvis is not flattened. This form is produced late in childhood after the child has commenced to walk; the tendency to widening and flattening is then counteracted and there is a certain amount of **softening** of the sacrum and ilia in this kind of deformity. The triradiate or pseudomalacosteon form of rachitic pelvis is produced after the patient begins to walk; in it the acetabula are forced inwards and softening of the bones occurs. In the case of the scoliotic oblique pelvis the curve is generally to the right in the dorsal region, to the left in the lumbar part of the spine. Owing to the lateral displacement of the line of the body-weight, that side has to bear most of the weight, with thickening of the bones and muscles. The acetabulum is pushed more on this side and the symphysis pubis is displaced towards the opposite side. Certain cases show irregular deformities, or pelvic crumpling as it is called.

Pharynx

Spinal deformities may be associated with the presence of adenoids in the nasopharynx, as I have often observed. Indeed, rickets is one of the affections in which such growths and hypertrophy of the tonsils should always be carefully examined for. Some writers contend that they are due to lack of exercise, but it would seem that they are part of the general hyperplasia of the connective tissue of all the glandular structures of the body; if the fibrous component preponderates, the tonsils may be as hard as a fibrous tumour. These enlargements may interfere with swallowing, as well as the free entrance of air into the windpipe, and they and their associated nasopharyngeal growths and catarrh are sufficient to give rise to deformity of the chest in the shape of pigeon-breast. In every case a radical operation should be advised. Deafness not infrequently results from obstruction of the Eustachian tube and children with enlarged tonsils are more apt than others to contract infectious diseases, such as scarlatina and diphtheria, which not infrequently prove fatal. Mouth-breathing, snoring and nasal voice are common and characteristic phenomena and the widely open mouth gives these individuals a characteristic physiognomy. At puberty these tumefactions tend to take their departure, though it is advisable that this be anticipated by early operative measures.

Respiratory Organs

We have already seen that the shape of the thorax is not without influence upon the condition of the respiratory organs within. The lungs of infants are comparatively small and the air-passages narrow, the ~~domeshaped~~ diaphragm ascending higher in the thorax than in the adult. The chest yields readily to lateral pressure, as it has little power of recoil: so that, owing to the smallness of the lungs and the delicacy of the bronchi and vesicular tissue, which are easily stretched or collapsed, any obstruction is very apt to give rise to atelectasis or emphysema. The spinal and abdominal muscles are also accessory to the act of respiration, which is governed by a special centre in the medulla and automatically performed independent of afferent stimuli, direct stimulation occurring from the access of venous blood. Anything interfering with the respiratory movements, or any obstruction to the pulmonary circulation or even the commonly present anaemia of this disease, interfering with the normal interchange of gases in the blood and the alveolar air, ill

produce venous blood and so give rise to particular effects upon the system. In short, the delicate respiratory mechanism, dependent as it is upon the due association of the various tissues of the body, is easily thrown out of gear and respiration, like all the other systemic functions, must perforce operate under a severe handicap in a rachitic child. The narrow and compressed chest, the soft inelastic ribs, the protuberant abdomen, pushing up against the diaphragm, and the enlarged liver and stomach and intestines, distended with gas, interfere in the rickety child with the normal act of respiration, which is still further hampered by the atonic condition of the abdominal muscles. The respiratory act may also be adversely prejudiced by the spasms of laryngismus stridulus, or by any catarrhal state of the bronchial or alveolar tissue, causing obstruction to the free entrance of air: so that the thoracic wall is drawn inwards laterally to a still greater degree, with the result that the proper expansion of the lungs is obstructed. The diaphragm participates in the flabby condition common to all the muscles and the crowding up of the abdominal viscera serves to hamper its freedom of action. It never completely loses its fulcrum, however, because the encroachment of the enlarged abdominal viscera into the base of the chest keeps the lower costal arches supported and widely expanded at the same time. The spine is lax and therefore cannot render assistance and the mechanism of the intercostals is almost paralysed by the softening of the thorax, as well as by the want of support usually afforded by the ribs and normal interosseous spaces. The loss of abdominal power, together with the weakness of the dorsal muscles, causes the child to be unable to adopt the erect posture and the force of elastic recoil is diminished; the diaphragm, not having any rigid fulcrum, - as in adult abdominal atony, - cannot force down the contents of the belly and the expiratory elastic recoil is diminished to an equal extent. It is therefore evident that the act of respiration in the rickety child is performed under most disadvantageous conditions, to which must be added also the great tendency for catarrhal conditions of the mucous membranes of the bronchi and lungs to occur, which are readily excited during the course of the disease and constitute a special danger to life. The rickety child is always catching cold; its mucous membranes are in a sensitive, irritable and unhealthy state and readily attacked by the germs of disease. The embarrassed respiration resulting from the alterations in the chest wall and diminished capacity of the thoracic cavity, leading to deficient aeration of the blood, produces, according to its severity, dyspnoea, hyperpnoea, orthopnoea or asphyxia; in some cases interference with the respiration is marked. The general perversion of nutrition obtaining in rickets in itself predisposes to bronchial catarrh consequent upon deformities of the chest and weakness of the respiratory muscles, with possible culmination in acute bronchial inflammation, bronchopneumonia or collapse of the lung tissue. Enlargement of the tonsils is, we have seen, frequently present in rickets and the same, together with nasopharyngeal growths, may further interfere with the entrance of air into the lungs. In the case of bronchitis the contents of the swollen bronchial tubes will impede the proper circulation of air; the air imprisoned in the alveoli becomes absorbed and the vesicles collapse, particularly at the roots of the lungs and in the middle lobe of the right lung. To supply the deficiency compensatory emphysema occurs, the support of the adjacent lobules being withdrawn. The result of this is a loss of capillary area and consequently of aerating pulmonary surface, which will render the respiration still more feeble and inefficient. There may also be collapse of pulmonary tissue from hyperscoliosis. Some mention has already been made of the production of grooves in the chondro-costal articulation areas, with projection of the breast bones. To fill up this space the air cells are distended during inspiration; emphysema is thereby produced at the anterior edge of the lungs in the parts which are least supported. During the intake of air the beadings on the ribs, which project inwards, press upon the lung and prevent the lobules from becoming distended and in this way there is occasioned a collapse groove corresponding to the rickety roary. It very often happens that the bronchopneumonia or bronchitis of rickets seriously threatens the life of the child. The respiratory muscles are weak and the ribs softened; the lower ribs are everted, rendering, especially in the presence of the characteristic

"pot-belly", the movements of respiration less efficient. A kind of vicious circle is thereby induced. On account of these mechanical difficulties and defects the lung is imperfectly filled with air and the collapse thus induced favours the occurrence of bronchitis and further atelectic conditions. The superaddition of catarrh of all the mucous membranes of the body renders the mechanical deficiencies worse. Further, the rachitic child is usually badly nourished, very often lives amidst unhygienic surroundings and is very liable to a variety of dangerous debilitations. Some of the cases closely simulate pulmonary tuberculosis, especially when such glands as the thymus are enlarged in addition to the chest lesions and the adenopathy may in itself be the cause of fatality. These patients are apt to suffer from asthma or tuberculosis in after-life and there may then be gastrointestinal or other chronic catarrhs. Chronic bronchial catarrh or bronchopneumonia may be occasioned by the smallness of the lower half of the contracted chest, which affections are still further predisposed to by the tumefaction of the tracheal, bronchial and mediastinal glands, the dulness over which may give rise to a suspicion of tuberculosis, especially if there is wasting of the body at the same time. The type of respiration is often altered in rickets. The normal sequence in an infant is a short inspiratory act, a moderately-long expiratory one and a short pause. It may become inverted, with the result that the expiration is prolonged, the inspiration short and jerky and the pause abnormally long. The type of breathing is abdominal and inferior costal, one great peculiarity of infantile respiration being its extreme rapidity and great versatility of form. Even when there is no evident pulmonary disorder in existence, the breathing is rapid and shallow, possibly owing to some irritation of the respiratory centre. The rickety child sometimes prefers to go to sleep in a kneeling position, - genu-pectoral, - with its head on the pillow, a position often adopted by the subjects of cardiac dyspnoea. In rickets, owing to the atony of the muscles, the belly remains motionless and protudes very little during inspiration. This, together with the failure of the upper part of the chest to expand, throws all the work on the midriff, the only active respiratory movement being inferior costal. There is no normal inspiratory expansion, but the action of the muscular structure just named causes recession of the thoracic wall, which makes room for re-expansion during the next expiration. This probably can be explained by the fact that the lowered resistance of the ribs and cartilages is more readily overcome by the atmospheric pressure than by the imperfect expansion of the lungs: so that, instead of an increase of the volume of the lung, there is a recession of the chest during inspiration, which can be measured to some extent by the depth of Harrison's groove. Very often, owing to collapse and emphysema of the lungs, the rickety child suffers from dyspnoea and cyanosis. Carmichael regards chronic cough as one of the most common symptoms of rickets and holds that, whenever a young infant suffers from that symptom, there is a presumption in favour of the disease. Duklesky (Arch. Ped., 1904, p. 790) writes of a new sign which he terms "polypnoea", by which he means rapidity of breathing, the respirations being in his cases increased variously from 56 or 80 to 118 per minute. He says that the sign is produced by either irritation of the respiratory centre in the medulla by the excessive amount of carbonic acid in the blood, by the diminution in the size of the chest, or by the hyperexcitability of the central nervous system induced by rickets, all his patients recovering under proper treatment of the underlying disease named. Jacobi says that, though the heart is of average size, the arteries are abnormally large and there is a lowering of the pressure of blood, which gives rise to a slow and sluggish circulation in the respiratory organs, with a tendency to congestion and catarrh. Trousseau dwells upon the marked interference with the respiration in some cases. "This habitual oppression", he says, "is a constant phenomenon in confirmed rickets, particularly when it occurs during the first two years of life. At a later age, that is to say in children of three years, it is an exceptional phenomenon. In the very young infants it exists in an extreme degree. Bear in mind that at a very young age the disease first appears in the chest, which is the seat of the earliest deformities; while in children who have begun to walk they are first seen in the lower extremities; the degree to

which the thoracic deformities proceed and the extent to which - ~~in~~ the play of the respiratory organs is embarrassed should also be remembered. The embarrassment of the respiration, which plays so strong a part in the disturbance of nutrition by rendering imperfect the process of haematosis, makes rickets more liable than other children to acute pulmonary affections, bronchial catarrhs, catarrhal pneumonia, which, in consequence of the obstacle to free inspiration occasioned by the thoracic deformity, assume a very great degree of gravity; he adds that he has found that consumption of the lungs is not often encountered as a complication of rickets. According to Wachsmuth, carbonic acid in the blood leads to a chronic intoxication provocative of all the rachitic lesions.

Scapulae

The shoulder-blades are apt to be thickened in rickets, especially along their lower border; their anterior aspect may be more concave than normal or perhaps too round. If there is much deformity, proper movement of the shoulders may be interfered with.

Skin

Various affections of the cutaneous system may be observed in rickets, some of them of a more or less troublesome character. It occasionally happens that there is a more or less general hyperaesthesia; but the presence of this would indicate a complication with some other affection, such as scorbutus, though it would seem that it can appear independent of the latter. Trophoneuroses, local ischaemia, coldness and cyanosis of the limbs have also been seen. The skin of rachitic children is usually pale and flabby; but not infrequently there is an abnormal amount of fat, so that the patients appear to be extremely plump and well-nourished for their age. Sometimes, however, there is great emaciation, especially if there has been a tendency to wasting from syphilis or other disease. Perspiration, especially of the head, is a very early symptom; it is usually seen at night and may be so profuse as to soak the pillow. Some authorities have insisted that this head-sweating is connected in some way with the hyperplastic changes in the cranial bones, whereas others maintain that it is due to the action of some irritant on the glands of the skin of the head or some reflex nervous irritability. The veins of the scalp are markedly enlarged and by their pressure form grooves in the softened cranial bones. Some of the cases do not present any signs of sweating of the head: so that it is not a pathognomonic as has been alleged. The sweating gives rise to various sudamina, which vary in size from a slight eruption to a papular efflorescence. In addition to ordinary perspiration of the head, there may be an excessive secretion of the sebaceous follicles, leading to seborrhoea, crops of miliaria and, if the head is much neglected, impetiginous eruptions. The hair of the rachitic infant, instead of having the bright and glossy appearance of health, is not infrequently dry and dull, thin, wispy and of poor quality. The skin of the body is, in general, soft and easily irritated: so that erythematous and eczematous rashes are of frequent occurrence, more particularly about the head and neck, as the excessive perspiration keeps up an unhealthy, sodden condition of these parts. Nevertheless, skin eruptions are not found in all cases of rickets; for not infrequently I have seen cases in which the skin is apparently quite healthy, though rather pale. Strophulus, - a tooth-rash or papular infantile eruption, - and kindred dermic lesions are apt to be wrongly ascribed to dentition when really due to rickets, and various rashes on the body and extremities are somewhat difficult to cure, owing to their dependence upon the general perversion of nutrition induced by the disease. The glands of the skin may undergo enlargement and continual scratching of the body surface with infected fingers may lead to crops of boils, which, with the enlarged glands, are apt to occasion much suppuration. There may be eruptions of eczema, impetigo, or lichen about the body and, if there is much gastrointestinal disturbance, urticaria - in an acute form and suggestive of a toxæmia - may be observed; these and other rashes are apt carelessly to be assigned to the infant's teething. Irritable eruptions, such as prurigo or pruriginous strophulus, are sometimes in evidence, the latter being characterised by the development of discrete watery papules. The child is continually on the scratch and the part begins to

bleed; if this condition being allowed to become chronic, it is possible that later on it may progress into the chronic prurigo of Hebra, which is almost incurable. The diarrhoea and acid and slimy motions are apt, in the absence of assiduous care, to scald or irritate the buttocks, genitals and the back of the thighs: so that in these parts of the child's anatomy various eruptions are nearly always to be seen, the most common being erythema, eczema, impetigo and echthyma. The last-mentioned one may leave such deep and permanent scars that a suspicion of congenital syphilis may be raised.

Spleen

The spleen is apt to be enlarged in rickets and, post-mortem, a condition of fibrosis or hyperplasia may be observed. It is not peculiar to this disease, as it occurs in such others as syphilis and the anaemia originated by insufficiency of that organ.

Stature

Rachitic children are apt to grow up into individuals of comparatively low stature, owing to the permanent shortening of the ~~limbs~~/extremities, the rachitic child of three years not infrequently measuring from six to eight inches less than a healthy one of the same age. The blighting and growth-limitation of the bones may culminate in permanent dwarfism - a condition, however, sometimes produced by cretinism or other affections. We have already referred to Guérin's affirmation that the diminution in length is so constant that, the dimensions of a rachitic bone being known, the dimensions of the other components of the skeletal system could be approximately determined with considerable accuracy.

Stomach and Intestines

We have seen that the rachitic infant's stomach is very apt to undergo dilatation, which may persist into adolescence long after the originating disease has been recovered from. Some of these children have a tremendous appetite and are very thirsty - phenomena which have surprised in quite a recent case. By the continual overloading of the stomach with food, any gastric catarrh present is aggravated and the constant distension keeps up the dilated condition of the stomach. There may be a primary gastric catarrh present, though more often a secondary one excited by excessive or injudicious feeding. This leads to the production in the stomach of mucus and free acid, which interfere with the proper assimilation of food, the resulting anaemia being responsible for the anaemic condition of the patient. The gastric catarrh of rickets is invariably of the acid type, the neutralisation of which should always be effected as an essential part of the dietary. In common with all the mucous membranes of the body those of the stomach and intestines are very liable to catarrh, which same is readily excited by cold or dietetic errors; it is particularly obstinate to treatment and exhibits sometimes annoying relapses. The intestines, particularly the colon, are apt to be dilated; in this way, moreover, the occurrence of inguinal or umbilical hernia or of rectal prolapse may be favoured. Diarrhoea is a somewhat constant symptom and is always aggravated or excited by bad feeding. The stools may be acid, green, foetid and slimy, containing much undigested food; or there may be plentiful pasty and stinking evacuations deficient in bile, with flatulence, fermentation, colic and abdominal pain. Constipation may alternate with diarrhoea and there is a great tendency for the gastrointestinal disorders to become chronic, especially if the parent rickets is allowed to proceed untreated. Rickets is said to be the great slime-producer and its malign influence may perhaps be seen in the mucous disease of older children, the dyspepsias and gastrointestinal affections sometimes persisting long after the bony deformities of the disease have disappeared and constituting a more or less continual source of annoyance or danger.

Teeth

The condition of the first dentition affords a useful clue to the presence or absence of rickets, in which disease the teeth fail to make their appearance at the proper times, the infant of twelve months not infrequently having toothless gums. Indeed, a toothless mouth at even the age of ten months should excite a strong suspicion of rickets and lead to the proper treatment being instituted without delay. It may happen, too, that the infant cuts one or two teeth prior to the development of

the disease, which will lead to an arrest of dentition for several months and afford considerable diagnostic aid. In addition to their late appearance, there may be cross-cutting, in which condition the teeth fail to appear in their normal sequence; in all cases there is difficulty over the teething and the teeth, when cut, are apt to be very brittle, deficient in enamel, - which is often striped and pitted, - and prone to early decay down to the alveolar edge, leaving the child with unhealthy stumps in its mouth long before they can be replaced by the teeth of the second set and constituting a prolific source of infection of the buccal cavity and parts lower down, with the usual local disorders. The development of convulsions is not infrequently seen in these cases. The second dentition is also apt to present peculiarities, the teeth being not unlike the peg-top ones of syphilis as regards the incisors, but differing therefrom in the respect that in rickets the malformation in question is essentially an arrest in the development of the enamel, whereas the syphilitic tooth is pegged and possessed of good enamel. The two varieties of teeth may occur together, if syphilis be present at the same time. In the case of rickets, so defective is the enamel that the rough dentine looks as if it had been pushed through a sheath, or the condition may only be indicated by two or three dotted grooves parallel with the free border, giving the tooth a worm-eaten appearance, or one like cliffs worn away by the action of the waves - the so-called tidal-mark tooth in contradistinction to the peg-top one of syphilis. It is probable that this early dental decay is associated with a deficiency of lime salts in the teeth and is sometimes associated with cataract of the zonular type and convulsions.

Temperature

According to my experience, the body-heat in rickets is rarely above the normal in the absence of heat-raising complications; indeed, I have very often, particularly in marasmic or asthenic cases, found it subnormal, with occasionally slight evening rises. Bronchitis, bronchopneumonia, acute gastrointestinal catarrh or other severe complicating conditions, would, of course be responsible for considerable hyperthermia. In general, even when the child seems very hot and is continually kicking off the bedclothes, there is little or no elevation of temperature.

Thymus Gland

The thymus is sometimes found enlarged in rickets, particularly in convulsive cases, which fact may have some bearing upon the nervous anomalies observed.

Tongue

Apparently there is nothing characteristic about the condition of the tongue in rickets; it is usually clean, though sometimes slightly coated, when, if nocturnal grinding of teeth and nasal irritation be present, the presence of worms may be erroneously diagnosed and treated in the usual cathartic way. When there is much catarrh of the gastrointestinal tract the tongue is more red than normal and not infrequently sore, especially at the tip and edges, where small papules may sometimes be observed to occur and break down and ulcerate. In certain cases the so-called geographical tongue is seen; on the dorsum of the organ there may be seen little islands, which are red, margined and deprived of their epithelium; these patches increase in size and number and extend backwards to the root of the tongue, often healing up and then making their appearance again. Parrot held that they were of syphilitic origin; but this is not the case, as the islets correspond exactly with the erosions seen near the solitary glands and those of Lieberkühn in the intestines and may merely indicate an incompetency of absorption in that locality and abnormal secretion.

Tuberculosis

The frequent attacks of bronchitis, to which the rachitic patient is so prone, may culminate some day in attacks of asthma or pulmonary tuberculosis, is an intercurrent and also predisposing bronchopneumonia do not prove fatal beforehand. Rickets, as was formerly supposed, does not protect from tuberculosis, which latter, in whatever form it is engrafted upon rickets is almost certain to carry off the child, perhaps very suddenly.

Urine

I have never known the urine to exhibit any striking characters in rickets on analysis, though it may contain a slight excess of lactic acid, but no increase in the amount of lime salts, as a rule. It is generally of a pale-straw colour when passed and, in gastrointestinal cases, is said sometimes to have a high earthy-phosphates content. Baumann (Jahr. f. Kinder., 1906, July 18, p. 212) says that there is no increase in the ammonia, it is of acid reaction and has no peculiarity of odour when freshly voided, though at times perhaps strong. When catarrh of the bladder is present, there is liability to infection with the usual germs and irritability may be observed. Then increased frequency of micturition will be observed, which Carmichael (Dis. of Child.) regards as an early diagnostic sign of rickets. Some have reported a strong odour of methylamine in these cases. Fagge (Trans. Path. Soc., xxxii) tells of an increase of uric acid in rachitic urine - an observation which my own experience has not confirmed.

Vertebral Column

In view of the rachitic softening of the various osseous components of this structure and the weakness and flabbiness of their ligaments, it is not surprising to find that certain deformities are prone to occur. One of the commonest of these is hump-back or hunch-back - kyphosis as it is called. In it the spine is bent backwards and a hump is developed in the dorsal and lumbar parts, with the usual compensatory curve below it forwards; the normal curve is increased in the cervical part of the spine and the head seems to sink upon the shoulders, a peculiar attitude being perforce assumed. The face may even be directed upwards by this backward falling of the head. It is important that this condition be not confused with the caries of the spine which usually results from tuberculosis - i.e., with Pott's disease. But in the rickety curvation there is more rounding and an absence of the sharp angular projection seen in tubercular spinal disease. Further, if the child be laid on its belly and lifted up by the legs, the curvature of the spine, if due to rickets, will straighten out and disappear; whereas this phenomenon is not seen in spinal consumption. Holt maintains that marked lateral curvature in children who are not over three years of age is usually due to rickets, for the recognition of which the history of the case and the condition of the muscular system would also be of importance. Nevertheless, it sometimes happens that Pott's disease and rachitic curvation exist together at the same time, with diagnostic confusion as a result. In some cases there is a blending of scoliosis with kyphosis - kyphoscoliosis, as it is termed. Scoliosis alone may be observed; if the infant sits on the mother's right arm, the trunk curves to the left side and vice versa. Kyphoscoliosis may compress the lung and give rise to collapse of the organ. The hump-back and scoliotic deformity may be appreciated by placing the child in the prone position and employing gentle traction on the legs. Many of the cases present such deformities for life. Rachitic children sometimes complain of spinal pain, a symptom common in other diseases which should not be confused. Ewart (Brit. Jour. Chil. Dis., ii, 157) dwells upon the flexibility of the spine seen during early life and shows that it assists greatly in the respiratory function and at the same time gives practice to the muscles, which in later life will hold the body erect. In the adult the spine is a firm bony column, but in the infant it is a flexible rod capable of vermiform writhing movements, which become very noticeable when exaggerated breathing movements are present from any cause, any deformity of the chest, as in rickets, or obstruction to the proper performance of respiration reacting with malign effect upon the child's vertebral column. In all cases, particularly if in diagnostic doubt, search should be made for the presence of adenoids in the nasopharynx, as they are nearly always found and prove suggestive.

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D I A G N O S I S

GENERAL CONSIDERATIONS

In most cases of rickets there is little or no diagnostic difficulty, particularly if the various osseous anomalies of the head and chest and limbs, the lateness of walking, the head-sweating, the nervous disorders and the general nutritional perversion be borne in mind. The contrary is the case, however, in the early indefinite cases of atypical evolution; and the same obtains in cases in which only one part of the body or some particular tissue is affected. Thus, it may happen that the only disorder which the very young infant presents is constipation with some anaemia; nevertheless, the persistence of such should raise a suspicion of the disease in the absence of apparent cause. This remark applies also with equal force to the development of inexplicable catarrhs of the respiratory and gastrointestinal mucous membranes - not a few of these cases suffering perpetually from colds, mild attacks of bronchitis or bronchopneumonia, with perhaps repeated attacks of indigestion or bowel inflammation and even gluttony. There may also be suggestive symptoms observed in connection with the nervous system, such as nocturnal restlessness, head-sweating, night-terrors, dislike of bedclothes and convulsions, which latter, occurring in an apparently healthy child after the age of six months are usually indicative of rickets when not referable to teething or similar irritative neuroses. It is very necessary that too much importance should not be attached to the fatness of the child, this being no criterion of the absence of rickets, despite the assurances of patent medicine vendors and trustful mothers aspiring to prizes at baby-shows. On the other hand, it is just as important to note that emaciation is no certain sign of rickets, as the patient may be much attenuated by marasmus without the former taint. According to Eustace Smith, "the head, elongated from before backwards; the square, straight, prominent forehead; the small face; the beaded ribs; the deformed chest; the twisted, distorted limbs; the immobility and quiet of the little creature as he sits - if he can sit - with bowed spine and head thrown back, gazing around him with vacant eyes; all these characteristic symptoms leave no room for doubt as to the nature of the disease!" It is the attenuated forms, the partial varieties and the initial stage of the malady which require close investigation before rickets can be diagnosed with something like certainty. The parents themselves may not suspect the presence of the affection; for, as stated the fatness of the infant is their great pride. The absence of teeth, often at a late age, is apt to be regarded as nothing very unusual, or at least by no means harmful, particularly if the family tradition is to the same effect. In short, no alarm is created until something definite of the morbid kind occurs. It is a good rule that, if the ninth month passes without a tooth being cut, the cause should be carefully investigated and will not infrequently be found to be rickets. The first thing that alarms the mother is usually delay in walking; it is paralytic she now fears, though professional examination demonstrates the fact that the motor nerves are not diseased, as the muscles, though weak and flabby, are capable of action and there are other signs of rickets possibly in evidence which she has overlooked. The reflexes are normal in these cases and there has been a gradual onset of osseous affections. Beading of the ribs, though very common in this disease, is not certain proof of its presence, as it may be found in normal children soon after birth from some non-rachitic cause. The softening of the cranial bones, with thinning of their edges and separation of the sutures, particularly if accompanied by a yielding under pressure, may be regarded as suggestive of rickets. Holt (Dis. of Child., p. 267) says that "the most important symptoms for diagnosis are sweating of the head, cranio-tabes, great restlessness at night, delayed dentition and enlarged fontanelle. All these separately may mean something else, but collectively they can mean nothing but rickets" If it is carefully remembered that the latter is a constitutional disease, with a slow and insidious onset and not merely an affection confined to the osseous system, the diagnosis will be made in its earlier stages and the affection arrested before permanent deformation or dwarfing of the body occur.

It has often been my experience to find that infants suffer from continual gastric disorders in early life due to improper feeding; in these cases it is often impossible to discover any signs of rickets, but I, nevertheless, endeavour to keep a careful watch upon the child and warn the mother that persistent improper or excessive feeding may produce malnutrition, which will inevitably occasion the development of the disease in question. In no case do I feel satisfied if the child is merely fat and putting on weight when its tissues are flabby and the teeth are not being cut at regular intervals, or when standing or walking are delayed; in such cases I feel justified in making a provisional diagnosis of rickets at least. In actual practice I have found it very useful to make a sort of tabulation of the cardinal symptoms of rickets and commit them to memory, with a view to running over them when examining a case; in such a way little of importance is missed and much useful information is forthcoming in many instances. Thus, anaemia is a constant and early symptom and, in the severe forms of rickets acute splenic anaemia may be observed. The child's chest is narrow, there is beading of the ribs, Harrison's groove and prominence of the sternum, with central depression, may be noticed. The malady is rarely congenital and is usually encountered after the sixth month of life. Constipation may be present, but is by no means a regular feature of the disease. Dentition, as we have seen, is delayed in the majority of instances. The epiphyses are enlarged, the extremities bowed and stunted and there may be prominent swellings or tubercles on the long bones. All parts of the body are uniformly affected, as a rule, though there may be special shortening of the lower limbs and permanent dwarfism in later life as a result thereof. The face appears to be small and ill-developed in comparison with the head, the shape of which latter is square and box-like, with prominent Parrot's nodes and sometimes patches of craniotabes and a bald area on the occipital bone covering. The hand is normal in size, but may show Koplik's bowing of the fingers, or the beading of Neurath, and a likeness to a ninepin may be created by the thickening of the terminal phalanges. The anterior fontanelle is widely patent and late in closing. There is profuse sweating of the head and neck at night, as well as restlessness and other nervous disorders in certain instances. The mental faculties are apt to be late in their development, but, though the child may be late in talking, it may be quite of the average intelligence in later life. The skin is not always affected; there may be much deposition of fat or eruptions from excessive perspiration or gastrointestinal complications. There may be curvature of the spine, which is usually of the nature of kyphosis or a long hump. In most cases the temperature is normal; but in the presence of complications there may be pyrexia, though a subnormal body-heat may be seen late in the disease. The patient is late in making efforts at standing or walking and the entire muscular system seems to be very weak. These principal symptoms are of great importance as regards -

DIFFERENTIAL DIAGNOSIS

Achondroplasia

This condition may be defined as a lack of development of cartilage, achondroplasy being Parrot's designation for a form of foetal rickets in which the limbs are short, the curves of the bones exaggerated, with an absence of the proliferating zone of cartilage at the junction of the epiphyses; the children are generally still-born and the condition is not unlike foetal cretinism. The trunk is normal, but of a size more suited to an individual of larger stature; the limbs are exclusively affected by dwarfism and the shortness of the arms and legs is particularly striking in comparison with the rest of the body which is of normal length. The upper limbs appear to have their attachments at a level more posterior than usual and seem to hang back considerably, there being a tendency to hold the arms away from the body. There is no thickening of the epiphyseal cartilages. The hand is small, square and reduced in all its dimensions; the fingers are almost of equal length; the last phalanges of the middle and index fingers diverge and constitute the characteristic "trident hand". The face is not relatively small and the bridge of the nose is bulbous; the features are large and dentition is of normal evolution; the nostrils are gaping and the end of the nose is bulbous also. The head is of large size and,

in view of the shortness of the limbs, gives the individual a peculiar appearance. The shape of the head is not unlike that of hydrocephalus, being globular, with marked frontal and parietal bosses. The closure of the anterior fontanelle is delayed. Mental impairment may not be very marked, though the patient is apt to be somewhat childish, with the stability of the nervous system easily unbalanced. Taylor (Nerv. Dis. of Child., p. 343) says that the usual standard of intelligence of these patients is low and justifies the opinion that most of them are weakminded; whereas equally competent observers affirm that an actual sharpness of intelligence may be encountered. The temperature usually remains normal and no marked anaemia is found, as a rule. There are no dermic anomalies, though there may be a redundancy of tissue productive of folds about the legs. The chest is usually well formed and of normal dimensions; there may be some beading of the ribs from periosteal overgrowth, or affection of the periosteum may lead to displacement of the cartilage. There is marked lordosis of the spine in the lumbar region, together with a tilting upwards and backwards of the pelvis. There may be retardation of the art of walking; in general, the development of the muscular system is not unsatisfactory. Contrasting these facts with the cardinal symptoms of rickets above enumerated no diagnostic difficulty should be experienced.

Cretinism

This is an endemic (sometimes sporadic) disease characterised particularly by goitre and a condition of physical, physiologic and mental degeneracy and non-development, its subjects seldom reaching five feet in height; The sporadic is the congenital form of myxoedema characterised by the absence of the thyroid gland, diminutiveness in size, thickness of neck, shortness of arms and legs, prominence of abdomen, large size of face, thickness of lips, large protuberant tongue, imbecility and idiocy, Anaemia is usually present and takes on the chlorotic type of blood disorder with diminution in the percentage of haemoglobin, the patient being of yellowish-green complexion. There is generally obstinate constipation, dating from birth, due to infiltration of the coats of the bowels and resisting all form of treatment except the administration of thyroid gland. The limbs are more affected than any other part of the body; they are shorter than normal and there may be slight curvature of the bones. Walking is late and coordination poor, though there is no definite paralysis, but enfeeblement of the muscles. The general proportions are of infantile type. The hand is flat and spade-like, usually cold and blue, with short and stumpy fingers and a puffy and oedematous skin. The head is of small size and the hair dry, scanty and short, the scalp not infrequently being of unclean appearance. The anterior fontanelle is late in closing. The bridge of the nose is depressed, the forehead is low, the eyebrows are scanty, the palpebral aperture is narrow and immobile, the lips are thick and the jaw is projecting. The mouth is usually open and from it protudes a fleshy tongue; The teeth are late in making their appearance and liable to early decay. The thyroid gland is small or absent in the sporadic form, usually enlarged in the endemic. The patients exhibit some variety as regards mental faculties, sometimes being ~~passive~~, placid and goodnatured, but more often restless, destructive and vicious, the mental condition being, as a rule, one of considerable backwardness. Unless under thyroid treatment the cretin remains little better than an idiot and, even under the most favourable conditions, he scarcely ever attains a normal standard of intelligence. The skin is harsh and dry and, unlike the rachitic, the cretin never suffers from perspiration; it shows the characteristic infiltration of myxoedema, is swollen, dry and scaly and there are often fatty pads in the supraclavicular regions; as in achondroplasia, there may be a redundancy of the tissues about the legs, causing folds in the skin which seem too large for the bones it covers. Not infrequently there is kyphosis of the vertebral column, together with muscular weakness and laxity of the articulations. The voice has a peculiar nasal tone, being harsh and croaking. There is great sensitiveness to cold, the temperature being always subnormal, though amenable to the specific treatment. As further aids to diagnosis we have the history of the case and the therapeutic test mentioned above.

Dentition

The presence of nervous irritation from teething is sometimes suggested by the ~~not~~ uncommon association of rickets with convulsions and nocturnal restlessness, as well as the other neuroses named. Tetany and laryngismus stridulus invariably suggest rickets and are never referable to cutting the teeth; we have also in rickets the characteristic shape of the head, the contracted chest and the osseous affections of the extremities, so that the diagnosis should not be long in doubt.

Epilepsy

There are cases in which the convulsive seizures of rickets may be erroneously assigned to the above affection; but the distinction should be effected by the history of the case and the order and character of the spasms.

Fracture

The roughness and irregularity of the surface of the bone consequent upon greenstick fracture may excite a suspicion of rickets; but confusion should be obviated by recognition of, in rickets, the thickening of the epiphyses and the presence of ~~sush~~ other symptoms as beading of the ribs and sweating of the head, as well as a careful study of the history and circumstances of the case.

Hip Dislocation

The presence of rickets may sometimes be simulated by congenital dislocation of the hip, as the child is usually in the latter knock-kneed and walks with the toes turned inwards. But in hip dislocation of congenital origin the head of the femur is found to be lying ~~outside~~ the acetabulum in the iliac fossa and the great trochanter is situated at different or abnormal levels; further, the latter can usually be drawn upwards and downwards without painful sensations, which, obviously, would be impossible in the case of rachitic deformity of the thigh-bone. In all cases of doubt careful measurements should be taken of the affected limb and the patient submitted to skiagraphic examination.

Hydrocephalus

The presence of chronic hydrocephalus is sometimes suggested by the large size of the rachitic head; but the square one of the ~~former~~ affection should not be confounded with the globular one of the latter, which also presents a greater widening of the sutures and fontanelles and shows a greater disproportion between the head and the face; there is no sensation of thickening of the bones of the vault of the skull on palpation and, in contradistinction to the depressed anterior fontanelle of rickets, that opening is more patent ~~than~~ the case of accumulation of liquid in the head. In those cases in which the malady commences after ~~fontanelle~~ closure it may be impossible to distinguish the two conditions, anything like certainty of distinction only being achieved when there occur signs of pressure from increased tension of fluid in the cerebral ventricles. When the two diseases co-exist the diagnosis is also far from easy; but hydrocephalus may be excluded so long as there is depression of the anterior fontanelle observed; further assistance may be derived from the presence of rickety changes in other parts of the body and, finally, the diagnosis should be accomplished automatically by the observation of progressive enlargement of the head in hydrocephalus.

Marasmus

The above and various disease cachexias are apt to occasion some diagnostic difficulty in the case of the superficial observer. There may be some uncertainty of recognition, for instance, if, during the evolution of rickets, there has been prolonged and acute gastrointestinal catarrh, with the usual severe wasting - this being particularly true when it is impossible to detect enlargement of the epiphyses, beading of the ribs or osseous deformities in their incipience. But, in the case of marasmus, a previously healthy infant commences to show progressive loss of flesh and slowly wastes away. The subcutaneous fat disappears, the skin is dry and inelastic and the face is small and wizened like an aged individual. The rectal temperature is normal, the bowels are irregular, the stomach is dilated, the appetite is enormous and yet starvation of the tissues is observed. The nervous symptoms are twitching of the face, rolling of the eyeballs, licking at the bedclothes and convulsive attacks. Sweating, if present, is general all over the body, but not

confined, as in rickets, to the head and neck. In the final stages of the disease there may be oedema of the face and extremities. It is important, if possible, to avoid confusion of the affection with rickets, as the marasmic child is able to digest only small amounts of fat and, contrary to the rachitic, requires a fat-poor diet with a gradual, but slow, increase in the percentage of proteid, particularly the soluble albumins. In all instances the history and circumstances of the case will call for very careful study and it is therefore desirable to make the diagnosis provisional at first.

Mongolism

This is a congenital affection of very slow evolution with anaemia, the prominence of which is intensified by the occurrence of scarlet patches on the cheek, giving the impression of the application of rouge. Examination of the blood shows a marked diminution in the haemoglobin percentage. The long bones are shorter than normal and there is considerable laxity of the joints, which are unusually loose and mobile, owing to the slackness of the ligaments and increased tone of the muscles acting thereon. The subjects of this disease are small at birth, but they are not invariably the subject of constipation thereafter. The patients are usually smiling and good-tempered and make strange grimaces; the smiling face of the Mongolian idiot seems to suggest the possession of some secret source of joy, in marked contrast, indeed, to the sad countenance of the cretin suggestive of the harbouring of a secret sorrow. If the child attempts to smile the peculiar wrinkling of the skin of the face makes the expression more horrid than pleasing. There is a great tendency to the deposition of fat about the period of puberty. The head is brachycephalic, small and short at the base, the antero-posterior and lateral diameters being almost equal. The occiput is flattened and there is a falling-in at the temples. All the fontanelles and sutures remain open longer than normal. The bridge of the nose is depressed and the aperture of the eyelids is a long narrow oblique slit as seen in Chinese natives. The mouth is small and usually kept open; the tongue is large and protruding and not infrequently the subject of fissures. The teeth are late in making their appearance. The high arched palate is a striking feature of the disease; the nasopharynx is small and the child generally snores loudly when asleep. The hand is small and tapered; the second phalanx is small and there is a peculiar crooking inwards of the little finger. There is delay in acquiring the art of walking and, in the absence of proper management of the limbs, lack of power of co-ordination is observed. There is a certain amount of enfeeblement of the mental faculties in these cases; though the patient is lively, it is apt to be an imbecile or an idiot and, in contrast to what obtains in the cretin, the administration of thyroid gland preparations does no good; the general condition is one of placid backwardness, the patient being quiet, not addicted to crying and seldom vicious. The temperature is subnormal, though this feature is not so pronounced and persistent as in cretinism. These individuals are not infrequently born with some disease of the heart, a club-foot or an imperforate anus. There is usually a normal cutaneous surface, no myxoedematous anomaly being observed. Some of the cases develop squint or have continuous nystagmic rolling movements of the eyeballs. Bearing these facts in mind, no diagnostic confusion should be experienced.

Osteomalacia

This affection, being one of adult life, progressive and of peculiar etiology, should not be mistaken for rickets. By chance, however, it may be seen in children in the form of considerable softening and bending of the bones, but without the enlarged epiphyses, rachitic head and other features of the essentially juvenile disease. Some of these cases, it may be, are probably varieties or relations of the adult malady above named, though in certain instances a condition like the puppy experimental osteoporosis may be seen in very young persons. A careful study of the case in all its bearings, however, and the course of evolution of the same should eliminate all such apparently confusing conditions from the diagnosis.

Osteomyelitis

It sometimes happens that rickets affects only one bone and then syphilitic inflammation of bone or periosteum may be

simulated. But acute septic osteomyelitis generally commences at the end of the diaphysis and the rise of temperature, suppuration and other inflammatory signs will soon give a clue to the nature of the existing affection. When the tibia is attacked, a congenital bone syphilis may be suggested. But the syphilitic tibia is not curved, but swollen, nodulated and deformed by gummatous or osseous depositions; whereas in rickets there is actual curvature of the bone, particularly in its lower third, a sabre-like structure being formed.

Paralyses

The paralyses seen after an attack of diphtheria is sometimes not unlikely to lead to an erroneous diagnosis of rickets. The former may occur in cases of a mild type when the throat symptoms have been overlooked or were too trivial to call for examination and treatment. But, in view of the existing laryngeal or palatal affection, there is usually some alteration of the child's voice in the form of a sort of nasal hoarseness, there may be a toneless cough and some regurgitation of the food through the nose. Not a few of these patients develop a squint and the patellar reflex is usually lost. In rickets, however, the muscles, though weak, are not paralysed, as the child can use its limbs freely, the disinclination for walking and general debility being merely the result of muscular malnutrition. In short, the rachitic condition in question is but a pseudoparalysis from general constitutional debilitation.

Infantile paralysis, or acute anterior poliomyelitis, may, in certain instances, be set down as rickets. The former occurs during the first or second or third year of life, being seldom seen after the fourth year or in grown-up persons. The affection commences with only a very trivial amount of temperature rise, which may escape attention; there also may be convulsions, vague pains, arthralgia and formication. Then there occur inertia and a general paralysis, which later gradually disappears, to fix itself upon certain muscles. Next there develops a flaccid paralysis of one leg or arm, with abolition of the reflexes, preservation of sensation and vesical and rectal functions, - sphincters being unaffected, - without eschars or trophic disturbances. The lower limbs are more often paralysed than the upper; rarely do hemiplegia or crossed paralysis occur. A single muscle or single group of muscles may be affected - e.g., anterior muscles of the leg or muscles of the calf. This limitation of action is noticed only after the lapse of a few weeks, the child limping, dragging the leg and refusing to walk. The joints are unaffected. At first the contractility to electricity is diminished or abolished and later the paralysed limbs may become cold, purple, livid and moist. Sometimes ulcers or eschars accompany this local asphyxia. Atrophy is not seen before a month or sometimes six months; the muscles are soft, flaccid and emaciated, the limb is thinner than its fellow. The atrophy of certain muscles and the badly balanced action of their antagonists result in bad positions, such as clubfoot. There may be abnormal mobility of the joints, which may take on the flail characters. To make up for these defective attitudes of the extremities, the spinal column may be in lordosis or kyphosis. The presence of the reaction of degeneration in the affected muscles and the general features of the case should suffice to make a diagnosis from rickets easy.

Paraplegia should give rise to no difficulty in distinction, as in rickets the power of movement of the legs is present, there are no signs of degeneration and no girdle pains.

The possibility of the presence of pseudohypertrophic paralysis may be suggested in certain rickety cases with marked lordosis. The affection commences with a lack of power in the lower extremities, the child becoming easily tired, spreading his feet far apart, walking badly and leading his body back after the style of lordosis. Later the pseudohypertrophy appears, the calves enlarge and become as hard as wood, the thighs, buttocks, shoulders and arms then become affected; there may be atrophy of the upper extremities at the same time as hypertrophy of the calves. The disease may extend to the face and tongue; it is not infrequently limited to the calves and always leaves the anteroexternal muscles of the leg, the pectoral, the rhomboid and the cervical muscles unaffected. The hypertrophy reaches its maximum in about a year and a half or two years, then the paralysis progresses, prevents walking and extends to the upper extremities. If the child lies down, he can only rise by placing his hands on

the thighs and clambering up with difficulty. In the recumbent position the lordosis disappears. When erect the child has to balance himself to maintain his equilibrium, swings himself about and walks upon his toes; flat-foot or other like deformity appears. There is diminution of electrical contractility, but no loss of sensation and the tendency of the disease is towards ultimate fatality. Unless the muscles are only slightly prominent or only slightly so, there is little difficulty in diagnosing the condition from rickets, which, however, has the usual special signs.

Pleurisy

This affection in certain cases has contingent anomalies somewhat suggestive of incipient rickets; but its recognition is effected by the history of the case and the absence of the classical features of the the bone disease.

Pott's Disease

Caries of the spine is productive of a humpback condition which is apt to be mistaken for that seen in rickets. But in the latter affection there is not the angular gibbosity of the tubercular disease of the vertebrae, it being rather a general curving of the whole of the dorsal spine, with perhaps some lateral curvature. In mild or moderately severe cases of rickets this back-bending disappears when the patient is made to lie on his face and traction is made gently on the lower extremities, and the same also occurs when he is lifted up and supported by the arms; in very severe cases of a chronic character, however, this fact may not obtain. Caries of the spinal column from tuberculosis is not often seen under the age of eighteen months; whereas rickets is often seen below that age. There is great local pain in Pott's disease and a rigidity which is at first voluntary, but afterwards enforced by ankylosis; further, there may actually be paraplegia from implication of the spinal cord. The ribs attached to the tuberculous vertebrae are carried backwards; they lose their ordinary curvature and pigeon-breast results. The history of the case and the absence of rachitic indications are of great diagnostic utility; this is also true as regards cervical and lumbar localisations, while the occurrence of abscesses in various positions must also be remembered. It would seem that a lateral curvature of the spinal column is nearly always rachitic, though it may also arise from congenital malformation of the vertebrae, static scoliosis, shortness of one lower extremity, dislocation of the hip, contractures of the knee or hip, knock-knee, and various paralyses producing unequal muscular action; it is likewise seen in hysteria, but suddenly vanishes under general anaesthesia; scoliosis also occurs in girls about puberty, from pleurisy and empyema under suggestive circumstances, and numerous miscellaneous conditions, such as congenital elevation of the scapula, with attenuation of the overlying muscles, unequal focal length of the two eyes giving rise to a twisting of the body to the weaker side, nasal obstruction and wry-neck. Finally, it must be borne in mind that the two conditions may be present at the same time, so that frequent careful examinations are required before a definite conclusion can be formed.

Scorbutus

The presence of this disease may be suggested by the occurrence of a general enlargement of the bone in a chronic osteomyelitis so occasioned, when the pain, tenderness and the constitutional disturbance are not considered conclusive. In general, infantile scurvy makes its appearance about the same age as rickets, viz., between the sixth and the fifteenth months. In the early stage of the former, when there is malaise, fretfulness, anaemia and malnutrition, it may be impossible to distinguish the disease from the latter; but pain in the limbs usually appears early and suggests the real nature of the malady. The tenderness is chiefly in the shafts of the bones, the limbs lie motionless and there is dread of movement or of being handled finding expression in agonising cries. In the later stages of scurvy the haemorrhagic symptoms are very suggestive and distinctive, such as the purple bleeding gums, - particularly noticeable around a newly-cut tooth, - the subperiosteal haemorrhages, the puffiness and ecchymoses of the eyelids and the passage of blood in the urine, all of which symptoms are never observed in pure rickets; further, the prompt recovery of the child under antiscorbutic treatment, which in no such way influences rickets, should clinch the diagnosis in ordinary cases and also when the two affections are present at the same time.

Syphilis

I have already pointed out that a definite connection between the above and rickets has from time to time been alleged, though disproved. In dealing with a suspected case of syphilitic cachexia careful examination should be made for the presence of the stigmata of the disease, such as chronic snuffles, desquamation of the skin, cicatrices or mucous patches. Syphilitic affections usually affect the infant in the early months of life, not later than the third month, i. e., before rickets makes its appearance, and teething is usually early. In further favour of syphilis we have the history of the parental attack and possibly also one of abortions or miscarriages in the case of the mother subsequently. In general, the diagnosis between the two affections should give rise to no difficulty; but, if there be present marked craniotabes, Parrot's nodes and enlarged spleen in combination with typical rachitic anomalies, it may be concluded that both are present at the same time. In the later months of infancy rickets is more apparent and the symptoms of syphilis gradually take their departure. After six months the detection of syphilis becomes more difficult. Syphilis is a bone producer, rickets forms cartilage. Syphilis gives rise to more extensive and diffuse thickening of the of the lower end of the diaphyses, also nodes and gummata. In general, the lesions of congenital syphilis appear much earlier than those of rickets and the later bony changes are not confined to the epiphyseal junctions, as in rickets. Carpenter (Syphilis in Children, p. 73) says that by twelve months of age the syphilitic wave is at its height and may continue feebly for another year; on the other hand, at six months of age the rickety wave is just beginning to make headway and between the first and second years should attain its maximum. Congenital syphilis usually puts in appearance about the third week, when the nails may be shed and a coppery rash develop upon the cutaneous surface of the body. The teeth often appear before the normal dentition period, but are prone to early decay, this being particularly noticeable as regards the upper incisors. There may also be snuffles, syphiloderma and subcutaneous gummata. The osseous lesions of syphilis are destructive and lead to separation of the epiphyses and shaft, as well as the formation of abscesses. The early form of osseous lesion is acute epiphysitis, with inflammation of the shaft and soft parts secondarily. The epiphyseal enlargement of the wrist in syphilis attacks usually only one bone, thus markedly differing from rickets. Beading of the ribs is seen in both rickets and syphilis; an enlarged spleen and craniotabes in a rachitic child should always arouse a suspicion that syphilis is present also. It is sometimes impossible to differentiate the craniotabes of rickets from that of syphilis; each case will therefore have to be estimated by the presence or absence of other symptoms and further aid may be obtained from the family history forthcoming. Though craniotabes is one of the earliest signs of rickets, it is seldom seen before the third month and rarely also after the second year of the child's age. Still says that it occurs in rickets apart from syphilis, but that the condition is aggravated when the latter affection is present. Examination of the blood for the specific parasite should, of course, be made in all doubtful instances. If the affection be limited to the tibia, it may be regarded as a lesion of syphilis, in the late hereditary form of which a chronic osteoperiostitis sometimes occurs in that bone and produces the syphilitic tibia of Lannelongue. In the latter condition the bone is not curved, but swollen sometimes to twice its natural size and there is a forward curvation of the bone like a cavalry sabre, with sometimes a bending inwards at its lower third. Swelling near the epiphyseal junction may give rise to interference with the function of the adjacent articulation and there may also be ulcers and scars along the affected bone, with perhaps nodes here and there breaking down and culminating in a necrotic process. To distinguish the rachitic curve from the syphilitic one it is useful to recall the fact that rickets occurs usually under three years, whereas syphilis may be seen up to a much later age. The history differs in the two affections; in the one we have signs of rickets, in the other a history of syphilis and its classical expressions. In rickets the direction of the curve is anteroexternal or antero-internal; whereas in syphilis it is anterior only. In rickets the curve is generally located in the upper or lower third; in

syphilis it is in the middle third of the shaft. The crest of the tibia is sharp; in syphilis it is smooth and rounded. The surface of the bone in the case of rickets is flat or concave; in syphilis there is a convexity.

Tuberculosis

The tubercle bacillus may attack the infant and give rise to affections with a resemblance to those of rickets, though it used to be supposed that infantile tuberculosis is of rare occurrence. The presence of enlarged glands in the neck or under the manubrium sterni, together with bronchitis or bronchopneumonia, in a rickety child may give rise to considerable confusion and it may be almost impossible to differentiate abdominal tuberculosis from rachitic bowel complaints; further, during the time of cutting the temporary teeth, rickets often passes unnoticed, such expressions of the disease as diarrhoea, gastric catarrh, nocturnal discomfort, head-banging and convulsions being erroneously assigned to dentition. Such cases call for very careful study and analysis, with bacteriological examinations and well-directed observations of the clinical evolution of the disease. *Tabes mesenterica* is a very common disease in poor-class practices and may be suggested in rickety cases by the enlarged belly and other local symptoms. But what has just been said here also applies, particularly watching of the case and its general history.

Worms

A diagnosis of the above may be formed in view of the stomach disorder, the abnormal tongue and the nervous disturbances seen in certain cases of rickets; but the close study of the patient's condition and the efficacy of worm powders, &c., should lead to an accurate conclusion.

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P R O G N O S I S

It appears to be quite exceptional to have a death in a case of ordinary rickets, though the reverse obtains when there are complications or sequels. There is, however, the possibility of not being able to rear any infant and when rickets occurs this is not infrequently converted into a strong probability. The use of antirachitic remedies at an early date may cause the disease to be checked or averted without the severe osseous lesions having a chance to develop; but, if the affection secures a firm hold and has osseous and visceral anomalies, the cure will only be slowly effected and the child not be out of the wood for many months, if it actually survives. Rickets is one of the great causes of the present high infantile mortality during the first two years of life and it would appear probable that a very large number of deaths in infancy can be assigned to disorders of the gastrointestinal and respiratory systems primarily rachitic. In general, prompt improvement is seen under proper dieting and management; the enlarged epiphyses undergo diminution, the deformities of the chest subside and other osseous lesions tend to take their departure, though not infrequently some slight deformity, such as bowlegs, remains and the individual becomes stunted in stature. A speedy recognition of the suggestive signs of early rickets is of great importance, as mild forms of the disease can forthwith be arrested before the development of the serious osseous disease. But if the malady is allowed to secure a firm hold upon the system and to attack the bones or become complicated with visceral or respiratory anomalies, the cure will be tardy indeed. The greater the severity and extent of the bone lesions, the more will be the disturbance of the process of ossification; and the more profound the interference with and undermining of the general health and nutrition, the longer will the child take to get well again. The outlook will be more favourable of the affection or deformity is limited to one limb than when the general system becomes rachitic, though the intensity and depth of the lesions are better guides to the future than the extent and distribution of the malady. Severity of osseous affection can be interpreted as indicative of the fact that the ailment has been in progress for several months and will be slow in cure; while if there are marked permanent deformities, surgical interference will be necessary and the usual operation risks obtain. Even when a cure is effected and the disease has apparently left the system for years, one should bear in mind the possibility of the occurrence of the so-called late rickets at or about the time of puberty, when such suggestive signs as muscular fatigue and nervous inertia should be given due attention. When the female child grows up into womanhood and becomes pregnant she and her offspring may be in great danger from the rachitic pelvis already described. In all cases of infantile rickets the prognosis will be influenced by the course and severity of the disease, which is usually insidious in onset and of slow and chronic course perhaps covering some years. In the rare cases of acute rickets, however, the malady invades suddenly and lasts for only a few weeks; further, it may give rise to so profound a cachexia that the child succumbs to its **vigilance** and collapses into death. The earlier the age at which the affection commences the more rapid and severe are the changes produced in the bones and the complications in such a system as the respiratory, interference with the patient's health and nutrition being thereby occasioned; the outlook depending on the nutritive disturbance and the complications, it is more favourable the older the sufferer, it being more favourable in the second year than earlier. The time of the year is said to affect the prognosis somewhat, the malady being worse in the winter than the summer when the child can spend a lot of time out of doors. The robustness of the child has some bearing upon it, of course, as has also the social status, a slum child being less likely to receive proper management than one in an affluent home. Rickets is responsible for a high convulsive mortality and the nervous complications, in general, add largely to the danger of the disease, as sudden death may occur during a fit or tetany, while the risk of suffocation from laryngismus stridulus is very real in not a few instances. The nervous system in these cases may be permanently weakened; while we have it on the authority of Gowers that a tenth of all cases of epilepsy have a rachitic history, 17 per cent. being Couts'

estimate. The occurrence of hydrocephalus means a bad prognosis, particularly in severe cases; even in mild ones the individual is apt to grow up neurotic, excitable and lacking in the power of concentration and self-control, it bearing badly the strain and competition of life, suffering from headaches, disturbed sleep, nightmares and enfeeblement of the mind. If there is much deformity of the thorax and pronounced softening of the ribs, the outlook is not entirely favourable; for, owing to the interference with respiration in these cases, there is deficient aeration of the blood, with resulting deficiency of oxidation and removal of metabolic waste materials. Such patients not infrequently suffer from catarrhs and, no matter how carefully they be protected against chills, such affections of the mucous membranes will arise, owing to their extreme sensitiveness to changes of temperature; such catarrhs would perhaps have very little effect upon healthy infants, but are very dangerous in the case of the rachitic and often culminate in bronchitic or bronchopneumonia, which are life-threatening in the already debilitated child, partly because the contracted chest interferes with the proper expansion of the lungs and partly because the thoracic and abdominal muscles are weak and unable to deal with the extra strain thrown upon them by the acceleration of the respiration, the coughing and the efforts of the patient to empty its bronchial tubes and lungs of mucous accumulations. Death is common in such cases; but even when they survive, the proper growth and development of the child is apt to be interfered with by the imperfect expansion of the lungs and the poor quality of the blood. It is the above-mentioned tendency to catarrh which makes measles and whooping-cough more to be feared than perhaps any other disease. Pertussis throws an increased strain upon the narrow and contracted chest, with little elasticity and only feeble power of recoil, as well as imperfectly expanded pulmonary tissue; so that the patient not infrequently succumbs to spasm, suffocation or sheer exhaustion. Infectious diseases in general are very dangerous when engrafted upon rickets, scarlatina, for instance, carrying off many cases through its increased severity. Catarrh of the stomach and intestines is also of unfavourable prognosis, as some intercurrent disease may prove fatal; any prolonged disturbance of this region is apt to lead to general malnutrition, which in itself is unfavourable. Even mild indigestion is apt to become very chronic and resistant to ordinary remedies. In general the gastrointestinal complications are governed by the severity of rickets; if the case is mild, the digestive disorders are slight; but if the rickets is severe, the various acute and dangerous catarrhs may ensue or perhaps severe inflammations. The bad effect of rickets upon the digestion may be apparent for many years, as they sometimes outlast the cure of the disease; the child may be left with a weak stomach and a tendency to chronic catarrh thereof and of the bowels, while it may become the subject of mild liver affections, gastric dilatation or obstinate constipation, perhaps hernias also. As a rule a properly treated case of rickets progresses satisfactorily towards recovery; otherwise the deformities may become permanent and partially or totally disable the individual from useful and remunerative employment or render him a subject of aversion from hideous anomalies in the bones.

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 T R E A T M E N T

I n t r o d u c t i o n

The proper control and treatment of the rachitic dyscrasia comprises not only the actual cure of the affection, but also has to take into consideration numerous difficult problems of social and even Imperial importance. Thoroughness of management will therefore involve a minute investigation of the causes which are operating, such as bad feeding, starvation, poverty and ignorance generally. It seems scandalous, indeed, that in this so-called enlightened age practically a half of the children one has to treat show signs of rickets in some form, past or present. It is, however, a matter of congratulation that the excessive infantile mortality has, particularly of late, attracted considerable attention, the whole question being now under careful consideration with a view to correction of the awful social defects. One must always bear in mind that, though many infants die in the early months of life, many more struggle on ill-fed and ailing, ultimately growing up full of the seeds of disease for future malign operations. They may escape the dangers of malnutrition or acute diseases of the great vital tracts, but their constitutions have been debilitated by rachitic disease, so that they develop into stunted, deformed and crippled individuals, prejudiced in both mind and body adversely. The males of this class have their wage-earning capacity diminished and the females have great perils to face when they become pregnant. The question of rickets is just as important as that of alcohol and it is high time that certain facts should receive due emphasis in public speech and press. Too much cannot be done in the way of efficient legislation for safeguarding the infant during its early days; on such lines more stringent measures should be enforced to control the sale of patent foods, because it is recognised that these are just as deadly as alcohol and other poisons to the growing child. The dangers arising from defective drainage in the home and messuage, as well as the unfavourable influence of dirt and defective hygienic appointments, are tolerably well dealt with by municipal organisations; still, an infant is allowed to be slowly and surely poisoned by bad food and ignorant management - a conditions of things as dangerous to its vitality and well-being as the chronic inhalation of sewer-gas and the like. We shall presently see that another important step in the thorough eradication of rickets is the careful and correct training of the parents, especially the mothers, in the up-bringing of children - the object of this being that the little ones shall grow up strong and sturdy scions of Empire, not sickly, neuritic and ever delicate cripples or weak-minded degenerates. It would not be proper to dissociate rickets from the various factors operating to produce the heavy annual mortality; so that it surely reasonable to speculate that, if some of the various causes responsible for this heavy infantile toll of life are eliminated, rickets will gradually disappear at the same time and allow a hardier race of children to be reared by intelligent parents in healthier and happier homes. So it will come that there will be less and less need for drugs; there will not be such a feverish search for a specific remedy for this disease and the medicinal agents about to be described will become more or less of historical curiosities. Better feed, purer air and more sunlight are bound in time to lend material aid to the eradication of the disease, which to future generations may become actually unknown. My idea of prevention is to attend to the care of the mother during pregnancy and lactation and her child during its first two years of life, watching the latter carefully during that period of rapid growth and development, with due education towards adoption of normal and regular habits of living, so as to prepare for the multitudinous struggles of coming years. Above all, the urgent of breast-feeding must be dinned into mothers' rears; only in very

exceptional circumstances may artificial feeding be allowed.

(A) P r o p h y l a x i s

(a) PARENTAL.

1. P a t e r n a l.- In view of the well-recognised fact that prevention is better than cure in most instances, and particularly in rickets, preventive measures should be of the most thorough sort and be directed to the supervision of the father, as well as of the mother and child. The entire training of the female child should have maternity possibility in view, domestic management being assiduously instilled and an affection for, and practical interest in, children of all ages encouraged, with pride in the home appointments and all things which concern, or are likely to concern, future years. The influence of the father upon his offspring may be very considerable; but, in his endeavour to safeguard the unborn babe from rickets, the family doctor can seldom do much in the way of bringing influence to bear upon the husband, unless in quite exceptional circumstances. If, however, one or more of the children show signs of rickets, it might be advisable to inquire into the family history of both parents, in order to find out if they themselves had been rachitic in infancy, in any such positive case pointing out to them the necessity of special precautions for the future, for the reason that the later members of a family may become tainted with the disease. The father must be made aware of his responsibilities as regards provision of the necessaries of the home, it standing to reason that his wife cannot prove an efficient mother and practical breast-nurse if she has to neglect her obvious and usual maternal duty to go out and work. Such a fact cannot be disputed, for many an infant is deprived of its natural feed, or only receives it at irregular and uncertain intervals, because its mother is obliged to toil all day as cleaner or hawker and the like to assist the finances of the home or keep a lazy, drunken man. To relieve poverty and its concomitant evils is ever a noble undertaking; but it must be remembered that indiscriminate philanthropy may actually intensify what it is intended to allay or pander to actual vice or other wicked things. So it comes that, although little can be done in relation to the father's health as regards the coming child, we may do much at school or in later life to educate him in the responsibilities of paternity; as far as possible, then, he should be taught to act the manly part, independent of philanthropy or the cadging of impecuniosity, borrowing or hand-to-mouth or intemperate existence, the real danger in the manufacture of paupers being not the waste of resources, but the unmaking of men. He should be made to realise that his wife's proper place is at home attending to the children; on no account must he allow her to return to work in the factory or elsewhere if this will interfere with the proper and natural rearing of her child. This is obvious; for, if she is away all day, it is impossible to keep the house bright, clean and attractive, so that the husband, through lack of comfort and the unpleasing sight of dirty and neglected children, wanders away to the genial warmth of the tavern or the cinema halls. There are far too many unemployed men about nowadays for there to be any necessity for a nursing woman to engage in work; but, alas! the tendency of the present age is to rely more and more upon the earnings of married women for the support of the home, which fact must in time affect the moral fibre of the husband, making him anything but a suitable sire of healthy children. If in such cases the charitable endeavour to effect a remedy by taking the burden from the parents by providing free meals, much will be done towards effacement of the virtues of home life, the parents then evading responsibilities and their children in time, from bad example and absence of proper stimulus of independence, becoming a burden upon the rates. This is a subject upon which much might be written, but space forbids; let it suffice, then, to further emphasise the fact that such home-life destructive evils must injuriously affect the early nutrition of the infant, prevent the all-important performance of breast-feeding and pave the way for rachitic disease. The causes of the latter cannot so much be traced to poverty as

ignorance, neglect and over-indulgence in improper food: so that the remedy lies in the better instruction of women in the performance of maternal duties, with legal action against glaring paternal neglect. It is pleasing to know that most married men have a natural affection for their offspring, which same should be encouraged by developing their self-respect and sense of family responsibility. Domesticity must be encouraged, which can only be done effectively by increasing the wages of the father, by preventing as far as possible the labour of married women and by improving and brightening the conditions of home life. It is easy to imagine that the children of a tainted parental stock must be physically and morally unhealthy and, living in unwholesome environment, must fall victims to diseases of which rickets is not the least important. The father should be made to understand, therefore, that there are great advantages to posterity in good and well-ventilated homes, and so forth, which same are largely rendered imoperative by deprivation of mother's milk, lack of maternal care and artificial foods. The dangers of alcoholism must also be pointed out, pernicious liquids of this kind being not infrequently consumed from deficiency of breast milk; this is no mere surmise, for statistics from various quarters have conclusively shown that women with poorly acting mammary glands are usually daughters of alcoholics, particularly of the third generation. Further, it has been similarly shown that when the mother was able to nurse her child and her daughter could not or only partly could, in some 80 per cent. of the cases this inherited abnormality is due to immoderate use of alcohol on the part of the father; it is also interesting to note the statistical assertion that when the father is a drunkard the daughter loses the power of suckling her child and that this power is lost for future generations. It is for such reasons as these, then, that the father must not be regarded altogether as a negligible factor in the prevention and cure of rickets; it is true, however, that the bearing he has upon the disease in this respect is mostly indirect, his duty being to do all in his power to enable his wife to suckle her child in order to avoid the evils that arise from artificial foods.

2. **M a t e r n a l.**— Despite the fact that even the children of the poorest receive a good education, it is true that it may not always be the one best adapted for future adult requirements in the battle of life and the stress and turmoil of infant-rearing. Still, there is no denying the fact that the school-age is the period of receptivity, the obvious indication being for the reception of impressions that can be stored up and prove of value in later life. The elementary facts of cleanliness, cookery and domestic duties might well and largely replace the generally unimportant truths of history and geography, music, singing and so forth. I do not believe in trying to teach children, by elaborate courses of physiology and domestic hygiene, though the same may in certain cases do good, as the bulk of such crammed facts are soon forgotten and rendered useless or confused in adolescence. A much better plan is to give frequent demonstrations on practical matters connected with the home, training the children to use their eyes and hands in the details of housewifery. Dolls come in here; most little girls love a baby, so that lessons in the proper management of infants could be made a pleasure instead of a task as in lectures or prosy discourses on technical points. Indeed, the doll should be used far more than it is during educational years; for children play at being grown-up when they have a doll and here lies a grand opportunity of teaching them how to wash and dress it instead of allowing them to toss it about at will. What a power for good, then, the games of childhood may be! I understand that in Japan the little children are trained to carry the baby on their backs, the younger members of the family being in such ways familiarised with the nursing rôle they will probably have later to play. By the simple expedient of strapping a doll on the back in the position which should in adult married women be occupied by a living child the children are trained by dummy to handle a baby in an intelligent manner. Girls here as there should be taught how to wash the infant, to cut out patterns for clothes, how to put them on and what are the names and uses of the various materials. In time a living baby could be substituted

for the doll, with careful instruction on the arrangement and administration of the nursery, particularly as regards keeping it clean and not overlooking the smallest hygienic detail. There should be no difficulty in explaining in an understandable manner the value of milk as a food and its modification for an infant, together with the proper methods of preparing whey, gruels and nursery feeds. Due insistence should be made on the necessity for scrupulous cleanliness of the person and all utensils, as well as the virtues of method in all matters. There is also a grand chance for emphasising the dangers of artificial foods, making a sort of aphorism for memorisation of the fact that it is twice as dangerous to feed infants upon condensed milk as on that fresh from the cow, or a hundred times as dangerous to feed a child on condensed milk as upon the maternal supply. Such lessons should be short and frequent and never assume the form of sermonic lectures. Such facts must often be repeated, lest they make impression for future life. The older girls might go in for courses of lectures as devised by various education committees, lessons being set from one of either of the simple text-books now on sale. Such persons might possibly derive benefit from working now and then at a crèche under a qualified instructress and the appealing methods of the magic lantern or cinema halls might well be taken advantage of to this end. Something short and to the point might be said in the schools about the influence of heredity, taking care, of course, to avoid all mysterious scientific terms and constantly urging the fact that vicious or abusive living or habits are sometimes handed down from parent to child. Such forms of moral training should be used to stimulate school children to grow up useful members of the community, full of the commendable pride of race. They should come to realise how the race is perpetuated or altered in process of time and how the effect of environment, the modifications of organs and limbs used for special functions, or even the abuse of food and drugs or manner of living may alter types of animals or manking from good to bad. In short, the simple truths of evolution of home application could be very easily and effectively instilled. Many of these children after leaving school must earn their living in domestic service or as nurserymaids. An unfortunate infant is too frequently left to the tender mercies of callow, ignorant and incompetent girls and consequently suffer at their hands, to the extent perhaps of having intestinal or stomach disease from bad food or sweets given to coax off a cry. Indeed the servant question is ever a source of worry and vexation in the home; but, if at school the young candidate for domestic service had been trained in the elementary laws of hygiene and cooking and been duly certificated therein, there would be fewer risks to the children and greater happiness accrue. Nevertheless, in only special cases should the mother be relieved of the responsibilities contingent to the superintendence of the nursery and her child; never should the nursemaid been given full charge. A proper school training would qualify the latter for efficient work and the certificate which has earned would be suitable evidence of her trustworthiness. In service dated certificates of conduct should be given when she leaves. The young mother should learn very early the responsibility of attending to her own health, with a view to favourable influence upon her unborn child. Far too often these persons are totally uninformed and are quite ignorant of the elementary facts of maternity, having little or no idea of what to do with the child when it comes into the world. Some of them have a natural intelligence and consult the family doctor and all is well; but most of them seek advice from almost equally ignorant neighbours and friends. District visitors could do much good in such cases, as well as mothers' meetings of an educational kind. "Straight talks to mothers" might well be instituted by the local medical men in turn, with demonstrations on the living child by maternity nurses. I am not convinced that prosy public lectures for mothers are productive of lasting good; the publication of simple text-books would seem to be a more useful idea. It is very necessary to do something as regards the food of the pregnant woman. Morbid cravings must be discouraged and treated as pathological, only proper food-stuffs being allowed owing to its influence upon the unborn child. The

use of spices and condiments in excess must be avoided and the use of the nerve-poisons, tea and coffee, should be warned against, particularly amongst the poorer classes to whom they do incalculable harm. No good can be done by the administration of stout, porter or alcoholic drinks. Infants born of drunken mothers are difficult to rear. The quality of the food is of more importance than the quantity to the pregnant woman and intemperance in eating and drinking must be avoided; it seems probable that errors of diet, especially as regards excess of nitrogen, may react very powerfully for evil upon the foetus, possibly by overstimulation, or interfere with post-natal development. An excess of carbohydrate food is unnecessary and harmful, as it throws extra work upon the kidneys of the mother and does not benefit the child in her womb, the latter deriving its nourishment mainly from a small quantity of nitrogen. The pregnant woman should be discouraged from living on a chronic diet of bread and butter, cakes and tea. The working class population do not seem to consume the kind of foods supplying the most energy, they preferring strong infusions of tea, with bread and butter, when they would do much better on porridge and milk. Auto-intoxication of the mother by food may affect the child, unless the placenta forms a sufficient filter and is able to prevent the passage of toxins. The mother has also to deal with the excretory products of the foetus: hence the importance of her not having to get rid of a surplus supply of her own. Fresh air, sunlight, regular exercise, - short of actual fatigue, - daily interesting routine and cheerful companionship help to quicken the vital processes. In short, regularity of life promotes appetite and induces healthy sleep. But the routine walk should be avoided as monotonous, much more stimulation being forthcoming from the general interests of housework and ordinary daily occupations and recreations. The surroundings of the home should be bright and cheerful, all emotional and mental disturbances being avoided as much as possible, as likely to have an unfavourable effect upon the supply of milk, which is usually poor in quality and scanty in quantity in nervous or excitable women. An outdoor life is the ideal one; an approximation to such should be encouraged. There is much to be said in favour of the German system of maternity funds, provided by employer and employed, and the French one which endeavours to keep the pregnant women in a home some week before confinement and drafts her into a maternity hospital afterwards, with practical and financial supervision during the first year of the infant's life. These systems are regularly organised and competent; they are far superior to the promiscuous philanthropies of other lands. The evils of factory employment have already been referred to; the nation should set its face against the employment of married women in such places, as their proper sphere is in their own homes; in the case of factory women the employer should be made to provide a maternity fund and probably, if he is thus penalised, he will prefer not to employ married women - to the great advantage of the labour market and the community at large.

(b) HYGIENIC TREATMENT.

The above method of dealing with this disease is of vast importance, fresh air, warmth, sunlight, proper clothing, rest, exercise, baths, etc., having a great influence upon its prevention and cure. Given such favourable conditions as these, it seems justifiable to hope that in time the affection would be stamped out, particularly if due attention is directed towards the provision of healthy dwellings, proper clothe and breast-feeding of infants, a function so much disliked and neglected nowadays. Fresh air is of cardinal importance; during the early months of the fine part of the year the child should be taken out into the country lanes or parks as soon as it is seven days old, at first in the nurse's arms and later in its carriage. In cold or wet weather the fresh-air treatment should take the form of its being carried into another room, previously warmed and ventilated. The nursery should have a southern aspect and the windows should be freely opened. In the winter, when it appears to be dangerous to open the windows wide, it is advisable to have a board fitted under the bottom sash, so that a free current of air may enter the room between the upper ones and take on an upward warmth-receiving direction. The temperature

of the apartment should be about sixty, be large and sunny and fireplaced. Electric light is better than gas, as it consumes no air and gives off no products of combustion; gas and gas fires are to be condemned on these grounds and candles and paraffin lamps are no less objectionable. Removal from a cold and damp place to a dry and warmer climate, or to the seaside, is to be commended; the effect of light and sunshine there and elsewhere is striking sometimes. As long a time as possible should be spent at the seaside, so that the full benefit of the pure air may be obtained. Gastrointestinal cases seem to do well there, but respiratory ones do better at higher altitudes. Sunlight has a marked antirachitic effect, as may be instanced by the comparative rarity of the disease in the tropics and abundance in colder parts. The child should have a sufficient amount of sleep, for the first two or three days of its existence most of its time being spent in this way; for several weeks it should not ordinarily be awake for more than two or three hours during the twenty-four, up to six months from eighteen to sixteen hours sleep being desirable. Sleep should not be induced by such abominations as comforters and soothers, which are apt to become dirty and a prolific source of gastrointestinal infection. Rocking the cradle is not always the good thing it seems, for it tends sometimes to churn up the contents of the stomach and so give rise to various digestive troubles. The clothing of the child requires also very careful supervision. Long clothes are to be condemned, as they are heavy and confine the limbs. Shortening of these should take place after the first few weeks, but should not be carried to excess. The lower limbs should not be exposed for the purpose of hardening, which is often injurious, but so covered that, while protecting from the cold or wet, the legs shall have unrestricted movement. In this disease the general circulation is depressed and the infant not infrequently suffers from cyanosis of the limbs and coldness of the feet; the perspirations also contribute to the coldness of the body surface. The underclothing should be of soft woollen material and the limbs and neck should be properly covered. A flannel nightdress is advisable, as the child is apt to kick off its bedclothes; a flannel binder should cover the abdominal walls, atonic conditions of which it tends to correct. It supports in these cases what is usually a weakly part, it stimulates the respiratory functions and is not without effect upon the sluggish abdominal circulation. Some people hold that the belt should be worn continuously and be made of elastic, so as to act as a perpetual massage; whereas others insist that only its intermittent use for an hour or two each day. The napkins of the infant should not be applied too tightly and as soon as they are soiled they ought to be changed. Every morning the child should receive a bath of warm water and soap and at night it should be sponged with tepid water at about ninety degrees, with warm frictions afterwards. Rickety children are very susceptible to cold, which fact should always be borne in mind. Salt water baths are often of service and good results have been reported from immersions in sand. Careful massage is generally of use, if there is not much softening and tenderness of the bones. Every morning, after the bath, the child should be laid upon his face and be rubbed with the open hand along its spine, perhaps with a little olive oil, for ten minutes or so with repetition at night. Massage of the belly is generally very beneficial, as it stimulates the muscles of the region, as well as the intestinal musculature, thereby relieving the flatulence or constipation; it is also effective upon any congestive conditions present and the respiratory function can also be stimulated in this way. Some good can be done in certain cases with a mild continuous electric current to the skin, aided by an electric bath. The Roentgen rays have also been tried and phototherapeutic and sunbaths are said to do good.

Medical Treatment

(a) GENERAL TREATMENT

In dealing with the question of the drug treatment of this disease it is presumed that the case is being fed on the appropriate diet presently to be outlined and that the hygienic and other factors have received due consideration. The main part of our endeavour must be directed towards promotion of the child's nutrition, fresh air and healthy environment; for drugs, though they afford valuable aid, should only be used as auxiliaries to the above. We shall presently see that there are various indications in the course of the disease for their employment, though it must always be understood that, despite the claims of such agents as phosphorus and cod-liver oil, no actual specific medication is known.

PHOSPHORUS. This has been much recommended, though its indiscriminate exhibition is not entirely free from risk. The preparations of the remedy are, however, not infrequently of great utility, particularly the hypophosphites and the glycerophosphates. It appears to have been first used in France in the form of a "phosphorus butter". Some of its advocates regarded it almost as a specific, especially in those cases suffering from craniotabes, it appearing to them to assist in the closure of the anterior fontanelle and ward off to some extent threatening laryngismus stridulus. It was said also to have a good effect in cases marked by severe inflammation of the epiphyses, debilitation, gastrointestinal catarrh and scorbutic manifestations. It became very much used because it tended to hasten the healing of fractured bone, even in small doses, experimentally. It has not yet lost its popularity on the Continent of Europe and elsewhere, though it appears to me that much of its efficacy is due to the cod-liver oil with which it is so often prescribed. As stated, there is, however, a certain amount of risk attached to its prescription, cases having from time to time been reported in which the child has developed icterus and died from the usual poisonous effects of the metal, with the classical autopsical findings. It should therefore be administered with great caution or, if there seems any risk, it had better be left alone. Its use might well be reserved for severe and intractable cases and it must be avoided when gastrointestinal affections complicate the case. The dose should be the very small one of one six-hundredth to one two-hundredth of a grain three times a day, given in a flavoured oil. I have never known it to do good in itself, except in craniotabetic or nervous cases and in such I have often thought I might have done as much with other agents. It is possible that it may prove effective by neutralising the toxic products of the intestinal canal responsible for the origination of the disease by infecting the osseous and nervous systems. In order that there may be uniformity of dosage, it is necessary that it be thoroughly mixed with the oily vehicle with which it is given; if the bottle be not well shaken, there is risk that the last doses thereof will contain too much of the drug and give rise to symptoms of poisoning or even death. The best way to give it is to dissolve it in ether and then in oil of almonds, with vigorous agitation, and then incorporate the whole with cod-liver oil and place in a water-bath for some minutes to get rid of the ether. Then hermetically seal the bottle and label for vigorous shaking before administration. Various compounds of phosphorus have found favour of late, such as the syrup of the hypophosphites, the glycerophosphates and lecithin. The well-known compound syrup of the phosphate of iron is useful in doses of a half to one drachm, with or without cod-liver oil, in anaemic and other cases. The glycerophosphates are also of great service sometimes in cases of rickets complicated with anaemia, neurasthenia and general malnutrition. The basic glycerophosphoric acid forms both acid and neutral salts, the acid preparations being reserved for oral administration and the neutral for hypodermic; the former drugs are soluble in water, amorphous and very stable;

they take up water very readily. Such medicaments are productive of improvement in tissue nutrition and have the advantage over other phosphorus compounds in that they are stable and easy of assimilation. Protulin and Fersan are of this class and contain albumin; the former is said to be very well borne in rickets and effective in bone disease; it may be combined with iron, bromides, etc., according to indications. Various organic preparations have been made from animal and vegetable sources. Pertossan is one of these; it is cheap and beneficial in rickety cases, is a neutral soluble compound of Phytin, is said to improve the general nutrition and growth of the patient and may be given in milk and other foods. Phytin itself is made from the seeds of plants and contains nearly 23 per cent. of phosphorus in an assimilable and non-poisonous form. It appears to be an acid calcium and magnesium double salt of a certain phosphorus principle discovered in plants, which is stored up as a phosphorus reserve in all seeds, tubers and rhizomes. On theoretical grounds at least we may regard it as readily assimilable and likely to promote the general metabolism of the patient. Lecithin is poorer in phosphorus, containing only about 4 per cent. thereof; it has no tissue-building or calcification-producing elements, is expensive and not to be recommended for routine exhibition, at least according to my experience. I have tried it in a few cases of rachitic marasmus; the results were only partially satisfactory, though while the drug was being administered some of the cases showed a temporary gain in weight, which may have been due to the rest of the treatment. The simplest and one of the best methods of giving lecithin is to give the raw yolk of a fresh egg, which contains a large proportion of the remedy in an easily assimilable form. It is a constituent of the brain and is also present in the seminal fluid, the blood, most of the vital organs and in the yolk of the egg. In the intestines it is decomposed into stearic acid, glycerophosphates and neurine. It produces sometimes marked increase in the body weight and augments the number of the corpuscular elements of the blood. I have always given it by the mouth, though some advise that it produces better results when given intramuscularly. Some firms manufacture chocolate tablets of it, each containing five-sixths of a grain, in which form it is pleasant to take and attractive to children. I have never adopted of some to give it hypodermically, as in children who are suffering from such a chronic affection as rickets and whose nerves are so readily disordered, this method does not commend itself to me for serious consideration. There is a preparation of lecithin on sale, which is known as Bioplastin, consisting of the former emulsified with three-quarters of a per cent. of sodium chloride solution. It has been given hypodermically and is said to influence the general nutrition of patients, particularly those suffering from nervous exhaustion. In Italian clinics they use intramuscular injections of thirty centigrammes of lecithin, emulsified with normal saline, every other day, and for it ascertained an average increase of ten per cent. haemoglobin and corpuscular numerical augmentation, thus removing the dyspepsia and one of the great causes responsible for the production and persistence of the rachitic disease and osseous perversions. It does not appear to produce intolerance or digestive disturbances which occur under the phosphated oil, over which it has an obvious advantage. A lecithinised cod-liver oil is also sold for tea-tablespoonful dosage; it appears to be of signal service in cases in which the cod-liver oil alone would do good.

COD-LIVER OIL.

This is more of a food than a medicine and it, or some substitute or combination should be tried in every suitable case. In general, it can be regarded as a fatty food, though it has been classed amongst the drugs by reason of the presence of certain animal extractives which are supposed to have decided therapeutic properties. It is important to administer only small doses of the oil at first, especially to young children who, if under one year of age, tolerate it badly. In most cases the initial dose should be five to twenty minims; the stools should be subjected to regular daily inspection for its presence, which, if noted, should call for its temporary discontinuance. It would appear that rickety children are only too frequently drenched

with cod-liver oil and chemical food, which they can only imperfectly assimilate: with the result that the stomach is still further disordered and progress towards recovery is indefinitely delayed. Therefore, it should never be given if signs of stomach or intestinal disturbance appear. I usually give it raw or in an emulsion with lime-water and milk-sugar, flavoured with aromatic water or wine. In some cases a single dose at bedtime causes less discomfort than frequent doses during the day. It is sometimes better borne when given with a few drops of ether or alcohol; the presence of the former occasions a stimulation of the movements of the stomach and so assists the expulsion of the oil into the duodenum; to some extent also it prevents unpleasant eructations of food. Cod-liver oil may more readily be absorbed if given in the form of a fine emulsion, which should always be freshly prepared and not prescribed as a proprietary article, even though the products of certain firms are said to be thoroughly reliable. The yolk of egg and brandy are added to some emulsions and seem to do good. The oil and extract of malt form a useful combination of ready assimilation; the result is the ingestion of both fatty and carbohydrate food, and it is sometimes also prescribed with the addition of hypophosphites of sodium, magnesium and calcium, as well as with iron, quinine or creosote. The oil itself is often made the vehicle for administration of free phosphorus. A useful preparation would contain in each drachm dose eighteen minims each of cod-liver oil, syrup of the lactophosphate of calcium and lime-water, one grain of sodium hypophosphite, seven or eight minims of mucilage, with cassia-oil flavouring. In the case of weakly infants unable to digest the oil, administration may be performed by by inunction, it being absorbed to some extent through the skin and making its detection possible in the stools. I have treated a large number of cases in this way and been well pleased with the results. The oil is of great value in the treatment of the respiratory disorders of rickets, which improve more rapidly under it than under routine cough mixtures; it also helps to increase the resisting powers of infants against disease and so minimises the risk of infection by the tubercle germ. There are many who speak highly of the combination of cod-liver oil with creosote; the latter undoubtedly prevents fermentation, increases assimilation and promotes nutrition of the tissues; with or without the oil it is a valuable remedy for the treatment of chronic intestinal catarrh common to the vast majority of rickety cases. Any of its approved preparations may be used, such as the tannate, with or without elixir of pepsin or glycerophosphate of sodium, especially in cases of summer diarrhoea; in this way one may expect stimulation of the appetite, diminished offensiveness of the stools and increase of body weight, the urine being watched and signs of renal disease contraindicating. I have known good results to accrue from a combination of cod-liver oil with lactophosphate or hypophosphite of calcium. It is a very common thing to give extract of malt and maltine alone or in combination with cod-liver oil. Their principal value lies in the presence of a diastatic ferment, so that they are readily absorbed by the stomach and intestines and constitute foods. Some of the malt preparations contain a small percentage of alcohol, which is added as a preservative and to tone down their stickiness. The exhibition of malt extract is contraindicated if there is already an excess of starch or sugar in the food; but in cases of malnutrition, especially when there is inability to digest carbohydrates, they may be of considerable value, though in the majority of instances it is advisable to add the malt extract direct to the starchy material and give it as a dextrinised gruel or food, the maltose thus formed being finally absorbed by the tissues as dextrose. It is an excellent vehicle for the administration of hypophosphites, glycerophosphates or cod-liver oil and such antiseptics as creosote when needed. It has, however, been urged that the malt extracts have been too much lauded as remedies, both by profession and public, for the reason that, unless the diastatic action is required, equally good results can be obtained from the use of honey; further, on account of the care involved in the their preparation to prevent the destruction of the diastatic ferment, the cost is out of all proportion to food value - a pound of malt extract costing perhaps three shillings, whereas the same quantity of honey would cost only a few pence and provide more sugar to the child.

ARSENIC.

According to my experience, arsenic is of particular value for the acceleration of tardy convalescence, adenopathy and toning up of the system by its well-known nervine effects. It is also useful in cases of intractable vomiting of chronic catarrh of the stomach; half a minim of the liquor, with alkali and aromatics, may be given three times a day to a child of three months. Similar small doses for age sometimes have a striking influence upon stubborn glandular enlargements. I find that children bear the drug better than adults usually. Nevertheless, its administration must be discontinued if toxic symptoms make their appearance. An arsenical water may be given if preferred and for special indications.

IRON.

Anaemic is sometimes a troublesome and chronic condition in rickets, and for its relief some preparation of iron is clearly indicated, provided the gastrointestinal tract is in a fit condition for its comfortable reception. Great care should be exercised in the selection of a safe preparation, such as the pyptonate of iron, with or without manganese, the liquor ferri dialysatus, the saccharated carbonate or the ammoniated citrate. The syrup of the iodide of iron is well tolerated, even by young infants; it may be combined with the compound syrup of the hypophosphites, and is specially indicated if there is glandular enlargement. The iodine liberated from its combination with the metal often checks fermentation in the stomach. It is usually safe to give one minim for each month of the infant's age, up to ten drops, thrice daily after meals. Some authorities, however, disapprove of the iodide of iron, holding that it is capable of producing such skin lesions as purpura, as well as of impoverishing the blood and depressing the patient. When, in addition to anaemia, the spleen is enlarged, success may be achieved by combining the saccharated carbonate, in three-grain dose, with half a grain of thyroid extract in powder thrice daily. Iron vitellin, or oviferrin, has been much advocated in certain quarters; it is said to act very quickly upon the anaemia and allay other distressing symptoms. Ovivitellin has pronounced advantages over inorganic and other ferruginous preparations; it gives rise to a rapid increase of the red blood cells and haemoglobin, improves the appetite, but fails to cause constipation or disorder the digestion. It is almost tasteless, palatable and is well borne, even in large doses, by most children. The good effect of ferruginous preparations may often be enhanced by the addition of small doses of arsenic or strychnine or other similar tonics.

QUININE.

This is a good general tonic for administration in milk; the officinal iron and quinine citrate is a useful form in dose for age.

STRYCHNINE.

This may be given as the officinal liquor or in the form of the tincture of nux vomica, gradually added to the acid or alkaline mixtures being taken for the correction of the digestive disorders and tone up the system. In the later stages some form of strychnine may enhance the slower-acting glycerophosphates.

FORMIC ACID.

The administration of formic acid and the formates is much advocated nowadays for the relief of the muscular weakness of rickets; such medicaments may be combined with the glycerophosphates and be obtained in palatable form from the drug houses. Formic acid is credited with increasing the muscular power, toning up the muscles, increasing muscular energy, abolishing the sense of fatigue, improving the appetite and generally sharpening the physical and mental powers.

COCA.

Small doses of the elixir of coca may be combined with strychnine or other tonics and advantageously given over short periods when muscular mobility and weakness of the heart cause anxiety.

ORGANOTHERAPEUTIC MEASURES.

Encouraged by success in other directions, the field of organotherapy has been extended to rickets, with a view to the relief of the profound and extensive nutritional disturbances. Various extracts of ductless glands have been tried, with a

a certain amount of success, but nothing like the discovery of a specific for the rachitic disease. In fact, I would accord such extracts only a small place in the treatment of rickets; for they are not indispensable, it being in only rare instances that they are of real service or prove more valuable than agents which have stood the test of time and universal experience. The thyroid gland, for instance has been used for the cure of this disease, but I find no reason to believe that it possesses any special value and regard its costliness as rendering its prescription amongst the poor prohibited. It seems to me that some of the cases benefited by its administration were suffering from cretinism plus rickets. In view of the theory that the virtue of cod-liver oil lay in its organic extracts, numerous observers have tried thyroid extract over long and carefully-studied periods, but without being satisfied that any signal benefit resulted. Some reserve it for advanced cases of rickets, whereas other protest against its employment in that way. The theory has been advanced that the success of phosphorus depends upon the fact that it supplies a stimulus to development which, owing to some failure in the thyroid gland, is wanting in rickets; on theoretical grounds the agent should do good, in view of the richness of thyroid in phosphates. The extract has been put to special trial in cases of anaemic and splenic enlargement, but probably the good resulting was due to the iron given at the same time. The benefits resulting from thyroid administration in cases of absence or insufficiency of this gland would seem to suggest at least a trial in rickets, as in the former instances the nutritive and general metabolic processes commence to be normally performed, the percentage of haemoglobin increases, the processes of ossification are stimulated into greater energy and there is greater power of bearing fatigue. On the other hand, if excess of thyroid is administered, there follows a train of symptoms that would have to be feared in a rachitic person, such as great nervous excitability and vasodilator troubles, together with less of weight, gastrointestinal disturbances and passage of albumin in the urine. Therefore, if it is decided to give it in rickets, the greatest caution should be exercised. As rickets is mainly a disease of infancy and early days, it seems natural that clinicians should tackle the thymus question also in the hope of finding therein an etiological and remedial agent. The thymus is a conspicuous organ in foetal life, but commences to wither up from the second to the third year. If it persists, there may be some defect in the thyroid and the presumption that the one is complementary to the other in function. The thymus contains combinations of iodine analogous to those of the thyroid and it has been suggested that, by some internal secretion, the former exercises considerable influence upon the development of bone and that the enlargement of the spleen in rickets may be a compensatory hypertrophy, the spleen replacing the function of the thymus gland. But the value of this theory is to some extent discounted by the fact that the spleen is only enlarged in five per cent. of rachitic cases. Thymus removal has been performed in the case of dogs, with the result that the animals were smaller than the rest of their breed, less intelligent, sluggish and more easily fatigued, with softer and more readily broken limbs. Under the microscope the compact layer of bone was thinner in the diaphysis and the epiphyseal line was wider and more irregular, though there was no hyperplasia of lymphoid tissue. In many cases of rickets at autopsy the thymus has been found diminished in normal size for age and observers, acting on this fact and the above theoretical considerations, have given the gland to rickety children. The preparation conveniently employed is the fresh gland minced and made into a thick soup, each patient receiving a gramme for month of age; tablets may be used if preferred. No harm seems to result, though later extensive trials must determine the efficacy of administration. Suprarenal extract has also been tried in rickets, usually in the form of tablets of the gland; I am not convinced that they are of use. A half to one minim of adrenalin solution has been advocated for the relief of rickety muscular asthenia; good results have been claimed for its administration. A favourable effect is said to be observed upon the general condition, on the profuse sweating and nervous manifestations and upon the

craniotabes. But it has practically no effect upon the enlargement of the epiphyses, the rickety rosary, or in cases of laryngismus and it does not seem to hasten the closure of the anterior fontanelle. It is also claimed that children rapidly regain the power of walking and progression, with disappearance of the softness of the thoracic bones and eruption of the teeth; the amelioration of the symptoms is most rapid during the first eight days of its exhibition; the best results are observed during the first fortnight, after this period only slow improvement being noted. So much for theory and apparently biased reports in brief; in special cases, however, I do not deny that suprarenal extract may do good, but I maintain that equally good results can be obtained by the employment of cheaper and equally reliable drugs and that, therefore, its exhibition is not justified in a practice such as mine. I observe that Klotz (Munich. med. Woch., May 21, 1912) ascribes very pleasing results to the administration of pituitary extract in rickets. He tells us that as the colouring matter of the hypophysis is particularly rich in phosphorus, the drug meets a theoretical indication in this disease. He gave with it suitable doses of calcium carbonate to provide material for the growth of bone; in a little over a month five children showed remarkable improvement in their condition and from this and his studies in osteomalacia he concludes that the true cause of both this and rickets is some disturbance of the phosphorus rather than in the calcium metabolism. The Pituitrin of Parke, Davis & Co. is a convenient preparation for the exhibition of the drug.

SALT.

I have noticed that rickety children sometimes show a peculiar fondness for common salt and there seems no reason why its free ingestion should be prohibited. A sufficiency of this condiment should be put in the broths and given with the animal foods allowed, as it contributes to the healthy performance of digestion and appears to act as a prophylactic against intestinal worms

GLYCEROPHOSPHATES.

These agents seem to have a decided effect upon the nutrition of the nervous system, so that they are indicated in all cases of nervous asthenia. Their physiological effect is to accelerate the nitrogenous metabolism, with the rapid assimilation of albuminoids and subsequent elimination of nitrogen. They are not infrequently more readily absorbed and assimilated than the hypophosphites and the phosphates, with which they may be given in combination or in a mixture containing malt extract, cod-liver oil or bone marrow, to which, if necessary, are also added quinine or iron or strychnine.

SANATOGEN.

This proprietary article contains five per cent. of sodium glycerophosphate in casein of milk. It is a white powder, which can be made into a paste and mixed with the food; in some cases it is a simple and effective method of administering a preparation of glycerophosphates. Experiment has shown that, when in cases of disease, the remedy disappears from the stomach half an hour sooner than egg albumin or casein dissolved by a sodium salt. In the case of rickets the appetite is improved, the weight is increased, any troublesome diarrhoea ameliorates, rachitic troubles are checked and the bones grow firmer and more resistant. From various quarters we hear of its giving good and speedy results in children suffering from wasting diseases, especially when associated with diarrhoea and atrophy. In some cases, however, I have known it to be badly tolerated and produce actual indigestion. In general, it acts as a tonic and stomachic, giving relief to hungered and debilitated tissues by assisting the absorption and assimilation of nutriment.

MASSAGE.

I have found massage to be a valuable remedy in the treatment of rickets, as it promotes vascular activity and stimulates the respiration and circulation, which are feeble in the course of the affection, being made worse by the flabby and toneless condition of the muscular system. The waste products in the tissues, which act as toxins, are also removed more rapidly removable and carried into the circulation to be finally dealt with by the excretory organs. When combined with baths and douches, it accustoms the skin to changes of temperature and prevents the frequent tendency to catching colds and catarrhs.

It is also of great assistance to recovery of the tone of debilitated muscles, particularly of the extremities, spine and abdomen, and it is not without good effect upon any constipation present and the activity of the cutaneous function is also thereby promoted. The effect of massage may be enhanced by the employment of stimulating liniments or alcoholic rubbings, which, by producing slight counterirritation of the skin, increase the peripheral circulation. If the digestion be enfeebled, fat may be introduced into the system by inunction and rubbing with cod-liver oil, goose-grease or bacon-fat. The same end I have found to be achieved, however, by massaging the entire body with ordinary pure olive oil; this answers most requirements and is free from the objectionable characteristics of the above-mentioned medicaments. It is sometimes advisable to dry percussion and kneading of the skin, in order to stimulate the deeper muscles.

ELECTRICITY.

A mild current of this, by reason of its stimulating properties, promotes the vascular and nervous metabolism and may therefore be expected to do good in certain cases of rickets; its use, however, calls for limitation, as many children are much frightened when it is applied to the skin. I make use of the continuous current and the electric bath, finding that these allay somewhat the perspirations, the insomnia, the general pains and the laryngeal spasms. I understand that much good has resulted from the use of the sinusoidal current in the cardiac-abdominal atony of rickets with defective compensation; little fear or discomfort will be caused. It appears to be best given in a bath or through a double-hand or foot-bath, each separate trough containing an electrode; in this way the entire current is made to pass through both the limbs and the trunk. Further, the Roentgen rays, by reason of their powerful penetrating action, are capable of producing profound disturbances of the cellular equilibrium of the blood and organs. The full extent of their value for general medicinal purposes is not fully understood, though they have been used with good effect in cases of leukaemia and splenic anaemia; from which it seems probable that, if used with discrimination, they may also prove a valuable therapeutic adjunct to the treatment of rickets, particularly during the comparatively sunless period of the year.

BATHS.

Baths act as tonics to improve the circulation and encourage growth and nutrition; if used with proper care, they have a good effect upon the nervous system. I have found them of great value in rickets, but am careful of their use when the patient is very weak or suffering from some intercurrent disease; in such cases I suspend them and order sponging of the patient limb by limb while it lies on its mother's knee, or even in its cot. If the child is inclined to blueness about the mouth, I consider it advisable to wait for the process of complete body immersion until it is older and stronger and in the case of delicate children I prefer that they be bathed with tepid water at bedtime rather than in the morning. The usual method employed in a case of rickets, ~~after~~ infancy, is to stand or sit the child in a bath at a temperature of ninety to one hundred degrees and then rapidly douche it with tepid water, beginning at seventy or so and rapidly reducing the temperature as the child becomes more resistant and accustomed to sudden changes. Douching with salt water has a fine invigorating effect in cases of rickets and, if sea-water is unobtainable, rock-salt or some proprietary imitation of the marine substance, can be added with advantage to the daily bath. Curvatures of the bones sometimes yield to a prolonged course of sea baths, as do certain cases of spinal curvature, when assisted with douches and massage. It is essential that one should thoroughly dry the child's skin, as in rickets there is great liability to eczema, intertrigo and perspiration eruptions. The drying should be rapid, with vigorous frictions, in front of a fire, special attention being paid to all the flexures of the limbs, the folds of the buttocks and the back of the neck, which parts are very apt to be overlooked by the careless or hustling nurse.

FRESH AIR.

All are agreed as to the value of fresh air in consumptive and the like cases and it is high time that the public came to

recognise its equal value in rachitic, marasmic and similar affections. If the infant is a town resident, it should be taken out regularly every day into the open air, particularly that of the parks, which have been aptly called the lungs of the cities. Should the parents be in a position to afford a change of air, a prolonged sojourn on the sea coast should be recommended: for the tonic and bracing action of the air of the littoral, charged with ozone and salt, is of great service in rickety cases towards permanent cure. The longer the child stays at the seaside the better. We have many watering places in this part of the globe and I have found diarrhoea cases to do very well there, though the respiratory ones do better at a higher spot inland. A child can be safely taken out into the open air during the winter months, if it is well covered and protected from draughts. It may be advisable to give a little warm nourishment before leaving the house to assist in keeping up the body heat during the time that the child is out for the airing. It often happens that one is not able to effect much change in the household arrangements or the life of its inmates, as poverty or other adverse factors may stand in the way. Still, we must stimulate these people towards constant endeavour, particularly towards hygiene, proper feeding and fresh air. We must insist upon all the rooms, especially the living ones, being well ventilated, as the poor often prefer to spend their time in apartments in which the windows are almost hermetically sealed, they herding together in an atmosphere reeking of toxic emanations with the child as a central figure therein. Far too many nurseries are kept at an excessive temperature: so that, when the child is taken from the overheated room into the fresh air, it seems always to be taking cold and is then shut up in the old vapour-bath atmosphere until it shows signs of recovery, with consequent lowering of its powers of resistance to disease and diminished vitality. I have often come across cases in which the unfortunate infant has been kept indoors for the first seven months of its life at the colder time of the year, the usual rickets and its concomitants making their appearance. Should the social circumstances of the case allow, separate rooms should be used for the day and night nursery. The patient should be kept in a sunny well ventilated room, well lighted and kept free from dust by the almost total absence of superfluous pictures and draperies. The walls of the apartment should be coated with a washable distemper, thick carpets should be replaced by cork linoleum or cork and well washed and polished floors; heavy curtains must not be allowed to screen the windows and shut out the light or catch the dust and hold it. Too much furniture should not be allowed to intrude upon the floor-space. In short, the nursery should be plainly furnished and never be allowed to become stuffy. Even in the dwellings of the poor scrupulous cleanliness should obtain; this can hardly be expected when the mother has to spend the working hours of the day in the factory or hawking in the streets. It is well known to persons of intelligence that a child will never suffer from fresh air, however cold, though its skin and mucous membranes are very susceptible to sudden draughts - the moral being that, if it is allowed to crawl about the floors, great care must be taken that it is duly protected from this danger. The windows should be kept open throughout the twenty-four hours, all draughts, of course, being avoided. There is not usually great danger in the evening air, except when it is heavily charged with moisture and, even at these times, it is healthier than an atmosphere charged with carbonic acid gas and the foetid emanations of numerous sleepers. Fresh air is a stimulant and a tonic, it braces up the constitution, promotes appetite, improves the gastric functions and calms nervous irritability, thereby, in an ideal manner, fulfilling all the indications for the best adjunct to a rachitic cure. The efficacy of due observance of hygienic rules, combined with proper feeding, is well illustrated by a comparison between Hebrew and Italian children. The children of the slum Jews are usually strong and healthy, their homes and persons are clean and their food well arranged, with the result that only a small percentage will be found rachitic. On the other hand, the Italian children, so long as they are living under the bright sky and warm and sunny atmosphere of their native land, are generally hardy and healthy; but, when

transplanted to the tenements of foreign overcrowded slums, they become amazingly rachitic. If we inquire into these cases, we shall perhaps find that the new home is just as dirty as the one left behind in Italy; anyhow, it is no more wholesome and undoubtedly more degrading as regards morals, with the result that the emigrated Italians show more and more rickets with each succeeding generation. The ways of the slum dwellers are strange; they seem to delight in crowding together in a single living room, probably the kitchen, and, if there is a sick and ailing child, it is so surrounded by a simpering tribe of curious or sympathetic neighbours and visitors that it actually suffers from paucity of air, but a plenitude of the products of organic decomposition powerful for the production of this disease.

SUNSHINE.

From what has already been said, it will be gathered that sunlight is a prime factor in the prevention and cure of rickets, the more so as, in sunny countries, the affection is almost unknown, whereas in darker and colder climes it is all too prevalent. Sunlight, indeed, is Nature's greatest tonic; it promotes growth and energy and inhibits the hatching and development of morbid and other germs. It is no wonder, then, that the convalescence of a rickety child is markedly hastened if it can be kept constantly exposed to the influence of bright sunlight, which is also of especial service in cases of diarrhoea or respiratory trouble. It is not always possible to remove the patient to a country with a sunnier climate; short of this, every opportunity should be taken to keep the sufferer out of doors or near the open window for the fullest possible enjoyment of the beneficent action of the sun's rays. These patients, it would seem, seldom do well in ordinary hospitals; but, if they can be sent to convalescent homes in the country or at the seaside or sick children's institutions in airy and sunny situations, good results and rapid cures may reasonably be expected. In large and crowded towns and cities the introduction of roof-gardens could perhaps be made of great service in the treatment of this disease. Upon them the air is purer, sunlight is oftener available and there is less irritation from the noise of the traffic-teeming streets.

PHOTOTHERAPY.

Cases of rickets which haunt the hospitals of the towns should improve under various forms of artificial light, when judiciously used; and, in the summer or during the sunny days of the rest of the year, the exposure of the whole body to the action of the sun's rays passed through coloured glass, may be expected to cure or relieve, provided the case is given very thorough antirachitic treatment by liberal and approved diet. Light is a great stimulus to growth and nutrition and that form which is chemically active causes great dilatation of the capillaries and provides a stimulus to the whole organism; the interpretation of this in rickets is obvious. Nevertheless, sun-baths are much to be preferred to treatment by any form of artificial light in closed chambers, in which, of course, the pure air of the outside is not easy to obtain.

REST AND EXERCISE.

The slum children appear seldom to obtain the quiet and rest that their requirements demand. They should be disturbed as little as possible, teased by handling or irritated by noise of the streets or tenements. On the other hand, much may be done by the early and systematic training, even in unfavourable surroundings, to cause it to go to sleep at regular intervals. After some warm food, it should be put to sleep in a well-ventilated and darkened room or, if in its carriage, it should be laid flat and out of the direct rays of the sun. This caution would seem unnecessary were it not for the fact that sleeping rachitic infants are kept in their perambulators by careless nurses with the sun beating on their heads in not a few instances. Infants should be trained to go to sleep without the aid of a comforter or dummy teat; further, vigorous rocking of cradle or swinging in the arms may be regarded as quite unnecessary and sometimes positively injurious. The cot should not be thickly padded and only light hangings to keep off the draughts should be allowed, the sides being so arranged that the sleeper has no obstruction to the free circulation of air, particularly with a view to warding off the persistent tendency to the

debilitating rachitic perspirations. A more than ordinary amount of rest is essential to the cure of rickets, as the hypersensitive brain and muscles of the child easily tire and can only recuperate and gain tone and stamina from long periods of repose of body and mind. During the night the practice of allowing the child to sleep against its mother should be forbidden, as there is always the risk of overlying and it is a nasty unhealthy idea. A useful cot can be made from a box or basket. The bedclothes should be warm and light and, if the child suffers from cold feet or poor circulation, it may be advisable from time to time to supply extra heat with a hot rubber bag or bottle. Only a firm, tough pillow should be provided; for, if a soft one is used the child's head sinks into it and soon becomes overheated, thereby favouring the head-sweating so commonly observed and troublesome in this disease. To a large extent the restlessness of the patient can be controlled by keeping it comfortably warm in a cool and suitably ventilated apartment, by careful correction of the digestive disturbances, and so forth, with avoidance of routine feeding every time it wakes up. Sleep is an important factor in the promotion of growth and nutrition; but the value of rest and quiet to an undeveloped brain and nervous system is far too frequently overlooked in the treatment of infantile ailments. These growing rachitic individuals have hypersensitive brains and unstable nervous systems and, the nerve control being very imperfect, it needs but little excitement to bring on an attack of night-terrors, laryngismus or convulsions. The rachitic infant sleeps badly, particularly as it may have adenoids, indigestion or actual neurasthenia when coming of a neurotic stock. Children that are dragged about from place to place by fond parents, or taken into crowded or noisy or brilliantly lighted rooms, may in time develop nerve exhaustion; they usually sleep badly and either refuse food persistently or suffer from persistent vomiting. These patients, in the absence of an positive diagnosis in some other direction, should be regarded as neurasthenic and be kept particularly quiet the more so as the troublesome dyspepsia may be due to hyperexcitability of the nervous system occasioned by injudicious maternal management. In short, neurasthenia is very apt to occur in a rachitic child and considerably retard recovery. The patient requires to be kept quiet some little time both before and after its meals; small children should never be hurried home tired out to partake of food, a short rest before meals being very necessary. As the bedtime hour approaches children of all ages should be kept quiet, so that the nervous system shall be allowed to settle into a condition of comfortable equilibrium before the time of sleep. Just before being put to bed the child should not be taken into a brilliantly lighted room and danced up and down and handled by fond parents and admiring friends; nor should it be allowed to play or get overheated towards the close of its day, as such procedures can only lead to nervous excitability and restless nights or worse. In the case of the healthy child restful habits must, of course, be cultivated; but in the case of rickets there is a much greater need for insistence upon a tranquil existence, at least up to the time that the nervous system has been restored to stability. We must always bear in mind that rickets is a neurosis-maker and that if, to an already unstable nervous system, is added a condition of neurasthenia, any hope of permanent cure of the nerve trouble will be small, the child being rendered liable to grow up into a neurotic or become epileptic or hysterical and generally ailing. I do not consider that I am straining a point by including these measures under the treatment of rickets, as I held that it only by due attention to these minor details that a rachitic infant can be put under the best possible conditions for permanent and uncomplicated recovery at the earliest possible date. Now, though rest is important, exercise is just as much so, in view of the fact that the healthy infant delights in kicking unrestricted by the bedclothes or coverings. We must take a lesson from this and encourage rachitic patients to make free use of their limbs, such active movements promoting the nutrition of the muscles and accelerating the circulation of the blood, while at the same time the weight of the body being taken off the extremities, there ensues a minimised risk of development of the dreaded deformities. The lying-down position encourages expansion of the

chest and allows the spine to assume its natural shape; it is much to be preferred to the sitting position with its risk of curvations of the limbs. Crawling or sitting positions must never be allowed for any length of time, if the muscles are flabby and soft or when there is any tendency towards osseous anomalies as already described. Should the patient be old enough to go out for walks, it should not be taken any great distance or at too quick a pace beyond strength and muscular power. Unless the spinal muscles are strong and vigorous, the infant must be kept lying down in its perambulator when in the open air. The popular mailcart, in which the child sits bolt upright on a low seat, is not a satisfactory vehicle for rachitic patients; for the nose and mouth, being close to the ground, are apt to inhale a plentiful supply of dust and impurities, the cold wind strikes upwards from the road on to the face and legs, with the result that the child is exposed very much to the risk of poisoning from refuse, the invasion of disease germs and the possibility of sudden chills. During times of inclement weather and necessary confinement to the house, the child should be put into its outdoor clothes, put into a cot or perambulator and allowed to spend some time in a room with widely open windows.

PROTECTION FROM COLD.

It is very necessary that rickety children should be kept warm, as they are more sensitive to sudden changes from heat to cold than adults and have mucous membranes unduly sensitive to changes of temperature. We must therefore urge special precautions against the evil effects of chill. The depressing effect of cold upon the respiratory system must be remembered and it is just as important to keep the abdomen and feet thoroughly dry and warm, in order to ward off catarrhs of the bowels or renal organs. The heat of the body must be kept up and regulated by warm clothing, foods containing carbohydrates and fat and by studied protection from cold winds and sudden draughts. The patient should never be taken suddenly from a warm room into the cold air outside; but, if its living rooms are well ventilated and the windows are kept freely open, the risk of chill is considerably lessened, the body becoming more indifferent to changes of temperature. Children who are always catching cold have a depressed vitality and a low resistance to microbic infection. This fact is well instanced by cases in which children suffering from adenoid growths or rickets develop pharyngeal or bronchial catarrhs under the slightest provocation, which ailments they throw off with considerable difficulty during a period of weeks or months. The initial cause of the cold is a sudden chilling of the skin, though in most cases it will also be found that the system is overcharged and hampered by waste products throwing an extra strain on the excretory organs. In some cases, however, sudden abstraction of heat may be enough to cause a cold by lowering the vital powers of resistance. Children with digestive and excretory organs acting in a normal manner resist chills because they are better able to withstand its depressing influence; but in rickets the nutrition is low and vitality feeble, digestion is imperfect and encountering obstacles to its efficient performance, with the result that the patient has no reserve of effective disease resistance and suffers from even the slightest chill. These patients lose heat more rapidly than adults because of their smaller size, small bodies cooling at a quicker rate than those of larger size. This important point should always be remembered and given practical application, so as to obviate dangerous exposures to cold. It is an unfortunate fact that the slum children are far too often called upon to brave sudden changes of temperature by being taken out into places where the air is foul, vitiated and overheated, afterwards having to face the further depressing influence of the cold night air on the way home. It is quite common nowadays to see weakly infants out of doors late at night in the worst possible weather, returning or going to cinema halls or being further depressed by their brainless mothers in front of shops or gin palaces. They are not infrequently taken inside shops and brilliantly lighted places and subject there to noise and vitiated air. Many infants are taken out to spend the evening at the picture shows when they should be sleeping calmly at home; the sluttish mother will have her pleasure and, as she cannot leave her child at home,

she is bound to take it with her and risk its life; it is a wonder how so many of these children actually survive. In the summer time similar evil customs are observed daily; for every excursion train or omnibus carries many mothers with young children in their arms. All return tired and irritable and deprivation of natural rest, plus ever-fatigue, cannot fail often to react unfavourably upon both the nursing mother and her child. The common practice amongst mothers taking their infants and young children to the public houses and drinking-saloons is even more reprehensible and it is horrible to think that some of these degraded creatures actually give their babies gin or whisky to quieten their cries. So long as such social evils as these exist, we can hardly expect to eradicate rickets or other endemic maladies of children. The nature and characteristics of the clothing must also be taken into consideration in dealing with this disease. The garments must not be light or heavy, or there will be the undesirable risk of producing constriction of the body, irritation of the skin, restlessness, overheating, exhaustion and fatigue: in short discomfort from clothes will not allow an infant to obtain its natural rest. Badly fitting clothes may interfere with the various functions of the body; if too tight about the neck or chest, they impede circulation and respiration; about the abdomen, by interfering with peristalsis, they set up indigestion and constipation; from deficient ventilation, cutaneous disorders may arise; and any cramping of the limbs prevents the free and uncontrolled action of the various muscles and hinders their growth and development. In infants and rapidly growing children it is, therefore, very important that all clothes should be well made and of suitable material. In rickets, when the bones are soft, the evil effects of constriction or compression become more pronounced and must be guarded against; so badly fitting clothing may produce deformities or hinder proper recovery by causing pressure on the extremities. The pernicious effect of tight napkins must be obvious; care must be taken that they are loosely applied and never fitted tightly around the iliac crests or wedged between the thighs, lest they lead to retardation of the development of the pelvis, inhibition of growth or actual deformity of bone. I have reason to believe that keeping pads of material between the thighs is not infrequently the cause of genu valgum in children. The mother of the rachitic child, then, must be warned against the danger of tight diapers and, if there is already a tendency to deformity, she should be advised to fasten or sew the napkins loosely to the edge of the child's undervest, instead of fixing them round the waist. The napkins are usually made of towelling, but in rickets, when they are frequently soiled with foul and slimy motions, it is far better to use lumps of cottonwool, gamgee tissue or the like sanitary material for immediate burning, thereby obviating as far as possible danger of reinfection. Looseness, warmth and readiness of application are the three cardinal points in connection with infants' clothing. All garments should be simple in pattern, so as to take easily on and off. In order to avoid unnecessary handling of the hypersensitive patient, they should be made to fasten either all at the back or all at the front. As warmth is so important, the child should be dressed for the most part in flannel or in loosely fitting woollen garments of such weight that they shall not impede the movements of the limbs; the neck and arms and legs should be kept properly covered and an entire change of clothes must be made at night. The belly requires protection from sudden chills and for this indication the usual binder, when carelessly applied, is quite useless, as it crumples up on the chest, leaving all the parts below unprotected; on the other hand, it may be sewn so tightly round the child's body that it impedes both its respiration and the proper action of the abdominal organs. Attacks of diarrhoea are often excited by the exposure and sudden chilling of the surface of the belly. The binder is best made of soft flannel or knitted material and, in order to keep it in position, it should be fixed to an undervest reaching down to the hips. In certain cases special elastic belts may be prescribed for the relief of the abdominal atony; these things usually consist of a broad band, furnished with buckles at one end with straps at the other for fastening. The belt is sometimes further steadied by understraps passing outside the napkin underneath.

The lowest buckle should be fastened first and be the tightest of the lot and the top one, over the pit of the stomach should be loose. The elastic portion should pass over the front of the belly and come well down the front. Fit is everything with this class of apparatus and no minor detail should be neglected. It is easy to apply; it supports the weak abdominal walls and contained viscera; it tones up the general circulation of this area; it steadies respiration and, by giving a sort of peripheral massage, it improves muscular development. The heat of the body surface should receive equal distribution by proper clothing of proper weight. Far too often, in fear of colds, the mother indulges in excessive padding and binding of the chest, which will hinder the free expansion of the lungs and keep the patient in a continual state of perspiration: so that the normal function of the heat regulating apparatus of the skin is lowered and the trouble ensues from sudden abstraction of latent heat. The ailments arising from sudden chilling of the belly can not infrequently be referred to the action of cold upon the limbs, particularly upon the soles of the feet. These patients should never be allowed to go about in short-sleeved frocks and diminutive socks, but always wear a knitted vest with long sleeves under a long outer garments with sleeves to the wrist and comfortable woollen gloves. All constriction of the chest must be carefully avoided and the fit be perfect, the shoulders only bearing the weight of the clothes, without constricting straps. Bare legs are a real source of danger to a rickety child, as it very often suffers from coldness of the limbs and perhaps cyanosis of the hands and feet, being easily affected by any chill to these parts, with the added disadvantage of cooling of the body by the symptomatic perspiration and possible consequent congestion of the internal organs. There is no objection to socks under guarded conditions in the house; but in the open air, except in the mildest weather, long stockings or woollen gaiters should be worn. Bare arms and legs are a frequent source of diarrhoea, bronchitis, colds and chronic congestive disorders of the digestive organs; further, the growth of bone is unfavourably influenced by coldness of the limbs, so that the absurd custom of dressing children in flimsy frocks with low-cut necks and short sleeves, or in cotton undergarments and socks reaching only a little above the ankles, is merely pandering to the vanity of the parents, or the detestable theories of the "hardening" faddists, and has no justification whatever in rickety cases at least. Indeed, there is no need to harden a child; for, as vitality improves, its powers of resistance will automatically grow stronger without artificial aid: so it is far better to leave Nature to do its proper work in this direction. The nightdress should be of warm flannel; it should be as loose as a sack and fastened around the feet outside, as these patients are restless sleepers and kick off the bedclothes and become chilled by the night air of the room playing on the exposed belly when this sleeping-bag precaution is not adopted. It goes without saying that both the outer garments and the underclothing should be of washable material. All garments in these cases may well err on the warm side, rather than fall short thereof, as the action of the heart is feeble, the arterial tone is poor, the circulation is sluggish, the temperature is subnormal and the skin is rapidly cooled by the excessive perspirations. The clothing is therefore of prime importance in dealing with all cases of this disease.

In general, we may make it a rigid rule that the rickety child should be placed in the best possible surroundings; it should exist and repose in a properly ventilated apartment and have an abundance of fresh air and sunlight, with proper protection from cold and chills and damp; its clothing should be warm, but not too heavy or impervious to ventilation; and its general health should be further promoted by bathing and general friction and massage. In such ways as these the body is made more resistant to chill and the better-oxygenated blood can cope more effectively with the morbid elements in this disease. The rule of diet, as presently to be outlined, must receive proper organisation, medicine receiving a second place to the observance of hygienic laws. Health visitors can do a lot of good in these cases and their more general employment is much to be recommended; municipal authorities seem to grudge their salaries, but sanitarians everywhere know that the same may be regarded as money well spent.

(b) SYMPTOMATIC TREATMENT

General Considerations.

We have seen that the rachitic patient is specially open to take various intercurrent affections, especially those that affect the digestive system or the nervous: so that a proper cure is out of the question if we fail to deal with the underlying trouble. Thus, in catarrhal ailments of the digestive and respiratory systems, the symptoms may be relieved and the malady treated on general principles, if at the same time the patient is put upon some antirachitic diet and suitable drugs are given to eradicate the fundamental rachitic dyscrasia and prevent the recurrence of the intercurrent disease. All catarrhal conditions of the stomach and bowels ought to be corrected by careful attention to the surroundings of the patient, avoidance of chills to the feet or the belly, regulation of the diet and certain medicaments suited to the secondary disease. Further, as soon as the catarrhal condition in question has been effectively dealt with the rachitic dyscrasia must be eradicated and no case regarded as really cured or safe from relapse until it has been treated for a long time on antirachitic lines. After an attack of diarrhoea has been corrected, the irritant cause treated by mild aperients and the bowels soothed and disinfected by sedatives and antiseptics, vigorous measures should be instituted against the underlying rickets and the patient prescribed a diet rich in fat and proteids. The rachitic will do better without the use of astringents if such drugs as cod-liver oil and approved tonics are used. All recurrences must be guarded against sudden chills or overfeeding, special precautions being taken during the time of teething and during the hot months of the year. In a similar manner all catarrhs of the respiratory organs must be first corrected on the usual lines. The same is true of the nervous system, as the cerebral tissue is backward in development and the reflex nervous system is excitable to a very great extent: so that trivial conditions, which would not perhaps affect a normal child at all, may very readily set up various spasmodic affections of which we may instance tetany, laryngeal spasm and convulsions. There is also defective nutrition of the motor centres, the condition of this system being comparable to the badly nourished and hyperexcitable muscles, which are so readily stimulated and so easily tired. Therefore the treatment of all affections of the nervous system must be on the dual lines of soothing and allaying its hyperexcitable condition, with removal of any reflex cause of irritation and of feeding and building up the starved and badly nourished nervous tissues; indeed, for a considerable period it may be necessary to administer a combination of remedies which will meet these two indications. During the time that the patient is having such treatment as this, various antirachitic measures must be adopted. The diet should contain plenty of animal fat or cream or raw meat juice; the general hygiene of the case must be improved and plenty of fresh air insisted upon, the child being kept out of doors in all reasonable weather. Cod-liver oil should be prescribed, with or without phosphorus and arsenic and the like tonics for extended periods. It cannot too strongly be insisted upon that various complications, such as bronchitis and the like internal affections, may at any time interrupt the ordinary course of rickets and prove a source of great danger to the patient, who even during an apparently satisfactory convalescence may perish or have a very dangerous illness from superadded disease; an ordinary case of rickets is, as we know, largely a question of hygiene and dietetics, though the scene is changed into assiduous therapy when complications or sequels arise, which must be properly treated on the most persevering and intelligent lines.

BRONCHITIS.

The great liability of rachitic children to such respiratory complications as this must always be borne in mind and the utmost care be taken to prevent origination; when once in evidence, their treatment must be on orthodox lines with special regard to the already existing debilitation resulting from the fundamental rickets. Rachitic bronchitis is in not a few

instances found to have had its origination in adenoids, enlarges tonsils or hypertrophied mucous membrane of these parts, blocking the upper air passages and preventing the proper expansion of the lungs and oxygenation of the blood. Another occasional cause is the fat condition of the patient. It is not a healthy fat and causes the blood to be impeded in its course amongst the tissues in which it lies, with resulting capillary sluggishness, congestion and catarrh in the air passages. As seen as symptoms of bronchitis are observed the child should be placed in its cradle near the fire, duly protected from all draughts and chills; the bedclothes should be abundant, but light as their weight might impede the already hampered movements of the chest. The air of the apartment should be kept warm by steam impregnated by salsams, pine-oil or creosote until the more dangerous symptoms have subsided, a tent covering the cradle in order to secure the full effects of the medicated vapour. The popular linseed poultice is somewhat out of date; it is better to make use of the proprietary antiphlegistine (a kaolin preparation) or spongiopiline from the drug-store. As seen as possible after the acute stage, an abundance of fresh air should be allowed to circulate in the room; the windows must be kept open day and night, unless special circumstances contraindicate and the presence of too many visitors, vitiating the air of the room and exciting the patient, must be forbidden. The constitution must be braced up by an ample supply of good and wholesome food. The bowels should be freely acted upon at first and then a stimulating expecterant mixture should be prescribed. A good one is composed of acetate of ammonia solution, nitrate of potash, glycerine and water; another popular one contains iodide of potassium, nitrate of potash, solution of acetate of ammonia, syrup and cinnamon water. When the cough becomes easier the child should be given a mixture containing ipecacuanha wine, ammonium carbonate, honey, glycerine and water. Should the heart show signs of enfeeblement, it is advisable to prescribe a mixture of carbonate of ammonia, tincture of digitalis, syrup of Virginian prune and aniseed-water - in all cases, of course, dosage being according to age on ordinary lines. If, at the commencement of the bronchitic attack, there is much obstruction from sticky mucus, a full dose of the above-mentioned wine should cause relief by exciting a clearing vomiting. Mustard and the like counterirritants may, if thought advisable, be applied to the chest. The condition of the heart should be carefully watched and, if cyanosis be observed, a little brandy should be given now and then. Sometimes nitroglycerine is useful in heart failure with congestion of the lungs, the usual dose in these cases being one five-hundredth of a grain every hour or so, according to indications observed. It is as well, however, to bear in mind that the chronic bronchitis of rickets should not be treated entirely by squills, the above-mentioned wine, depressing cough mixtures and expecterants, particularly opiates, it being far better method to rely more upon tonics and cod-liver oil. In these respiratory affections, half a minim, thrice daily, of the officinal solution of arsenic forms an excellent tonic for convalescence, particularly if there is any tendency to enlargement of the bronchial or cervical glands. During the stage of recovery it is very important that the child be guarded from relapses - the more so when cold winds are about; nevertheless, it should not be coddled indoors, but be made to live an outdoor life, with an abundance of fresh air, and sleep with the windows open.

BRONCHOPNEUMONIA.

This is an alarming complication of rickets and one that will always call for the best therapeutic efforts of the medical attendant. The temperature is apt to assume a considerable height; tepid sponging is the best way to reduce it, at least in private practice when the parents object to the use of the cold bath or wet pack. I do not consider it safe to use antipyretics in these cases. The bronchitis kettle is useful when the bronchial secretion is deficient. Mustard and other counterirritants are of great utility sometimes. Antiphlegistine is well established in the treatment of this condition. The tubes may be cleared by the use of ipecacuanha wine, antimony tartrate and other expecterants. Aconite and squills are apt to be depressing and ought to be avoided in most instances.

Acetate of ammonia and bicarbonate of potash or potassium iodide or the sodium compound are of value in rendering the mucus less tenacious and, even at an early stage, nux vomica or digitalis may be combined with these. When free secretion is established, hot applications should be discontinued and the chest wrapped in cottonwool instead. I find that creosotal, guaiacol and other creosote preparations are of signal service in this disease, especially when the respiratory trouble is complicated with diarrhoea or digestive disturbances. Later on, the creosote preparation may be combined with hypophosphites and cod-liver oil and the patient given a diet rich in fat. In my experience this form of treatment has tended to shorten the convalescence more than if the chest disease had alone received attention on ordinary therapeutic lines.

COLLAPSE OF THE LUNGS.

This condition, also emphysema, may arise in the course of rachitic disease. At the commencement an emetic often does good by clearing out the bronchial tubes, a teaspoonful of the wine of ipecacuanha being perhaps the best agent to use, though all depressing drugs must be avoided. Stimulants are necessary from the first and may assume the form of brandy, whisky, whey or egg mixture, all of which are of often of signal service in these patients of tender age; the condition of the circulation will point out the dosage and desirability of exhibition of these agents. If it is weak, compressible, rapid or irregular, stimulants are clearly indicated, as well as when there is blanching or cyanosis of the cutaneous surface; they are most useful at the crisis of the disease, if the temperature falls suddenly or if definite forms of pulmonary collapse are observed. Then it is sometimes advisable to apply two or three leeches to the front of the chest over the region of the heart, some form of counter-irritant replacing them afterwards in this locality. Nitroglycerine is a useful drug in collapse cases, as also is a tiny dose of strychnine hypodermically - say, one three-hundredth of a grain for a child of twelve months. Caffeine and atropine are also useful as respiratory stimulants of the safe kind, the dose of the former being one-twentieth and of the latter one-eighthundredth of a grain. Massage of the chest is indicated, as is also the inhalation of oxygen in desperate cases. Creosote preparations are of use, especially when gastrointestinal affections coexist. The diet should be of the ordinary antirachitic kind and an occasional dose of calomel or grey powder may be tried when constipation is present. A hot mustard bath may be tried in certain cases of great severity and the dose of alcohol may be increased, if necessary. During convalescence the usual tonics are indicated and fresh air particularly.

CONSTIPATION.

Rachitic children are not infrequently much troubled with this irritating condition, which is often very slow to respond to treatment, drugs having to be given sometimes over a prolonged period and the diet very carefully arranged from time to time. It must always be regarded as expressive of the atonic condition of the muscular system and the general malnutrition obtaining; the early treatment should, therefore, be directed towards improving the asthenia before attempting any permanent cure of the digestive disturbances. The fundamental general feebleness of the muscular walls of the bowels and the atony of the abdominal walls should be treated to a great extent by alteration of the diet and improving the general health and condition of the patient. Improvement of the muscular tone of the muscles of the abdomen may be effected sometimes by minute doses of nux vomica, though there are some who warn us against the possibility of exciting a slumbering tendency to convulsions in this way; such, however, is not in accordance with my experience, as I have never known a fit to occur from a judicious use of this drug. It may be combined with syrup of senna and compound infusion of gentian for exhibition before meals. Massage and electricity, if properly applied, assist greatly the action of drugs in the cure of chronic atonic constipation of rickets. Teaspoonful doses of extract of malt and bone-marrow may here be preferable to cod-liver oil, the stools manifesting less tendency to fermentation and having a softer consistence, then they otherwise would. Virol is frequently of service also.

The use of the syrup of the iodide of iron, in small doses in conjunction with meat extracts, is to be recommended. I do not favour the prescription of purgatives by way of routine in rachitic constipation; small doses of grey powder are, however, useful, being both laxative and antiseptic. Cod-liver oil sometimes works wonders and a mixture of magnesia, syrup of ginger, dill-water and mucilage may do good when other measures fail. Faecal accumulations in the large intestine may be cleared out by daily enemata of tepid water and glycerine suppositories are to be recommended. If preferred, a small pointed piece of hardish soap may be pushed into the rectum for purgation. These cases sometimes do well on oatmeal porridge when old enough to digest it. Such mild aperients as aloin, belladonna and cascara can meet with no objections under approved conditions. The latter may be given with salines over a prolonged period. Castor-oil may be given in a large single dose, if a speedy action be desired; otherwise it should be ordered in small fractional doses several times a day and it is as well to bear in mind that the addition of salol, salicylate of soda (especially if there be any rheumatic tendency), boric acid or resorcin will usually enhance its beneficial effects. I find that a tenth of a grain of calomel is very useful in obstinate cases. Aromatic medicaments and mineral acids are also trusty agents when indicated. Fresh air and exercise are, of course, indicated here as elsewhere.

CONVULSIONS.

General convulsions are not infrequent sequels of attacks of laryngeal spasm in rachitic patients. Indeed, rickets, occurring in children over six months of age, particularly if fed artificially, should be suspected as the possible cause of the fits and carefully searched for. Fits require prompt treatment in order to save the child's life, the best initial treatment being a hot bath and clearance of the bowels with calomel or grey powder. Cold douching of the spine, after the morning bath, is of great service for conferring a suitable tone upon the nervous system, in addition to which the patient should be kept as long as possible in the open air every day. After the bowels have been cleared it is sometimes a good thing to lance the gums if there is any difficulty in dentition. The fits may be treated with sedatives; the inhalation of chloroform or amyl nitrite may be useful in checking the violence of the spasms and in warding off haemorrhage in the brain. One-grain doses of chloral sometimes is more useful than the bromides, which latter must be given in largish doses to be of use. During the time that the child is in the hot bath cold water may be poured upon its head. Some advise a hypodermic injection of one minim of the solution of morphia hydrochlorate in severe cases. When the fits have passed off and the irritant cause is removed the nervous system may be ~~restored~~ ^{rejuvenated} by the usual antirachitic measures and the body generally will be toned up in this way. If we merely relieve the convulsive attacks and fail to recognise the underlying rachitic condition, the convulsions will surely return; they may ultimately kill the patient, render him feeble minded or make him an epileptic later on.

DIARRHOEA.

Inordinate action of the bowels may alternate with/constipation and be very troublesome and accompanied by restlessness, thirst, flatulence, stomatitis, abdominal distension and perhaps vomiting. The stools in these cases may have a greenish hue and contain undigested/food particles or be frothy and yellow. In severe cases the milk should be stopped and albumin-water or barley-water administered until some improvement is observed. Half a teaspoonful of castor-oil or so should be tried at first or, if preferred, a tenth of a grain of calomel. Then a mixture may be prescribed containing carbonate of magnesia and lime-water in equal quantities, a teaspoonful every four hours being the dose. Urotropin is an intestinal, as well as a urinary antiseptic, and may be tried, as also may the well-known creosote preparations. I do not favour the use of chlorine-water advocated in certain quarters, as children resent it very much. When the stools are yellow, frothy and sour, it is useful to prescribe a mixture of carbonate of bismuth, tincture of catechu, compound tincture of cardomoms, spirits of chloroform, mucilage and dill-water. In obstinate cases good

results may follow the exhibition of a mixture of salicylate of soda, castor-oil, mucilage, syrup of ginger and peppermint-water, or one containing bicarbonate of soda, compound tincture of rhubarb, tincture of ginger, compound tincture of cardomoms, spirits of ether and cinnamon-water. There are many advocates for the use of tincture of opium in these cases, but it is advisable to be ever on guard for toxic effects. I have abandoned the use of Metchnikoff's milk for the lactic acid treatment, having been dissatisfied therewith. Naphthalin is a good antiseptic to destroy the bad smell of the motions and urine in this slime-producing disease; it may be given in cachets in doses of two grains for every year of the patient's age, mixture form not being so suitable.

HEAD SWEATING.

Rachitic patients, as we have seen, are apt to be much inconvenienced by sweating about the head and neck at night; the sleep is thereby much disturbed and the suggestion arises of there being some element of defective nerve control or some intoxication of the nervous system causing irritation of the sweat glands. One must pay careful attention to the diet and avoidance of food with excess of starch or sugar in the evening meal, together with fresh air, light bedclothes and tepid sponging last thing at night. A small dose of alcohol, I find, produces restful sleep and tends markedly to allay these perspirations. I am not in a position to claim any particular efficacy for the exhibition of suprarenal extract advised by some for the relief of this condition, though there appears to be a certain amount of virtue for phosphorus when judiciously administered. The ordinary antirachitic measures must, of course, be used. It is a good thing to sponge the parts with vinegar and water, following it with a dusting-powder of equal parts of oxide of zinc, boric acid and starch. It is also a good plan to substitute a hair or air pillow for the ordinary one of feathers and good results may sometimes be achieved from sponging the head with the liniment of belladonna or the administration of the tincture of that agent. Children stand this drug very well and I do not hesitate to give five to ten minims of the tincture three times a day in certain cases at one year, or as much as double that quantity for special indications. A minim of atropine solution at bedtime sometimes checks the perspirations of the head just as well. In general, however, it is comparatively seldom that the need arises for the exhibition of drugs for this condition, if proper dieting and hygiene are arranged.

INFECTION OF THE MOUTH.

Artificially fed very often are troubled with affection of the mouth, the hygiene of which cavity must always receive careful attention. I am therefore in favour of regular cleansing with hot water after each feed. Should any aphthous condition be observed, one should have recourse to painting with glycerine of boric acid or a solution of chlorate of potash, borax, compound tragacanth powder, glycerine and chloroform-water. When the gums are so tender and spongy as to suggest the presence of a scorbutic taint, it is good treatment to prescribe a mouth-wash containing glycerine of ~~barbolic acid~~ tannin and boric acid in water, the same to be used every four hours. The usual tonics should also be given and the diet be judiciously arranged.

INSOMNIA.

Ordinary treatment on general principles should be given for the night terrors and general restlessness of rickets. The patient should be put to bed early and allowed to go quietly to sleep in a darkened, well-ventilated apartment and covered with light bedclothes. Any irritant causing disturbance of the digestive tract must be forthwith removed and, if necessary, small doses of grey powder or calomel may be prescribed for the gastrointestinal disturbance recognised as excitant. As the patient increases in age it must not be allowed to have any food late at night, merely a light meal early on of milk and biscuit and bread. All games and excitements just before going to bed must be avoided. Regularity of bedtime must be insisted upon and a little bromide may be tried at first. Nevertheless, the fewer drugs tried for this condition the better; careful training and good management are much to be preferred.

LARYNGISMUS STRIDULUS.

The prophylaxis of this distressing affection is of very great importance. The mother should be warned that her rickety child is extraordinarily sensitive to any reflex stimulus and that a laryngeal attack may be excited by such trivial disturbances as a whiff of cold air. Existence in a vitiated and stuffy apartment should be forbidden and the child encouraged to spend its indoor time conditions of good ventilation, regular open air exercise, quietude and the usual antirachitic arrangements. The digestive system must be kept in good tone and all constipation or gastrointestinal irritation must be removed. In order to deal with the underlying rachitic condition we must arrange for a diet rich in animal fat or cream, with cod-liver oil, arsenic and other tonics. A few drops of whisky every four or more hours sometimes is of great service in these cases. For removal of the reflex internal irritation and soothing of the general excitability a dose of castor-oil or an enema will usually suffice, though calomel may be tried if preferred. Then the diet must be corrected, followed by bismuth and soda or other stomachics. Enlarged tonsils, adenoids or elongated uvula may be causing excitability and should be removed; catarrh of bronchi or larynx must also receive attention when present. If teething be the cause, it may be necessary to lance the gums. All emotional excitement must be avoided, fits of temper, laughter or tears controlled and late hours or frequent visitors to the nursery must be forbidden, especially near bedtime, so that the child may be encouraged to go to sleep as usual at the time that fits are most often observed. Small doses of bromide or chloral may be given for some time after the attacks until nervous equilibrium has been restored; some of the cases require these sedatives to be given, however, for weeks lest relapses occur and codeia and valerianate of zinc are useful alternative drugs, though some have greater faith in antipyrin, in dose for age, every four hours. The attacks of laryngismus themselves may usually be either prevented or relieved by dashing cold water upon the child's face or hands, or by smartly slapping the cheeks or by passing the finger to the back of the pharynx to excite vomiting. Severe cases may call for the administration of chloroform to relax the spasm; hot fomentations may be applied to the throat in front and the hot bath in these cases is not to be despised. Antispasmodic drugs do good in not a few instances and I would commend a third of a grain of musk for a child of twelve months every six hours. The tincture of belladonna is also of service and antipyrin, two grains every four hours for a child of a year, seems increasing in general favour.

MUSCULAR ASTHENIA.

We have seen that formic acid and the formates have advocates for the relief of this condition. As a rule, the weakness of the muscular system, so often observed in rickets, slowly improves under careful dieting, plenty of fresh air, baths, massage and electricity, especially when aided by tonic on the already-mentioned lines.

TETANY.

The commonly observed tetany and motor spasms are the direct outcome of the disturbed and excitable condition of the nervous system in rickets, as well as the general weakness obtaining. The indication is to search for any reflex cause of irritation, to remove it and give sedatives, following with assiduous antirachitic treatment on the usual lines. If the presence of worms be suspected, a vermifuge should be administered. Worms, however, are not so very often the cause, the latter being more usually referable to the presence of coarse or fermenting food in the stomach or general debilitation by prolonged diarrhoea, with or without dilatation of the stomach. The diet must therefore be judiciously arranged, with special avoidance of oatmeal preparations, jams, skins and seeds of fruit and vegetables with coarse stalks and fibres, such as celery. On the other hand, one must not starve the patient, but allow a liberal diet to relieve the exhaustion present. It is advisable to forbid the administration of ordinary milk and starchy foods for the time and prescribe instead meat, mince, meat extract or soups: in short, a full proteid dietary. In view of the fact that calcium chloride, - present in cow's milk to five times as much as in maternal milk, - has been experimentally shown to increase the irritability of the motor nerves

and sometimes cause actual spasms, it is just as well to exercise rigid control over the animal secretion in these cases. The diarrhoea present usually yields to intestinal clearance with castor-oil or calomel, followed by a course of gastric sedatives and such intestinal antiseptics as bismuth and chalk or salol, with small doses of opium when there is great pain. During the actual attack of tetany a mixture of chloral and bromide of potassium or small doses of antipyrin may be given for frequent daily exhibition over an extended period. The severe pains induced by the spasms may be relieved somewhat by hot baths or friction of the limbs. Later on, a stimulating antirachitic treatment should be advised, particularly a diet rich in proteid and fats, with or without cod-liver oil, phosphorus and strychnine, assisted by massage, weak electricity and tepid daily spongings. I am not in love with the administration of thyroid extract in this condition and I am not convinced that the parathyroids have anything to do with the excitation of the spasms, though some authorities have argued to the contrary and drawn interesting comparisons and analogies, which it does not seem advisable to reproduce here, despite their great interest and alleged importance.

NAUSEA AND FLATULENCE.

Infants that are reared upon the bottle artificially, as well as not a few of those upon the breast, are apt to be troubled with vomiting and flatulence. In such cases it is advisable to give a teaspoonful of lime-water before the feed and reduce the amount of the latter each time. Severe cases of bottle wind and sickness call for substitution of condensed or peptonised milk for the cow's product. An occasional dose of calomel or occasional doses of a mixture of fluid magnesia and lime-water may be expected to do good. Common salt should be added to the food and dosing with ammonium chloride sometimes relieves apparently intractable cases. Dill-water, pepsin and other drugs of the stomach-soothing character may be given, of course, according to indications from time to time arising.

HYDROCEPHALUS.

All sorts of drugs have been recommended for this distressing accident in rickets. Attempts to absorb the liquid have been made by small doses of grey powder, mercurial inunctions, iodine paint and the iodides internally. Leeches have been applied behind the ears and free purgation with calomel has been recommended. The head has been tapped, blebstered, and so forth; but all these measures are unsatisfactory, as the effusion returns again in the vast majority of instances.

(c) SURGICAL TREATMENT

GENERAL CONSIDERATIONS.

The treatment of the various deformities that may arise during the course of rachitic disease would require, in view of their number, a large amount of space for their proper consideration: so that I do not propose to give more than a brief outline of the more important points as they have appealed to me in actual practice in this country and at Home. Despite the fact that the actual correction of the osseous anomalies of this disease not infrequently call for the services of an expert surgeon, I have often found myself in a position to do much good with rest, baths, massage and the like general measures; indeed, in many cases it has been possible to postpone an operation indefinitely or even to regard it as quite unnecessary. During the period of the affection that the bones are soft I have sometimes been able to straighten them out by gradual and well-directed pressure or force; but, when once they have passed into the stage of eburnation, their extraordinary hardness has resisted all attempts to do so and called for operative interference. It will be found that the osseous anomalies of rickets not infrequently exhibit a tendency towards natural cure: so that being in an unseemly hurry to correct the curvatures is inadvisable and unnecessary in most of the cases. At an early stage the deformities of the arms and legs must be prevented by keeping the child off its feet and by the institution of such antirachitic measures as daily baths, douches and massage. The patient must not be allowed to make efforts at walking and, if we attend to this precaution, better and more rapid results will be obtained than from the employment of splints, which latter is a slow method, not infrequently clumsily carried out by the parent and extremely irritating to the child. In general, it is a safe rule not to have recourse to operative measures until the child is three years of age or a little more, in view of the already-mentioned tendency towards natural improvement: in short, we must give Nature a chance. Usually, if the patient has deformities well-marked and is under the above-mentioned age of three, splints should be applied and, if necessary, there should be forcible straightening of the extremities involved. If the deformity remains stationary for three months and is not benefited by padded, frequently and carefully adjusted splints, it is time to perform an osteotomy. The social circumstances of the case, however, must largely govern procedure in many instances. If the case is seen early and the parents are in good circumstances, the case can be frequently seen for splint adjustment and there is facility for systematic douchings and massage, there is generally little need for operative interference. The contrary, however, obtains amongst the poor, as they are unable to pay for skilled medical attendance and are not infrequently too lazy to take the child to the hospital, even if such is near at hand. Should fractures occur, the periosteum remains intact and the ends of the bones are not entirely separated: so that the limb can be straightened with ease and the broken parts kept in apposition by means of appropriate splinting material. These children, who not infrequently suffer from greenstick fractures about the limbs or collar-bones, should always receive good antirachitic treatment judiciously directed towards the provision of growth of serviceable bone.

ARMS.

With the object of preventing the occurrence of deformities of the upper extremities the infant must be kept from crawling about the floor; for, whilst doing this, it throws the whole weight of the trunk upon its hands and arms and the soft and decalcified bones, unable to support it, bend beneath the pressure above. One of the patent forms of rocking-chair will constitute a pleasing change of posture, if the patient is restless and cannot be kept continually on its back. Splints may be used, if necessary, but, as the deformities in this locality are seldom severe, osteotomy is rarely called for.

CLAVICLES.

Greenstick fractures are somewhat frequently observed in this region and, to prevent them, the patient must be carefully handled or exercised. When occurring, the usual splints are required and antirachitic treatment always.

HEAD.

It is not possible to institute any successful treatment for the rachitic bosses upon the frontal and parietal bones by surgical means; they often persist through life, though usually to a minor degree and inconspicuously.

PELVIS.

In the case of female rachitic children it is necessary to keep an eye to the condition of the pelvic bones lest they form a source of great danger during parturition in marital life. Should all the bones be soft it may be necessary to keep the patient in the recumbent posture, lest the pressure of the trunk from above and from the legs below forces the sacrum forwards and the ischial bones inwards, thereby producing the well-known pelvic deformity of this disease. But is advisable to bear in mind that lying upon the back may itself give rise a flattening of the pelvis, causing the pubic symphysis to sink backwards. Therefore, a certain amount of walking or free movement may be advisable and, if kept recumbent, the position of child should be frequently changed to prevent postural curvature of the bony parts. Patent splints, jackets or chairs may be tried according to indications.

SPINE.

Spinal curvatures of rickets are not actual disease of bone as in tuberculosis, but bendings due to badly nourished, and consequently weakened, ligaments and muscles. Neglect of their treatment may lead to permanent deformities. The patient should not be allowed to sit up too soon - at least not until it is certain that the above-mentioned parts are able to support the body weight. The common custom of carrying infants always on the same arm is a very bad one, being a prolific cause of curvature of the spine. If the patient is weakly weak in the back, it should be carried about on a pillow and kept flat upon its back, an abundance of open air exercise being allowed in favourable weather and good ventilation indoors. The tone of the weak and flabby muscles and ligaments should be restored as much as possible by massage thereof and the usual tonics indicated for the therapy of this disease, particularly cod-liver oil. One may also expect great benefit to arise from the use of douches to this region, using hot water under the age of eighteen months and cold water afterwards, with or without the stimulating addition of common salt. I am in favour of avoiding splints in most cases, unless the deformity of the spine be very great, when the child should be taken to a good surgical appliance shop and carefully fitted with the most up-to-date arrangement for giving support; whether this be made of gutta-percha or not, it should be well lined and pass well down over the belly, there being secured by a bandage and armholes above. Older children may with advantage use one of numerous patent rockingchairs intended for spinal support and gentle exercise in this disease. Plaster of Paris splints are useless and not infrequently injurious.,

RIBS.

It is very necessary that the tendency to fracture of the ribs be prevented and to bear in mind that the depression of these structures by the atmospheric pressure encroaches upon the space in the chest occupied normally by the lungs and so keeps up a condition of chronic catarrh of the bronchial tubes. If the lungs are chronically hampered in their free expansion, the deformity of pigeon-breast may remain through life. Therefore, the muscles of the chest, particularly those between the ribs, should be stimulated to their share in free expansion by frequent massage, with or without tonic embrocations. Much support can be obtained by the application of a firm flannel bandage round the belly; for it braces up the lax abdominal walls, prevents the respiration from being entirely diaphragmatic and obliges the patient to bring the intercostal musculature into proper action. This binder need not necessarily be worn continuously, but at least for an hour or two per day, being slackened whenever it occasions irritation or discomfort. All falls upon the ribs must be guarded against and trumpet-blowing or the manufacture of soap-bubbles may be expected to promote the desired total inflation of the lungs; attacks of crying seem to be Nature's method of doing this.

THIGHS.

All possible unsightly bending of the femora should be prevented, when feared, by keeping the patient from walking during the time that the bones are in a softened condition, pillow-carriage being instituted. Incurable cases should be submitted to the usual surgical operation upon the distorted bone.

HIP.

The hip-joint deformity known as cœxa vara should be prevented as above; when occurring, the part should be put up in a specially constructed surgical splint and incurable cases treated surgically. In this and other cases the underlying rachitic condition should, of course, receive the usual attention.

KNEE.

Knock-knee or genu valgum may be due to deformity of the lower end of the shaft of the femur or of the upper part of the tibia or to yielding of the ligaments about the articulations. It should be treated by the application of a pad between the two knees, with firm inward strapping of the legs, or by the long-continued use of special splints upon the outer side of the thigh and leg. Osteotomy must be performed in incurable cases.

TIBIA.

The occurrence of bowleg or genu varum should be prevented by keeping the patient lying down; special splints may be applied and incurable cases call for surgical interference.

FEET.

The prevention of flat-foot calls for avoidance of throwing too much body weight upon the part and the wearing of suitable boots specially devised by surgeons and described in their works. It is seldom necessary to prescribe an artificial or spring, if antirachitic general measures be instituted and deuching and massage are practised assiduously and intelligently.

(d) DIETETIC TREATMENT

(A) During the First Year

I n t r o d u c t i o n

It is not my intention to attempt here a complete account of the principles and methods of infant feeding, - as the same might partake of the supererogatory nature largely of a sort of rehash of students' text-book descriptions, - but to deal with the more important practical points concerning the dietetics of rickets as they have appealed to me in the course of a somewhat considerable pediatric experience.

One of the most important duties of the medical man is to impress upon the mother the urgent necessity of breast feeding, if she desires her infant to escape this serious disease. She must be made aware that no food can compare for a moment, as regards efficacy, with her own mammary secretion during the first year of the infant's existence until weaned. Even the excellent milk depots, established in various places, are of quite secondary importance in this respect and have the disadvantage in certain cases of discouraging breast feeding by their very existence under such convenient conditions. The same objection, however, cannot be urged against charitable restaurants of certain towns, where mothers of semi-starvation qualification are given free food. After the mother has been duly informed of the necessity for breast-feeding, she should be made aware of the danger of nursing her infant beyond the normal period, as her milk tends to deteriorate towards the end of the first year, particularly as regards its proteid content. She should be made to lead a careful, regular and quiet existence; so that worries, pleasures and excitements may not interfere with the quality of her milk. From the very first the breasts should be kept perfectly clean and aseptic and the infant should be taught regular dietetic habits from the time of its birth. Should she become very much debilitated or "run-down" as it is termed, it is a good plan to remove the child to another apartment at night and feed it on the bottle whilst she partakes of the necessary sleep; in this way the quality of the milk during the day is greatly improved, and after the fifth month there should be no nocturnal breast feeds in any case. Should the frequent slimy motions of the nursing indicate nitrogenous shortage, extra meals of modified milk should be given; if there occur proteid shortage or poorness or scantiness of breast milk, the general health of the mother should at once be attended to, and nocturnal feeds should be given up, meat and eggs entering into her diet and a little malt or iron prescribed. Lactagol may also be tried and infusion of aniseed is also popular for increasing the flow of milk. But no alcohol should be given in these cases. Too rich milk gives the child indigestion and colic, with past stools and actual loss of flesh; mothers so affected should be encouraged to take more exercise in the open air, with reduction of meat in the diet and total abstinence from all alcoholic beverages. Nursing mothers are apt to become anaemic and have perhaps an abundance of milk poor in quality and watery; here artificial feeding should be resorted to, at least until conditions improve under the usual ferruginous and other treatment. The occurrence of pregnancy during lactation at once calls for cessation of breast feeding; otherwise the infant will fall away in physical or mental health, or perhaps a miscarriage may be brought on. Regularity of feeding times is, as stated, of great importance, even in the case of the bottle. For the first month the feeds should be given every two hours; from then to three months every two and a half hours, with three-hourly meals thereafter during the first year. It is also important to tell the mother that the child must not be fed every time it cries, it being better to ascertain the cause of the discomfort, which may be not hunger, but colic from wind or cold napkins. Should thirst be suspected, a few sips of cold water may be given. Weekly weighings should be instituted and entered upon a chart or in a "progress-book" such as given away by wholesale vendors of babies' foods. Should the supply

of breast milk become scanty, alternate feeds of cow's milk may be given to make up, or the same may be allowed just before the child is put to the breast; in this way a supply of the natural ferments of the maternal milk is received. Mixed feeding with breast milk and cow's milk is also of great advantage in the case of twins and triplets, the strain on the mother being relieved and breast feeding being possible of practice for a much longer period than otherwise. In order to prevent rickets and other evils, weaning must be performed at the proper time, which is after the ninth month, for which operation the child should be prepared in advance by occasional meals from the bottle, the weaning becoming a gradual process. Sustained loss of weight on the part of the infant calls for discontinuance of the maternal supply, as would, of course, the development of rickets at or after the weaning period. It is also necessary to warn mothers to place no credence in the foolish superstition that prolonged lactation prevents conception; innumerable cases of rickets can, I think, be traced to this cause, as the custom can only result in insufficient nourishment and proteid starvation, which latter is suggested by delayed dentition. In short, prolongation of lactation will lead to starvation of the child and damage to the mother's health, with general upsetting of all the benefits derived from the performance of the natural act during the first year, rickets being as likely to break out in a prolonged-lactation infant as in once brought up on the bottle. During the puerperium the welfare of the infant should receive studied care and the quality of the mother's milk should be submitted to frequent investigation. Both the nurse and the mother should be carefully instructed in the simple truth of infant feeding, as there appears to an alarming amount of ignorance on this subject everywhere, with the result that the infant is given all sorts of unwholesome foods and beverages.

B o t t l e - F e e d i n g

It is an unfortunate fact that the necessity for artificial feeding often arises from disease, poor quality of the maternal supply and it is also practised through laziness or disinclination on the part of the mother. It is therefore the medical man's duty to see that the composition of the artificial product shall approximate the maternal secretion as closely as possible and be so arranged as to agree with the child. Cow's milk is most often used, but in order to deal satisfactorily with it, the child's stomach should be made to undergo some sort of training. In the case of rickets there is an inordinate appetite or an excessive thirst, to indulge which unnecessarily would mean gastric dilatation or chronic dyspepsia. The indication is therefore for small meals at approved intervals, as the digestion is slow and imperfect and the stomach needs its rest. Some people seem to prefer the milk of the goat because that animal is cleaner, its faeces being pellets, it is easy to feed and groom, it is not prone to tuberculosis like the cow, its milk forms a more digestible curd, the fat is milder and it and the casein more closely resemble the same from the human mother than in the case of the cow, the percentage composition of goat's milk being in proteid, sugar and fat respectively 3.8, 4.3 and 5.2. But the disadvantages of goat's milk are that it is not easy always to obtain a satisfactory animal and that the process of feeding on its milk is apt to be too expensive for general application. The milk of the ass is also not without utility as its curd is extremely fine like that of human milk, the fat is easy of digestion and there is a slight laxative effect obtained from its use; its percentage composition is in proteid, sugar and fat respectively 2.7, 5.3 and 1. But it is difficult to obtain, it is suitable only for temporary use and is very expensive. In all these cases the aim must be to obtain a substance which approximates as closely as possible in composition to the human supply; but, though the indication is clear and it is not a difficult matter to modify the milk of the cow, for instance, so as to obtain a liquid with a percentage composition practically identical with that of the human breast and to rearrange the proportions of casein and lactalbumin, very little can be done to alter the nature of the casein curd or the dyspeptic and oily fat. Even the milk of a wet-nurse falls short of that of the child's mother. The whole subject of the

artificial feeding of rickety and breast-deprived cases is one of great theoretical and practical difficulty and likely at times to test the ingenuity of most practitioners, whose set purpose is to obtain a milk with the average percentage composition of proteid 1.5, fat 3.5 and sugar 6.5, with a very faint acidity to phenolphthalein and assured sterility. As each case is a law unto itself, it is advisable to avoid too much chemical refinement; each case on its own merits must be the rule, the amount of food and its character being judged not by the child's age in months, but by its weight, its capabilities and the general condition of its development. Most of the text-books give careful and not infrequently very elaborate directions for feeding; but I prefer to arrange the matter according to the individual patient in terms of my own practical experience, the dietetics of rickets being largely a matter of common sense plus much clinical observation and a dash of theory.

MILK IN RICKETS.

In arranging for the use of milk in artificial feeding it is necessary to bear in mind certain theoretical points regarding its composition. The most important one which comes to mind is the proteid part, as the same has to do with the building up of the tissues and blood, the fat and sugar for proteid economy and the production of heat; only when the latter are insufficient should there occur a drain upon the proteids. The latter substances are the well-known casein and lactalbumin. Maternal milk contains more lactalbumin, which is a more soluble proteid than casein in the proportion of two to one, than the milk of the cow, which contains five times as much casein as lactalbumin in the proportion of five and a half to one. So it comes that the large quantity of the tough indigestible casein is the principal difficulty encountered when the child is fed on cow's milk, especially as rachitic infants sometimes fail to deal satisfactorily with the one and a half per cent. in the human secretion. According to my observation, a child should be able to digest a milk mixture containing one per cent of proteid at the end of the month, one and a half per cent. thereof at the end of three months and two per cent. of the same between the fourth and fifth months; in most of my cases I have not been able to satisfy myself of the perfect digestion of the three and a half percent of cow's milk proteid before the age of one year. In estimating this point my plan is to make frequent examinations of the stools of the patients for colour, consistence and the presence or absence of undigested casein. I have noticed that when the proteids are insufficient the infant suffers from starvation, it does not increase in weight, it sleeps unsatisfactorily, it has cold feet and limbs, the general temperature is below the normal, the stools are green, they contain particles of undigested curd, they are very soft or watery and not infrequently mucus appears therein. On the other hand, when the proteids are too plentiful, the child's digestion is disordered, the temperature is apt to be elevated, colic is suffered from and the stools are watery and curdy. The fat of milk entered into the nutrition of the nervous and osseous systems, it is a producer of heat and economiser of proteids in that important function. The infant requires a larger proportion of fat in its food than the adult, as many statistical and mathematical studies of the heat question have shown. The role of the fat in the nervous and osseous metabolism has been abundantly shown in theory and practice; so that its utility in the treatment of rickets is well established. But I have found that one can exhibit fat in the food unduly, causing the child to become too bulky for age, suffer from actual starvation and have an infinity of gastric derangements. It is important to bear in mind that the fat of cow's milk is rich in fatty acids, the human secretion being comparatively poor in the same; there being about four per cent. in each of these milks, infants can seldom digest that amount in cow's milk, but do better with a per cent. reduction of the latter and some authorities hold that it ought to be halved, and I would add particularly in hot weather. I therefore insist that, in dealing with rickets, it is a bad plan to increase the fat too rapidly, lest the above derangements appear. It is also as well to bear this in mind when exhibiting cod-liver oil for rickets. I find that the best form of fat for rachitic

infants is cream, which, being a fine emulsion of fat, is very easily assimilated and digested. Though the fat allowed must be abundant, it is very necessary that there be also a proper proportion of proteid at the same time; otherwise the general nutrition of the infant will fall off. The carbohydrates of the diet can be changed into fat, if necessary, though the main purpose of the sugar is to spare the proteids and produce heat. It is essential that the carbohydrate for the early months of life shall be in the form of sugar; otherwise it may interfere with the absorption of the fat. If cow's milk is used in the feeding of the infant, it is as well never to give less than five per cent. and seldom more than seven per cent. of sugar. The easiest way to prepare a five per cent. solution of milk sugar is to dissolve one ounce of sugar in boiling water and add to the food as required. The objection to lactose itself is its prohibitive price for the poor; these people use ordinary cane sugar and can therefore do with only half of lactose, though many do without it altogether.

In general, the artificial feeding of rickety cases is conducted upon cow's milk, modifications thereof being, in view of various forms of indigestion readily arising, somewhat ineffective or dangerous. Therefore, in order to secure the best results from bottle-feeding in these cases, it is essential that we are in a position to get a cow's milk that is absolutely pure as nearly so. The milk brought by rail to town is seldom or never pure, so that it becomes necessary to inquire very closely into the nature and source of the supply. Sterilisation may be required. Even if the milk is obtained from the dairy or elsewhere in a satisfactory condition, it is very necessary that all the virtues of the liquid be not upset by careless handling or storing in the patient's home. It should be poured, as soon as received, into a clean scalded utensil, covered up and kept in a cool place away from odours and other forms of contamination. It should never be left in a feeding-bottle and the article supplied by the dairies of towns in airtight containers is the best for the consumers. A certified milk of low bacterial content is much to be desired everywhere and is to be obtained in certain progressive towns, but at an increased price likely to interfere with its general adoption. Milk depots are of French origination and aim at supplying the poorer classes of the community with pure milk on advantageous terms. Consultations are sometimes established at these places, where the milk is modified and arranged to meet the dietetic indications. They are a step in the right direction. They are fully described in works in Infant Feeding and need receive no further notice here.

MILK PREPARATIONS.

Milk has been variously modified, in order to approximate the human breast product as closely as possible, both in the home and by traders. It is just as well to avoid, as a rule, the preparations sold under the misleading name of humanised milk; they may be quite safe and satisfactory sometimes, but are often not quite fresh and, if sent some distance by rail or road, the fat is so much shaken that it not infrequently converted into butter. It is far better to manufacture an imitation at home, which is done by allowing the milk to stand for three or four hours and then remove all the cream. Next divide the milk into two portions and convert a half of it into whey, thereafter adding cream, milk and whey together and a food is obtained with all the fat, but only half the cream of cow's milk, and the soluble proteids. Another way is to dilute cow's milk one-half with whey and add bicarbonate of soda to render alkaline. I have tried humanised milk on numerous occasions, but have found that it fails to meet nutritional requirements if long continued.

Whey is the thin serum of milk after separation of the curd and cream and contains the soluble proteids of milk, and no tough and indigestible casein; it also includes some fat, sugar and water. Its exact composition will depend upon the quality of the milk from which it is prepared, the whole-milk article containing more fat than ~~skim~~-milk whey, but both having about the same proteid figure. Cloudiness indicates the presence of some of the paracasein and a little fat. It is useful in rickets, as it enables us to provide a food containing soluble proteids and only a little casein; it is sometimes referred to under the term "split proteids" and has the additional advantage of containing all the lime and salts of milk. It is not difficult

to prepare, if care be exercised and the milk is heated to 100 degrees F., with subsequent addition of rennet in tablet or liquid form. The milk must now be heated fifty-five degrees more and the curd cut with a knife to allow the whey to escape. It is then ready for straining through muslin and there should be another heating, to destroy the rennet ferments before actual ingestion. One may slowly add to the whey small quantities of mutton-broth, egg-albumin, bread jelly or dextrinised gruels, with cream and whey mixture ultimately. White-wine whey is a good thing for use in cases in which the infant is in a condition of collapse from constant vomiting or profuse diarrhoea; for it is easily digested and a stimulant; it must be given in small quantities and is retained by the most sensitive stomach. It is readily prepared by adding a wineglassful of sherry to a pint of boiling milk, which is then poured into a basin and the whey poured off after sedimentation of the curd.

Pegnin is a milk preparation which contains rennet and sugar mainly and has as its object rendering the curd of milk more digestible. To make it, sterilise the undiluted milk by heat and allow to cool to 104. F. Add one measure of Pegnin to eight ounces of the milk at this temperature, with the result that there is an almost immediate clot, which vigorous shaking soon breaks up. The curd is much softer than ordinary casein curd of milk, which latter becomes more digestible in consequence and may be given pure or diluted with boiling water. I find that this milk preparation is indicated when the general condition is bad after exclusive starch diet, in rickets complicated with gastrointestinal affections and in nausea.

I regard buttermilk with favour in cases of rickets, when used fresh within twenty-four hours after making butter. The latter process the casein is finely divided and the relative proportion to the albumin is lessened and a soft digestible curd is formed. Buttermilk seems to inhibit the action of intestinal germs by virtue of the lactic acid which it contains. It is low in fat, has a moderate amount of sugar and a high proteid. It is useful, therefore, in rickets with gastrointestinal complications, in indigestion about the time of weaning, when fat disagrees and when the casein of milk upsets the stomach. Many authorities taboo its use in rickets, but I have justified in vaunting its exhibition in the kind of cases named. Sugar may be added to it and the so-called babeure formed, which should be fed through a wide nipple on account of the curd. After a time it becomes necessary to add cream in order to provide additional fat.

Ordinary milk may at any time be alkalised by the addition of lime-water or bicarbonate of soda. Lime-water makes the curd of milk more flocculent and better capable of being acted upon by the gastric juice without the formation of tough curds. About a twentieth part should be added to milk of mixtures thereof. Bicarbonate of soda combines with the stomach acids to form carbonic acid gas, which latter penetrates the curd and makes it more porous. It is a more powerful antacid than lime-water and retards curdling of the milk in the stomach. A grain or two should be added to every ounce of milk.

Citrate of soda may be similarly added to milk, with the object of rendering dilution less necessary and concentration of the milk possible, without danger of overfeeding with ordinary milk to get sufficient nourishment. It makes the curd of cow's milk more digestible, a combination occurring between it and ~~caseinogen~~ caseinogen, to form a compound of low molecular weight. Infants who reject cow's milk not infrequently do well on this, the vomiting soon ceasing and stomach regaining its tone.

Acid milk, or lactobacilline, has been much advocated by Metchnikoff and others in the treatment of various gastrointestinal affections arising from fermentation in the alimentary canal. The milk is treated with a special lactic acid bacillus supplied under the above-mentioned trade name. Full directions are given for its preparation. I have given it a trial, but am not convinced that it is the wonderful remedy claimed and that as much good may not be done by simpler and more convenient and less expensive means. I observe that it shows signs of a decreasing popularity.

Kefir is a preparation of fermented milk similar to the foregoing, which is made from goat's milk by the action of germs thereon. It is possessed of stimulating properties, but I have never used it from want of facilities in this quarter and on theoretical grounds because I fail to see that it has an advantages over buttermilk, because it is difficult to obtain and is expensive.

When the rickety patient has been tided over the perils of complicating affections it is sometimes advisable to arrange for milk mixtures to meet indications of age and condition, due regard always being paid to the proportion between the proteid, fat and carbohydrate, as excess of one may interfere with the proper assimilation of the others. The works on infant feeding show how these indications must be met, so that it is unnecessary to recapitulate them here. Whey cream mixture, however, stands out amongst many as providing digestible proteid and a sufficiency of fat. It keeps the caseinogen at a minimum and the lactalbumin at a maximum and so furnishes a moderately strong food and gives better results than from any other methods in which the casein is likely to interfere with the digestion of other elements. I am in favour of the use of twenty per cent. cream, which can be obtained from the top five ounces of a quart of milk that has been allowed to stand for three or four hours., adding white of egg or milk sugar if necessary. It is my custom to use only whey cream mixtures for temporary feeding and to endeavour to reduce as soon as possible the cream by using a larger quantity of top milk, or by adding plain milk to the feed.

Percentage feeding is much practised in America and large cities and towns in various other parts of the world. The idea is to write out a prescription for definite percentages of proteid, fat and sugar and to have all feeds made up at a milk laboratory or large dairy with facilities. This kind of thing is all right amongst the affluent and better-informed classes, but not likely to find favour with the poor, who cannot be bothered to send to the laboratory for a bottle feed each time and who cannot stand the expense. It is therefore necessary to adopt home methods and treat each case on its own merits, with variation of the diet as needed and on the lines laid down above.

Milk may be sterilised or pasteurised according to requirements and kumiss may be given when it can be obtained; it is prepared by lactic and alcoholic fermentation and much used in Russia, where a special strain of mares is kept for the purpose. Their milk is mixed with kumiss ferment and a somewhat complicated procedure observed. The result is very fine division of the casein. The ferment may be purchased at the stores and the article manufactured at home from directions supplied. Matzoon is another acid milk for fermentation cases, but has no advantages over the one just described.

Dyspeptic cases often do well on peptonised milk, prepared by one or other of the liquid pepsins sold by the stores, of which peptogenic milk powder is perhaps the best.

Condensed milk should be avoided in every case of rickets and, indeed, of infant feeding artificially, as it contains too much sugar and when diluted to reduce it there is too little proteid and fat. It is an abomination and itself responsible for innumerable cases of rickets. It suits lazy mothers and may be of use on a journey when cow's milk cannot be obtained. Whenever I come across a case being fed upon it, I at once forbid it under the threat of not having anything to do with the treatment of the child. There are many brands of condensed milk on the market, all of which, I find, can dietetically be assigned to one or other of the convenient and apposite categories of bad, worse and much worse.

GRUELS.

The gruels form a ready and useful means of feeding rachitic cases in general practice when the mother cannot be trusted to prepare whey or similar foods. One of the cheapest and simplest foods of this class is bread-jelly. To prepare, take a slice of bread and steep it over night. In the morning slowly boil in a pint of water for ninety minutes to convert the starch into dextrin and grape sugar. Strain and allow to cool into a fine jelly. Prepare and use twice daily as it soon sours.

This dextrinised gruel is a useful vehicle for the administration of meat-juice or cream; it may also be added to one or other of the broths. It should be given diluted with boiled water to make a food the consistence of thin cream, the proportion being a tablespoonful to eight ounces of sugared water and in this diluted form it contains, approximately three-quarters of a per cent, of proteid, half a per cent. of fat and four and a half per cent. of carbohydrate. Small quantities of peptonised milk may be added according to indications and in general the food is much to be commended. Ordinary dextrinised gruel may be used also as a temporary food or always to dilute the milk. It is prepared by beating up one or two tablespoonfuls of barley, or twice that quantity of rolled oats, into a paste with cold water, adding a quart of boiling water and cooking for about a quarter of an hour. After cooling, add one teaspoonful of malt and allow to stand and settle. It contains about half a per cent. of proteid and from two to four per cent. of soluble carbohydrate. It will often be found that this form of gruel may not be well borne under the age of three months; it is then better to use ordinary boiled water to dilute the milk.

EGG MIXTURES.

The white of egg, diluted to form albumin-water, may prove useful in certain cases which cannot tolerate milk. A little salt may be added and the whole taken as it is or added to a gruel or other food. But what generally suits better is the white of an egg with one or two teaspoonfuls of sugar and eight ounces of dextrinised gruel, the composition of the same being two per cent. of proteid and about five per cent of carbohydrate. If fat is needed and cream is not well borne, the yolk of an egg may be added to the above mixture, in which the fat will amount to one and a half per cent. The mixture may be heated to about one hundred and fifty Fahrenheit degrees without coagulation.

PATENT FOODS.

I do not intend here to enter fully into the composition of proprietary foods, as I dislike them all after many trials and never recommend them in practice, preferring to modify ordinary cow's milk, as already, in cases of intolerance of the maternal supply. Too much reliance should not be placed upon the published analyses, as I find that their composition varies considerably; they are deficient in fat and lime salts and in themselves productive of rickets. They are expensive, the starch is indigestible and there is too much of the latter and sugar. Their free use is apt to cause scruvy and diarrhoea. The children fed upon them sometimes look fat, but are pale, anaemic and flabby; they cut their teeth late and are apt to have debilitating perspirations about the head and elsewhere. Few rachitic children can digest them, even the most scientifically prepared. Not one of them can properly take the place of the mother's milk, particularly as they do not contain the antiscorbutic element. Lazy or clumsy mothers are apt to bungle seriously over their preparation. They are quite unnecessary so long as it is possible to obtain good and fresh cow's milk, cream and sugar and I would sooner retire from a case than allow them to be given before the age of six months, and then only as diluents or during a journey when ordinary milk could not be obtained. Given in small quantities after the age of six months, it would seem that not infrequently they have the utility of educating the stomach to the future digestion of starch. They are prolific causes of dyspepsia and I am convinced that they impoverish the blood. Children reared on these foods have a very poor chance of resisting intercurrent disease and, though they may appear to do well upon them for a time, sooner or later scruvy or other accidents of a nutritional character arise.

(B) During the Second Year

Having conducted the rachitic infant safely to the close of the first twelve months of its life, the proper methods of feeding now become much less difficult to arrange. The artificial foods which perhaps have been used have given a good training for starch digestion, so that a large number of dietetic materials are rendered early available at this time. It is a common experience to find weaning even easier in these bottle babies than in ordinary nurslings at the mother's breast, because of the former having been trained, as it were, to assimilate food additions. At this time it is well to bear in mind that gastric dilatation and gastrointestinal troubles are very common in rickets and to exercise great care not to overload the stomach with too much food, which is often done by mothers in the hope of making up for lost time. Even variety should be kept within reasonable limits, particularly the patent foods, which are far too much used in the home with its cupboards full of tins; the latter should be cleared out and replaced by freshly cooked materials. Mothers will perhaps object on the ground that the proprietary articles are so very convenient, despite the fact that they are somewhat expensive. I find that, with the possible exception of a few of the malted foods on emergency, these patent stuffs are quite unnecessary, it being a simple thing to prepare bread-jelly, barley-and oat-jelly or cream and rice. Herein lies the great advantage that we know what we are using and not open to the objection of staleness or varying composition. All foods containing starch should be steeped for some hours and slowly cooked. A penny cookery manual will give a variety of formulae for selection. In dealing with the food of the second year in rickety cases I am in the habit of studying the patient and arranging the diet to suit, without too much adherence to the hard-and-fast dogmatism of the books. I make milk the staple food and, in order to keep up the standard of the food, also allow meat broths, milk puddings, lightly cooked eggs and custards. Towards the end of the second year I advocate the use of white fish and meat in small quantities, with toast or bread and butter; but I always try to avoid falling into the error of giving too much starchy food at this time of life in rachitic cases. I find it necessary to be very careful in the use of vegetables; potato soup is to be commended and the iron-rich spinach is of great service in rickets, as are also the phosphate-of-lime-containing lentils, beans, calves' brains, sweetbreads and fish roe. It is a good plan also to give the juice of fruits an hour or so before meals. But it is always advisable to be very cautious in increasing the dietary in rickets, it being much better to feel one's way with milk mixtures modified to the existing digestive powers, whey and milk, milk with sodium citrate, rusk, bread-jelly or oatmeal porridge; the latter is especially serviceable because of the proteid and fat which it contains and some variety may be introduced into the diet by the administration of beef-tea, raw meat-juice, pounded meat or some yolk of egg. Wasting cases or thin patients may require extra allowances of cream and butter or even bacon fat. The function of the salivary glands may be stimulated by allowing the child to chew dry rusks or biscuits; with a drink of milk after completion of this salutary masticatory task; in this way, I find, the tendency to flatulence and fermentation, arising from soaked food, is considerably allayed and the general comfort of the patient promoted. Even though the patient is making good progress with the diet, one must never forget the great utility of milk at this time. In all cases sweetmeats are to be forbidden, as well as cakes; for they do harm to the digestion and destroy the appetite for good and wholesome food. The child should not be allowed to partake of the adult meal, or what is going, but have its own meals properly arranged. According to my observations, the test of proper dieting is a healthy-looking complexion, absence of rings around the eyes, cleanliness of tongue and well-digested motions. Not a few of these patients seem to have difficulty in digesting the casein of the milk and so assimilate insufficient proteid; it is then necessary to make up for loss by giving albumin in the form of white of egg with meals, or by adding

raw meat-juice to the feeds, a few drops at a time. I do not favour the use of the so-called peptonoids, as they contain too much alcohol and consider of less value than raw meat-juice such patent stuffs as plasmon, somatose, casumen, etc. Cod-liver, oil, phosphorus and other preparations already described may be allowed to meet special indications. In general, it will be found unnecessary to have recourse to patent articles, as the mother, carefully coached by an expert medical attendant, will learn to rely upon the resources of her kitchen and discontinue to litter her shelves with multitudinous tins of so-called foods.

Taking an all-round view of rickets, there is no getting away from the fact that it is a disease of civilisation and home-life; so that, in attempting to overcome its protean effects, it is necessary that a return to nature - at least as far as possible - should be made, the same comprising fresh air, exercise, proper food and above all, in the case of the infant, breast-feeding. Comparison with zoological specimens will bring out this point, for animals in captivity have their young particularly prone to the disease under consideration, as well as peoples removing from their more or less natural conditions to the crowded parts of cities and towns. It is as true nowadays as ever it was that the simple life for parents and child is the real cure for rickets. In the towns the strain and stress of modern life mitigate against the adoption of so easy and obvious a cure, so that the utility of the medical man in giving advice and warnings is very great; in fact, he should be almost as much a medical officer of health as a physician in these cases and, if he is possessed of a qualification in hygiene, all the better. He will also require to do what he can for the proper arrangement of the mother's welfare during pregnancy, her diet and mode of life - all with a view to the issue of a healthy infant in due course. Her breasts will have to be examined and the scientific reasons for the prime necessity for breast-feeding will have to be explained in the simplest possible language. If it is absolutely necessary to have recourse to bottle-feeding, only the foods which can be prepared at home should be used and the best of these pointed out, particularly milk modifications, whey, soups and gruels. In no case should the mother be allowed to rely upon the advice given by the patent food mengers on the labels of their tins nor should she use samples given to try by tradespeople or sympathising friends. With careful feeding, even the so-called gripe-water, &c., for the relief of pains caused by bad dieting, should not be required. Fresh and clean milk, as a rule, alone will be needed for the infant and preference should be given to the product of dairies which have the cleanliness and control of their animals at heart. I have already expressed my loathing for patent foods and would urge that their use should be prohibited by law, except under the prescription of a duly qualified medical man; they are usually almost as destructive to the health of the child as alcohol is to the adult; to stretch a point for the sake of simile, these liquids are the "beer of infancy" and the bugbear of all conscientious medical men. A practitioner who orders proprietary foods indiscriminately is a real danger to the home, particularly when he changes from one to the other when the child does not thrive in milk mixtures. There is really no need for this kind of thing; for, if a supply of clean milk is available, it is easy enough to give instruction in the preparation of suitable feeds, all the time carefully studying the child's gradual advance in development, teething and so forth. Thus we can safeguard the infant from the multitudinous affections of early life and become of real value in the home. The free appointment by municipalities of inspectors and health visitors is undoubtedly a step in the right direction, though the ratepayers may have something to urge against the same. Breast-feeding should be the rule and, short of this, milk depots and consultations at to the commended and advocated everywhere, so that all classes may obtain a good milk properly compounded to suit the exigencies of the case. In short, the fight against the origination and develop-

ment of rickets must be adopted on the hygienic and dietetic lines which I have laid down; the combat is sure to stubborn and long, but, given proper organisation and concentration of suitable methods of attack, victory will ultimately be achieved.

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CLINICAL EXPERIENCES

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Case 1.

This girl of one year and four weeks appears to have been fed most injudiciously and indiscriminately for five months before being first seen. She then had cut only six teeth and weighed a stone and a quarter. For about eight weeks she had suffered from diarrhoea with frequent slimy, stinking motions. She was very peevish and irritable and seemed to be without energy. All the early signs of rickets were present, such as enlarged anterior fontanelle, prominence of the head, backward dentition, rachitic rosary, spinal curvature, belly protuberance and swollen epiphyses. A dose of castor-oil was at once administered and barley-water was ordered to be given ~~exclusively~~ for the first twenty-four hours. Buttermilk and sugar was prescribed thereafter in small quantities every three hours, with increase on improvement of the digestive disturbances. Marked signs of improvement were observed within four days. The diarrhoea ceased and the motions became quite normal within seven days. The belly became soft and free from flatulent distension and the patient seemed stronger and made frequent attempts to ~~to~~ get up. The daily irritability and nocturnal restlessness disappeared and sound nights' sleep became the rule. The previously sallow and anaemic appearance of the cutaneous surface disappeared and all became pleased and impressed with the bright and healthy tint of the skin. The buttermilk was persevered with for three weeks longer; but as there was not yet any gain in weight, the diet was altered to oatmeal-jelly and milk, with a few rusks to give proper dental exercise. This diet was continued for about twenty-one days, with the result that the patient almost two pounds in weight. The child was now sent away for a change of air to the seaside, where the dietetic and hygienic treatment was continued and I understand that ultimately excellent health was gained.

Case 2.

The mother of this little boy died soon after confinement, so that bottle-feeding had to be practised. Almost at once he began to have diarrhoea and vomiting, with rapid loss of weight. Modified milk and barley-water were ordered and on this he showed signs of improvement, though he did not put on enough flesh and a good deal of subacute dyspepsia persisted. At the age of twelve weeks he developed sweating of the head, restlessness and disinclination for bedclothes, which were kicked off repeatedly. Some swelling of the epiphyses now appeared, as well as beading of the ribs and prominence of the forehead. Anaemia was also observed and small doses of iron failed to relieve it, but upset the stomach. Cod-liver oil was applied to the abdomen on a binder, modified milk for age was given and virol was prescribed. On these substances he did well and ultimately attained perfect health, which was to be expected, as he was most of his time in the open air.

Case 3.

This girl of six months was first seen with severe bronchitis and whooping-cough, which were treated in the usual way successfully. Thereafter head-nodding and ordinary rachitic signs were noticed and doubtless referable to the bottle-feeding being practised. Modified milk was ordered and a change of air advised. The patient went to the seaside and there very satisfactorily progressed to ~~excellent~~ health.

Case 4.

This infant, when first seen, had been vomiting its food for several weeks and had diarrhoea, offensive, ~~stinking~~ containing motions, dorsal spinal kyphosis, head-sweating and widely patent fontanelles. It was under weight. It had been artificially fed on strong milk, cream and water, but was now ordered one part of milk to three parts of water, in order to cope with the fat indigestion from which it apparently suffered. During the next

seven days the vomiting ceased entirely and there was now prescribed a mixture of eight parts of ~~malt~~ twelve of barley-water, with a drachm of malt extract, of which four ounces had to be administered every three hours. Upon this the weight rapidly increased and in four weeks had gained two pounds in weight. The patient passed from observation, but there is every reason to believe that it made a good recovery.

Case 5.

This boy of three months, when first seen, suffered from severe diarrhoea. He had been breast-fed for the first eight weeks of his existence, but thereafter on ~~milk~~ barley-water, and condensed milk. The directions on the label were closely followed by the nurse, with the result that the mixture was far too strong for the child, which at this time weighed just over ten pounds. On reduction of the condensed milk to one part in twenty-four of water the diarrhoea ceased and the patient was at once ordered a weak cow's milk mixture. At the tenth day of the treatment the diarrhoea returned, so it was decided to give a mixture of equal parts of weak mutton-broth and barley-water, in small amounts at frequent intervals, in order to make up for the loss of a pound in weight observed. But the patient now became much worse; several weak mixtures of egg-white, &c., were tried, but all passed through the alimentary tract without being digested properly. During the next fourteen days the illness assumed a dangerous aspect and the patient lay in a sort of comatose condition, with a dry skin and a temperature above the normal. The usual rickety signs were conspicuous and, as a last resort, it was decided to try vegetable broth. This was made with a teaspoonful of finely minced carrot, carrot, turnip, lentil, boiled with lactose in a pint and a half of water down to a third, duly flavoured with salt. The new treatment was commenced with a castor-oil preliminary bowel cleansing. The liquid was well borne and retained, the tendency to fits, previously in evidence, disappeared and the stools, from being green and slimy, took on a better colour and firmer consistence. The future treatment comprised white wine whey for a few weeks and then whey diluted with vegetable broth, later whey only. Some cream was added to the latter, but was rejected by the stomach and therefore discontinued. A mixture of bread-jelly, - half an ounce, - in equal parts of whey and water to eight ounces, duly sweetened and with the addition of a few drops of meat-juice, was well borne and the patient speedily commenced to gain weight. Peptonised milk was added in drachm doses and later boiled milk. Then there was tried a mixture of peptonised milk, bread-jelly, lactose and whey and a slow and steady progress was maintained. The existing anaemia was overcome with small doses of the ammonia-citrate of iron and glycerine extract of bone marrow. Some sweating of the head and nocturnal restlessness was still present and the peptonised milk was replaced with ordinary fresh cow's milk. The patient took a bronchitic attack about this time, but made a good recovery, though the anaemia returned as he was unable to take open air exercise. Fatty food, in the form of cream and cod-liver oil, was rejected by the stomach, so that all attempts to have it assimilate were abandoned. Though the slight ribbeading, the swelling of the epiphyses and the curvature of the shin-bones still remained in evidence and no teeth were cut, the patient seemed to be doing well. Modified milk was given to the end of the twelve months and thereafter the diet was already described for the subsequent period. The teeth were then cut without difficulty and the child made a good recovery.

Case 6.

This patient began to cut his teeth at the age of six months and from that date showed signs of rickets. He was ordered a modified milk and seemed to do well upon it, but at the age of twelve months, after an exciting day and being chilled, there suddenly developed during the night an attack of laryngeal spasm of the usual kind, with which the mother thought the existing constipation has something to do. The crowing was very marked and at times the breathing appeared to be entirely suspended. The chest became fixed, the head was thrown backwards and the pale face soon became cyanotic, with the eyes widely

and fixed in an unpleasing expression. There was also a certain amount of twitching of the facial muscles. It was possible likewise to make out a tendency to opisthotonos, carpedal spasm and general convulsions. The attack lasted less than sixty seconds and during it the child seemed about to die from impaction of the epiglottis. The deepening cyanosis of the face ended in a relaxation of the spasm; the air slowly entered the lungs again through the incompletely relaxed glottis; there was a characteristic prolonged, high-pitched and crowing sound and the attack ended in coughing and crying. The absence of pyrexia, hoarseness and cough after the attack, the suddenness and completeness of the arrest of breathing, the short duration of the paroxysmal ~~shuzure~~, the peculiar cowing inspiration in which it ended and the associated convulsive tendency constituted a characteristic clinical picture which could not but confirm the diagnosis made at the outset. The treatment during the paroxysm consisted in keeping the patient in the sitting position, splashing cold water over the face and naked body and lightly flicking the latter with a wet handkerchief. Smelling-salts were held to the nose. Ice was applied to the pit of the stomach, in order to excite inspiration, and with the same object in view a large sponge moistened with hot water was held against the throat and upper part of the thorax. Whilst all this was being done, a hot bath was got ready and the child was placed therein forthwith, cold water being at the same time applied to the head and neck in a gentle stream. I made an attempt to release the impacted epiglottis by passing my forefinger into the pharynx behind. The occasion did not arise for inhalation of chloroform. The child was given the usual antirachitic treatment according to indications and, as the teeth caused difficulty, the gums were lanced. Every care was taken with the daily action of the bowels and an abundance of fresh air was prescribed. The patient made a good and uneventful recovery.

Case 7.

This female child was first seen at the age of two and a half years. She appears to have been badly brought up and weighed only two stones. The teeth had all been cut, but the upper front ones were ~~entirely~~ decayed, the anterior fontanelle was partially open, the sutures of the head were thickened and this part of the body was of a typically rachitic shape. There were also a deformed thorax, a prominent rickety rosary, ~~Natural~~ sulci, enlarged epiphyses of the limbs and conspicuous backward bending of the spine. There were no signs of true paralysis, but the child could not stand up at all. The belly was vastly distended and tense and tympanitic; the stomach, however, was not dilated and the spleen and liver were not enlarged. There was a very frequent, offensive, slimy diarrhoea and some signs of bronchial catarrh could be made out on the left side of the chest. The complexion was dirty and unhealthy and the cutaneous system seemed to be slack and badly nourished. All the muscles of the body were flabby and thin. A large variety of antirachitic remedies appear to have been tried in this case, but without any appreciable result. The diarrhoea being the most troublesome and debilitating symptom, an attempt was at once made to check it. Metchnikoff's milk was tried, in the hope of the lactic acid bacillus overcoming the mischief. It is pleasing to note that it did good apparently within three days, though it is possible that the mercury and chalk powders administered at the same time had a lot to do with the improvement observed; to test this, the bacillus-milk was stopped after seven days, the grey powders (gr. i) were given every night, with the syrup of the phosphate of iron and belly rubbings of cod-liver oil, with small doses of the emulsion of the latter internally. The improvement continued, the belly became soft and flaccid, the stools became of ordinary appearance and all signs of wind, slime and general offensiveness disappeared. In less than a month the patient was able to sit up and to take a great interest in its surroundings. Ordinary food was gradually introduced and a change of air to the seaside was advised. The improvement appears to have been continuous and uneventful under the new conditions of climate and fresh air.

Case 8.

This boy of six months was fed at the breast up to the age of six months, when his mother died and bottle-feeding had to be introduced. From that time he began to fall away in health and develop rachitic signs, which affection appears to have been more or less of a family one, as he was the fourth or fifth of his strain who had suffered from it. There appeared marked enlargement of the epiphyses and the muscles of the body were flabby and attenuated. Anaemia was present and diarrhoea alternated with constipation. The head was typically enlarged, with sweating and wearing away of the hair behind, the sutures were not properly united and both the fontanelles were patent. Craniotabetic patches were appreciable on the frontal and parietal bones. The patient was ordered grey powder mixed with powdered rose lozenge, cod-liver oil was applied on a binder to the abdomen and fresh cow's milk, duly modified for age, was recommended. The use of patent foods was fought against and a variety of tins were banished from the home. Fresh air and careful handling, with protection from cold and chills were insisted upon, with the result that the child made a very good recovery.

Case 9.

This boy, of the same age as the above, appears to have been fed on the breast for its first three months of life, this natural food being abandoned owing to the failure of the mother's milk. Ordinary cow's milk and selections of patent foods were used, with the result that it did not do well, developing diarrhoea, nocturnal restlessness and perspirations of the head and trunk. When seen for the first time at the age of six months, his face seemed very small in contrast with the enlarged, square-shaped head. He was manifestly anaemic and thin, with soft and flabby muscles and weak extremities and spine. There was the usual rickety rosary at the junction of the ribs with the cartilages, but the rest of the bones of the trunk did not seem to be involved. The same treatment, adapted for age, was given as in the previous case, with particular regard to rest, exercise and hygienic arrangements. A perfectly satisfactory recovery ensued.

Case 10.

This condensed-milk and patent-food-fed girl, when first seen at the end of the ninth month of her existence, was typically rachitic and had diarrhoea and bending of the bones. The usual remedies were prescribed; but one night, after an exciting day and a dietetic indiscretion on the part of the easy-going mother, general convulsions occurred, with particular location on the right side of the body and fixation of the eyes. The spasms were at first tonic, becoming clonic in a short space of time. This condition of affairs showed alternation and a tendency to persist and occasion dangerous exhaustion; so that a hot bath was given forthwith. This failed to cure the condition, so an occasional inhalation of chloroform was administered during the space of one hour, when the fits entirely disappeared. As they seemed likely to return the next day, a full dose of castor-oil was given and a mixture of dose-for-age chloral and digitalis was prescribed for twenty-four hours, followed by potassium bromide for three. The usual antirachitic measures were adopted and the patient seemed to do well thereon. There was no return of the convulsions and a satisfactory improvement was maintained.

Case 11.

This boy of sixteen had always been delicate, but never troubled, it is said, with the rickets. He was tall and ungainly and at the time of puberty when first seen. He was undoubtedly anaemic, had a few râles at the top of the left lung, ascribed to cold, and had growing pains and a poor appetite. No signs of tuberculosis could be made out and, as there was enlargement of the epiphyses, difficulty in walking and general muscular weakness, incipient late rickets was diagnosed and treatment with emulsion of cod-liver oil and general tonics, as well as fresh air and virol, was advised. He was given plenty of rest, being wheeled about in a bath chair for three months. He practically lived in the open air and, improvement being maintained, he was sent away to the seaside, where he ultimately recovered.

Case 12.

This girl had been breast-fed to the age of six months, when the lazy mother decided to have greater liberty and wean. Undiluted cow's milk and a proprietary preparation were tried, with the result that rickets and diarrhoea soon made their appearance. The patient has been a particularly lively and happy infant, but now became irritable and restless, crying whenever handled, resenting attention of any kind; it seemed to do little else but cry when awake and was a real source of disturbance to the neighbours. One afternoon it was out a longer time than usual and contracted a chill, with the result that a smart attack of diarrhoea appeared and the epiphyses of the limbs underwent enlargement. Anaemia and muscular weakness and flabbiness followed in the course of a fortnight. Then haemorrhagic spots were seen to develop on the shins and gums, which assumed a scurvy-like appearance. Then it was seen for the first time. Patent foods were forthwith cleared out of the house and a properly modified cow's milk was ordered, with vegetable soup and weak lime-juice. The hygiene of the case was properly arranged and the patient recovered, under the usual tonics and cod-liver oil, in due course.

Case 13.

This male child was two months younger than the above when first seen, and made a good recovery upon much the same kind of treatment. It had been a physical failure from birth, because of the patent-food nourishment which it had received through the mother losing her milk, she being alcoholic and of an alcoholic stock. The child was not very thin, but was extremely weak in the muscles and there was much vomiting, purgation and flatulence. There was much sweating of the head at night, but the bones of the body were not markedly affected. Grey powder was prescribed in this case, as it was done in all others ~~of the same~~ age and cod-liver oil was applied to the belly. The patient seemed to do well for six weeks on this and hygienic treatment, but one day had much indigestion and a fit at night. The bowels were at once cleared out and a hot bath given, with the result that danger to life was averted. The next day the epiphyses were seen to be markedly enlarged and much restlessness and night-sweating ensued. It was decided to send the patient to the seaside for several months and from time to time good reports of his progress to health were received.

Case 14.

The mother of this twelve-month patient was a humpback dwarf and alcoholic. The patient was seen for diarrhoea and enquiry elicited the presence of head-sweating and nocturnal restlessness and debility. Rickets was suspected and proper diet for the second year instituted. At the age of one year and nine months the patient commenced to walk, with the result that the bones of the legs showed signs of bending under the strain. The following month the first tooth was cut, with much general irritability, diarrhoea and a temporary general eruption on the skin of the abdomen and chest. Fresh air was insisted upon, with the usual tonics, and the patient made a good recovery.

Case 15.

This patient, also a girl, was six months older than the above when first seen for enlargement of the epiphyses of the wrists, rachitic rosary and swelling of the abdomen, with bronchial catarrh resisting household remedies. The home-prepared cow's milk mixture and patient food were abandoned and proper diet for age instituted, together with syrup of squills and tincture of strophanthus for the cough. The patient appeared to do well under this and the usual hygienic measures, but after ~~some~~ six weeks of supervision passed from notice.

Case 16.

This patient, a boy, had just entered upon the eleventh month of his existence when first seen. He had been brought up upon the bottle and was troubled with head perspiration, enlargement of the belly, Harrison's groove, enlargement of the epiphyses, rickety rosary and diarrhoea. The head appeared to be much enlarged in contrast to the puny face and the complexion was

very sallow. The muscles were very weak and flabby and there were no signs of commencing dentition. Cod-liver oil was ordered to be rubbed into the belly, a suitable milk mixture for age was prescribed and an abundance of fresh air was advised. The patient did well and two months later virol was introduced into the diet on which a marked improvement, culminating in perfect recovery six months later, was observed.

Case 17.

This boy of eighteen months had been brought upon the bottle from birth, owing to the death of his mother from a puerperal disease. The diet, which consisted of diluted milk in increasing dose, seemed to have suited to a certain extent; but not entirely, as when first seen at the above age he could not walk and showed bending of the bones under his weight when crawling about the floor. He had insomnia, sweating of his enlarged head, diarrhoea and a "pot-belly," as well as enlargement of the wrists and beading of the lower ribs with lateral grooving. He was prescribed the usual hygiene and diet for the second year as already described, particularly as regards proteids and fat. He made a good recovery eventually.

Case 18.

This girl was first seen when twelve months of age and she appears to have inherited some syphilitic taint, as she had cleft palate and her mother, a humpback, admitted to syphilis early in her married life. All the bones of the body at this time of first inspection were weak and there was much night-sweating about the head and trunk. The epiphyses were enlarged and there was some beading of the chest. The patient had been weaned at the age of nine months and thereafter dieted with "anything that was going." This being apparently the source of the rachitic mischief, the intelligence of the mother was earnestly appealed to; she faithfully observed all dietetic and hygienic instructions and the result to the child was ultimately of the happiest description.

Case 19.

This girl first came under observation at the age of five. She appears to have been reared upon condensed milk for the most part and had suffered from various childhood ailments, such as measles, scarlet fever and whooping-cough. Walking was delayed to the end of the ~~third~~ year and at that time the spine began to bend. She was markedly dwarfed in stature, but thick-set and had to wear long dresses to hide her bandy legs. She was weak in the mind and slubbered a good deal. Residence in a charitable institution was recommended and the case is cited to bring out the evil of condensed milk and unsuitable food-stuffs at a tender age.

Case 20.

This boy, at the half-time of his first year of age, seems to have been born with a tendency to tuberculosis, his father having died of that dread disease when the patient was three months old and his mother ultimately succumbed to it, it appeared later. The rest of the family, which was a large one, were very delicate and frequently ailing. The boy had been fed from birth, owing to the delicacy of his mother, on cow's milk alternated and combined with patent foods, with the result that he became fat and flabby, restless at night and very irritable and weepy during the day. He had an attack of convulsions, for which he was first seen. It was relieved by the usual hot-water bathing and bromide and an antirachitic diet was prescribed, consisting of modified cow's milk, grey powder and cod-liver oil belly binders. The patient did fairly well for the next three months, but had a decided tendency to fits ~~with~~ dentition. Then he did very well with the increased diet which his greater age warranted and the rest of the teeth were cut without difficulty. He was given an abundance of fresh air and at the turn of the second year he was given small doses of virol, with cod-liver oil emulsion when he was two. He passed from notice soon after, but there seems no doubt that he made a good recovery in the district to which he went to reside.

Case 21.

This girl had been reared upon the breast of a very delicate mother to the age of eleven months, in the hope of preventing pregnancy, when he ~~was~~ under observation for the first time for general backwardness. Suitable diet was recommended, but not adopted by the mother, who had heard of some wonderful patent food and determined to give it a trial. The result was that the child became even more restless, sweated about the head at night and developed a papular eruption about the face. The child was again seen and found to have some beading of the ribs, enlargement of the wrists and tenderness of the bones on pressure. Diarrhoea was frequent, malodorous and slimy, with admixture of green indicative of fermentation processes within the belly, which had assumed the characteristic protuberance of rickets. The patient was two pounds under weight for age. Proper diet and hygiene were instituted, particular emphasis being laid upon the efficacy of raw-meat-juice. The result was of the happiest description and perfect recovery was in due course achieved.

Case 22.

This boy of twelve months appears to have been brought up from birth on badly adapted cow's milk and proprietary foods. He was of large size, but without strength and stamina when first seen; he was typically rachitic in spite of his massive build. Diarrhoea was particularly troublesome, his food coming through him soon after ingestion, for which he was given still more by the mother and the belly assumed a tremendous size and was drum-like in tightness. He was ordered cow's milk, duly modified for age, together with abundant fresh air, quiet and mercury and chalk, plus Dover's powder, of each half a grain, a tendency to convulsions being feared. One night, three months later, he was out late and contracted a chill. Bronchopneumonia developed, which failed to respond to the classical treatment of that disease and he died upon the second day. A post-mortem examination was not allowed.

Case 23.

This girl of one year and two months had been properly fed upon the mother's milk up to the age of a year, when he was allowed all sorts of things from the family table, the same culminating in an acute attack of indigestion, which only very slowly yielded to bismuth and mineral acids in alternation. It was now noticed that the head seemed unduly large in proportion to the face and that there was enlargement of the epiphyses, beading of the ribs and pot-belly, with patency of the anterior fontanelle. The usual antirachitic measures were instituted, after a month of which the patient contracted a chill and developed bronchitis, which, however, yielded to the usual remedies in the course of a week. From now the patient made a good recovery.

Case 24.

This patient, a girl of ten months when first seen, had been brought up on cow's milk and patent foods from birth, the mother being in too delicate health to undertake breast-feeding. Rickets developed at the age of six months and showed signs of being of a severe character. There was much beading of the ribs and swelling of the wrists, together with enlargement of the head and perspirations at the back of the same. The nights were very restless and the days marked by great irritability. The patient was put upon a duly modified milk and grey powder, but the mother becoming careless constipation commenced to alternate with diarrhoea and, at the end of a fortnight of this, general convulsions occurred late in the evening after an exciting day. This was treated in the usual way successfully and the next thing which occurred, almost immediately, was an attack of bronchitis, which required a fortnight of assiduous medication to overcome. Then a minor form of pertussis appeared, on the seventh day of which the patient took a general convulsion and died. A post-mortem examination was not allowed.

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