A Critical Study of Rickets with special reference to its treatment by "Artificial Sunlight" in a Scottish Industrial Area.

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1 Introductory

The Modern Significance of Rickets

"Rickets" wrote Osler in 1910, "is a disease of infants characterised by impaired nutrition of the whole body and alterations of growing bones".

The definition is wide enough to be still acceptable to-day, but, on the other hand, how vastly altered from that time is our conception of the disease, and how more precise our knowledge of its causes, its treatment, and what is involved in its prevention. From being a disease of purely medical interest it has now become one of the great social problems of the day, a problem in which nothing less than the physique of the nation is involved. The reproach of being labelled a C 3 nation is known to be due largely to the effects of this disease. "Rickets" said a modern writer (Lawson Dick 1922) "is the commonest disease of children in our large towns, and probably the most serious factor interfering with the efficiency of the nation. Rickets is the sum of the penalties which the young pay for the unnatural conditions under which they live ---------- . The retardation of growth and deviations in development of the adult of forty are commonly the result of the evil influences which bear so heavily on the young infant in the formative period of its existence. - - - - - Rickets stunts the growth of the body; retards the development of the brain; interferes with child-bearing; predisposes to bronchitis and enteritis, and increases mortality from measles and whooping cough. It produces the ineffective type of adult deficient in physical stamina and in nervous energy and stability".

Later work takes us even further than this in the indictment against rickets. For example, M. Mellanby has shown that in a certain series fully 80% of deciduous teeth in industrial districts were, on histological examination, found to be of defective structure and calcification; while it is also well known that about the same proportion of childrens' teeth are subject to caries. That is, deficiency of calcification, a process which is essentially of the same nature as rickets is very completely correlated with the serious national menace of dental caries. Clinical observation in the writer's experience tends strongly to confirm this view, and also th show that the sequlae of dental caries e.g. oral sepsis, enlarged tonsils and adenoids, middle ear disease, nasal obstruction, maldevelopment of chest, bronchitis and pneumonia, gastritis and enteritis, and sometimes even tuberculosis - in a word, the characteristics of the "catarrhal child" are all commonly associated with a degree of rickets. There is also direct evidence (p. 40) that the condition of enlarged tonsils and adenoids in children is very frequently accompanied by signs of rickets. Lastly, the writer has noted in more than one epidemic of measles in industrial districts that children who succumb to broncho-pneumonia are almost always rachitic, and again that deaths from diarrhoea and enteritis in children under 2 years of age occur most frequently in rachitic subjects.

The amount of rickets in our industrial populations is stated to have declined in recent years. This is probably correct since in 1910 Osler stated that 50% to 80% of all children in the clinics of London and Vienna present signs of rickets, while Findlay estimated that 50% of all Glasgow children had rickets.

The evidence presented in this paper on the incidence of rickets in a typical mining and steel manufacturing centre in the Lancashire coal fields, nevertheless shows how extensive the disease still remains. Certainly not less than 40% of such children between the ages of 6 months and 3 years now present signs of the disease.

Great strides have been made in our knowledge of the part played by unhygienic conditions in the production of rickets, particularly those of confinement in sunless or smoke-polluted atmospheres and of food deficient in vitamin content. The long list of causative factors which used to be given now tends to be reduced to the two mentioned, and of those, chief emphasis is laid on lack of sunlight. Rollier speaks of rickets as "La maladie de 'l'ombre par excellence". There is a distinct modern tendency to classify rickets as a disease of darkness, although as will be shown in these pages, many minor factors besides lack of light may exert an influence on the net result.

Thus rickets has broadened into a communal problem - the problem of bringing the bodies of city children into contact with sunlight or its effects, i.e. an affair of slum eradication, smoke abatement, provision of open spaces, transport, food reform, individual education in mothercraft &c &c. Pending these developments the provision of Artificial Sunlight Clinics for the irradiation of rachitic children with ultra violet light is an immediately practicable measure, the value of which will, it is hoped, be demonstrated in this work.

An attempt will here be made to give a concise account of the history and development of modern ideas on rickets, to review the factors which enter into the present conception of the disease, and to present first-hand evidence as to the value of treatment by ultra-violet light of children in an industrial district suffering from the disease.

2 Historical Outline

Rickets in Ancient Times

L. Findlay has recently brought to light a most interesting reference which indicates a high prevalence of rickets in Ancient Rome in the time of the Emperor Hadrian.

Soranus Ephesius (98 to 138 A.D.) a physician of Rome, in a work on "Diseases of Women" gave his reasons "why the majority of Roman children are distorted". "This is observed to happen" he says "more in the neighbourhood of Rome than in other places. If no one oversees the infant's movements his limbs do, in the generality of cases, become twisted, for the whole weight of the body rests on the legs, and the floor or pavement on which he walks is hard and unyielding, being, for the most part, laid with stones. The limbs must then of necessity give way a little since the bones are not yet stiff. Hence when he first begins to sit he must be propped up by swathings of bandages to counterbalance the ills that can gain the mastery over him, nor must he sit for long at first".

This important record undoubtedly shows that rickets was exceedingly common in the city of Rome at this period. Normal infants are not distorted by efforts in walking and there is no n eed to wrap the normal spine in "swathings of bandages", although such a practice is widespread even to-day. Soranus has, however, given good advice according to his lights for securing the best results with the rachitic bodies of these Roman infants. He did not know, as we do to-day, that the best way to strengthen such limbs is not by restraint or coercion from without (although that will modify the deformity) but by nutrition from within - by stimulation of calcium metabolism, from exposure to sunlight &c. The real answer to Soranus' question "why the majority of Roman children are distorted" is that they were rachitic. Andif we wish to enquire further why they were rachitic, we must consider the state of things in Rome during this age of the Antonines. Says Gibbon, writing of the period, "Under the Roman Empire the labour of an industrious and ingenious people was variously but

incessantly employed in the service of the rich; and H. G. Wells, "it was an age of commercialism in which the rich grew richer and the poor poorer". Is there not sufficient evidence here of rickets appearing perhaps for the first time on a great scale, as "that ugly scandal of malurbanisation". Rome had become comparable to the modern industrial area in respect of the unhygienic conditions under which the common people lived.

In contrast with this record of the most highly urbanised population of the ancient world, is the record of archaeological work in Egypt and in Ancient America. Elliott Smith states that "clear unmistakeable evidence of rachitic changes has not yet been found in human bones in any ancient cemetery in Egypt or Nubia". As regards the inhabitants of Ancient America, Lawson Dick concluded after reviewing the literature that rickets is not indigenous to that country; that ancient graves in California, Peru &c. show no sign of the disease, and that it has probably developed with industrialism.

Glisson describes rickets A.D. 1650.

The first monumental work on Rickets as a clinical entity is a book published by Francis Glisson in 1650 entitled "De Rachitide, sive morbo puerili qui vulgo "The Rickets' dicitur". He relates: "This is an absolutely new Disease and never described by any of the ancient or modern Writers in their **practical works which are** extant to this day of the Diseases of Infants.

"But this Disease became known (as near as we could gather from the relation of others after a sedulous enquiry) about 30 years since, in the countries of Somerset and Dorset; since which time the observations of it hath been divined into other places, as London, Oxford, Cambridge, and also all the Southern and Western parts of the kingdom: in the Northern counties this effect is very rarely seen, and scarcely yet made known among the Vulgar sort of people". As has been pointed out, the 30 years probably means no more than that is the average duration of human memory. The disease again appears to have

been observed chiefly in the larger centres of population, which in these days were in the South and West. There is some evidence that in Germany at this period, it was known as "The English disease of Children".

Glisson described as signs of rickets, the large size of the head, the wasting of the external members and muscles of the whole body, the thickness of the ends of the long bones (certain protuberances, which are seen to arise - not in the fleshy and membranous parts, but in the ends of the bones themselves, especially in their epiphyses".)flaccidity of the limbs, deformity of the chest ("the chest externally is thin and much narrowed, especially beneath the scapulae, as though compressed from the sides, and the sternum accuminated like the heel of a ship or the breast of a fowl"). He describes also the rosary ("the ends of the ribs which join with the cartilages of the sternum are nodular, like the ends of the wrists and ankles") and the protuberant abdomen. The morbid anatomy included the increased size of the liver, the normal condition of the spleen, along with gaseous distension of the gastro-intestinal tract, slight enlargement of the mesenteric glands, and normal condition of the urinary tract. In his description of the thoracic contents, he mentions pleural adhesions, pneumonia, empyema, and phthisis, but he considers them rather complications. As to prognosis, he states that "this disease in its own nature is not fatal". It is interesting to note that Glisson attributed rickets to disturbed nutrition by arterial blood, and believed the changes in the long bones to be due to excessive vascularity.

Following upon Glisson's epoch-making work, no great progress appears to have been made in adding to our knowledge of the disease until the latter part of the nineteenth century. It was then that various workers particularly in Germany and Austria began to make detailed studies of the histological structure of rachitic bone, and of the chemistry of the bones

and blood with reference to calcium and phosphorus content.

Kassowitz (Wiener Med. Jahrb. 1879-1884. Outlined by Osler p.427) stressed the importance of the hyperaemia of the periosteum, the marrow, the cartilage, and of the bone itself, and regarded it as the primary lesion, out of which all the others arise. He considered that this disturbed the development of the growing bone and excited changes in that already formed. The cartilage cells in consequence proliferated and the matrix was softer.

Thereafter came rapidly the beginnings of modern scientific observations.

3 Early Scientific Work

Chemical Pathological and Histological Studies of Rachitic Bone

The chemical composition of the skeleton in rickets was investigated by Marchand (1842) Brubacher (1890) Schabad (1910) and others.

Brubacher found as a typical example, that the femur of a child aged 4 years contained only 2.74% of oxide of calcium as compared with 11.00% in a normal control: and phosphorus pentoxide was similarly reduced in the same case from 8.58% (normal) to 1;97%. The fat also was considerably reduced and the water increased.

Telfer showed that in rachitic pups the defect of mineral matter was extreme being 17.7% of the dried limb bones compared with 44.9% in the normal.

The ratio of Ca O to P_2O_5, however, even in the most pronounced cases, does not differ from that of normal bone.

Pommer (1885) was the first to work out the pathology of rachitic bones. His work was confirmed and amplified by Schmorl (1906-13) and others. The essential pathological change in rachitic bones was found to be a cessation or retardation of deposition of calcium in the newly-forming bone or proliferous cartilage, i.e. underneath the periosteum and at the epiphyseal junctions. When such bones were examined histologically, the picture was one of imperfectly calcified bone or <u>osteoid tissue</u> in the same situations. This is the essential histological change in active rickets. Schmorl found, moreover, that the amount of osteoid tissue so formed varied chiefly according to two conditions:-

(a) the rapidity of growth of the individual, and

(b) the amount of weight and pressure to which the skeleton was subjected by gravity and by tension of tendons and muscles.

The greatest amount of osteoid is formed in rapidly

growing rachitic infants who are much on their feet or use their limbs actively. (N.B. It is in these that the diagnosis can most easily be made by X-Rays, as the large amount of osteoid can be recognised in the films), On the other hand, infants exhibiting poor growth and nutrition may have a minimal amount of osteoid. (N.B. This may be almost invisible in X-Ray films). Such a condition has been named by Korenchevsky "the latent rickets of atrophic children". In such cases the diagnosis, apart from clinical means, could only certainly be established by biochemical tests for calcium retention, the calcium excreted being found to be nearly equal to or even greater than the calcium intake, (low positive or negative calcium balance).

"Incipient" Rickets.

Schabad (1910) on the basis of biochemical tests comparing intake and output of calcium showed that (a) in the early progressive stage of rickets there is invariably a negative calcium balance. (N.B. This was criticised by Telfer (1922) who found merely a low positive balance in the active stage). (b) during the period of markedly manifested rickets, but with the cessation of the process of softening of the bones, the calcium balance is zero or slightly positive, and (c) during the period of convalescence or healing of rickets, the calcium balance is markedly positive. The matter is, however, complicated by the fact pointed out by Orgler (1911) that rickets may occur in a series of waves alternating between the active and healing stages, thus making the reading of results sometimes difficult in a continuous series of observations. Birk and Orgler (1910) and Holt, Courtney and Fales (1920) found a negative calcium balance in certain apparently normal infants. It was subsequently shown that such infants are in the stage of incipient rickets, and that the usual rachitic changes in the bones are found to occur after several weeks or months.

Considerable progress was thus made by the earlier scientific workers in elucidating the essential chemical and histological changes in the bones in rickets, and in differentiating the disease into its incipient or latent, its active, and its healing stages. Biochemical work has been continued in recent years and is still proceeding actively.

Modern Investigations.

(a) Biochemical

Rickets from calcium starvation (pseudorickets).

Schabad (1910) and Orgler (1911) made the observation that "an increased calcium intake in the diet of rachitic children at the progressive stage does not cause any increased retention of calcium".

This principle which is generally true was shown not to be true invariably by Grosser (1920) who found that subcutaneous injections of Calcium Glycerophosphate could produce a favourable effect on calcium metabolism in such infants.

The explanation of this discrepancy appears to be that a certain form of rickets (pseudorickets) is due to insufficient intake of calcium in the diet, and this form of course, would be benefitted by the introduction of calcium into the organism. Thus Telfer showed that rickets in dogs appeared on a Ca - poor P - rick diet and that it could be cured by increasing the Ca of the diet so as to obtain the Ca: P ratio of the maternal milk of the animal.

That the matter is of practical interest in human rickets was shown by Korenchevsky who pointed out that:-

(1) Normal human breast-milk contains no excess above the minimum amount of calcium which the child requires during the first few months of life,

(2) the milk from mothers of rickety children seems to be poorer in Ca. than that of mothers of normal children.

(3) a normal amount of Ca in the milk may prove insufficient for the child if its growth is great on account of overfeeding.

(4) Cow's milk contains a greater amount of Ca. than human, but dilution of the milk and bad absorption of calcium from it combine to make Ca starvation possible when feeding with

cow's milk,

(5) digestive disturbances may also affect the absorption of Ca.

(6) Therefore calcium starvation may play a part in the aetiology of human rickets or a special form of it (pseudorickets).

It appears, therefore, that calcium deficiency in the diet is a possible subsidiary cause of human rickets and that, therefore, Ca. medication may be occasionally beneficial. In practice, however, Ca is rarely found to be of much use. As will be shown later, Orr and his co-workers proved in growing pigs, and in lactating goats that Ca. absorption on a Ca. - deficient diet was greatly improved by irradiation of the animal with ultra-violet light. It is most probable therefore that one of the actions of ultra-violet radiation (u.v.r.) in human rickets is to cause this increased retention of Ca. and so diminish or abolish that part of the disease which may be due to pseudorickets.

Mechanism of absorption of Ca and P.

Many biochemical investigations on the absorption of calcium and phosphorus both in infants and in experimental animals have been recorded in recent years. Owing to the diversity of conditions under which such experiments have been conducted, results are, in some cases, apparently contradictory. In any case it is not always easy to ascertain to what extent the facts elicited have a practical bearing upon the actual disease, rickets, as it is found amongst the children of industrial areas.

The following examples of such work are recorded, however, as being of interest.

Seeman (1879) and Zander (1881) started with experiments of Bunge (1873) which showed that a surplus of Potassium salts in the food led to a deficiency of Sodium Chloride in the organism. Seeman pointed out that this leads to hypochlorydria in the stomach and therefore poor absorption of Calcium as Ca. Cl_2. He considered that rickets might be caused by deficient absorption of lime from the stomach in this way.

Telfer (1924) discussed this theory and showed by experiments on dogs that:-

(a) the absorption of calcium is initially dependent on the free acid of the gastric juice, which plays an important part in dissolving the calcium salts of the diet.

(b) absorption is normally restricted by the alkaline reaction of the intestinal secretions which tend to neutralise and so cause precipitation of the dissolved lime as insoluble phosphate.

(c) the free absorption of calcium may be limited to a comparatively small portion of the upper part of the intestinal tract while still in acid solution. To ensure adequate absorption an excess of calcium in the diet considerably greater than requirements is necessary.

(d) the fixation of phosphorus in the skeleton is dependent on that of calcium, and in a diet unbalanced with regard to the mineral elements, the absorption of an excess of phosphorus is followed by its excretion in the urine.

Telfer had previously shown (1922)that while the deficiency of mineral matter in the bones of rachitic pups may be extreme, the ratio of Ca O to P_2O_5 even in the most pronounced cases does not appreciably differ from the normal.

This work then again seems to indicate the necessity for an adequate allowance of calcium in the diet; that retention of phosphorus in the bone depends upon that of calcium and that a well balanced diet as regards mineral content is important for retention of both.

It may be noted here that Orr and his co-workers, who have been inclined to dispute the existence of vitamines have recently also shown the importance of well-balanced mineral content of the diet, but that irradiation with ultra violet light causes an increased absorption of Ca in Ca deficient diets and so reduces this disadvantage to a minimum.

Webster, moreover, has demonstrated the complementary fact that a minimal amount of phosphate in a deficient diet is utilised when radiation is given and is wasted in the faeces when there is no radiation.

J.M.Henderson (1925) in young pigs on a badly balanced diet, showed that the calcium and phosphorus in the urine became increased after irradiation with the carbon-arc lamp, thus suggesting an increased absorption from the gut or a decreased excretion. In pigs on a satisfactory, well-balanced diet, darkness, diffused light and irradiation caused little difference in the calcium and phosphorus retention, though such difference as there was, was in favour of the irradiated pig.

It appears, therefore, to be evident that calcium and phosphorus absorption is influenced by ultra-violet light irradiation most particularly where the diet is badly balanced in these minerals.

Calcium and Phosphorus in the blood in rickets.

Both the whole blood and the blood serum have been examined by many workers whose results, on the whole, are somewhat inconclusive, as the stage of rickets at which the examinations were made is not known. Generally a normal or somewhat reduced content of Ca in the blood has been found.

Howland and Kramer (1921) made the following important observations: "During the active stage of rickets the Ca concentration in the blood may be normal or slightly reduced. There are reasons for believing that this reduction is associated with a latent form of tetany. The inorganic P. of the serum is reduced in active rickets, sometimes to an extreme degree. During the process of healing, whether occurring spontaneously or as the result of the administration of cod liver oil, the phosphorus content of the serum gradually rises to a normal figure and often somewhat above this. Relapses are accompanied by a fall in the phosphorus concentration of the serum". These authors believe that the failure of Ca. deposition in the bones in rickets is associated with the low phosphorus content in the blood.

Wyman and Weymuller (1924) and others have confirmed these results as to the low serum P. in active rickets and low serum Ca in tetany and spasmophilia. The ca content of normal serum should be about 10 mgm per 100 c.c. and of inorganic P. about 6mgm per 100 c.c. (Howland & Kramer 1921) In active rickets the product of the two may not exceed 30 mgm per 100 c.c. an amount which the above and other workers in U.S.A. have shown to increase rapidly under ultra-violet light irradiation.

The acidosis theory of the production of Rickets.

This theory which has been vigorously championed by E. Pritchard depends not so much upon the results of actual biochemical work as upon pure reasoning, from the facts of physiology.

It is argued that all the various dietetic and environmental errors, including lack of sunlight, which have been blamed as causes of rickets inevitably conduce to the development of an acidosis which is compensated primarily by the use of Ca Na, K and NH_3 and other alkaline reserves of the blood, and secondarily by certain Ca salts, e.g. Ca CO_3. This prevents Ca being available in proper quantity for building into the bones.

The acidosis depends upon an incomplete oxidation of the food to the normal end products CO_2, H_2O and urea, thus leading to the production of large acid molecules. This condition can be ameliorated by any stimulant of metabolism e.g. exposure to ultra-violet irradiation, to cold moving air, exercise, physiological stimuli etc. On the other hand, the condition can be aggravated by depressants of metabolism e.g. heat, moisture, air stagnation, darkness, excessive sleep, confinement etc.

The active disease rickets is thus preceded by the status calciprivus, (or latent rickets) in which a child may exhibit symptoms of mineral depletion without showing rachitic changes in the bones.

This is a very interesting and instructive attempt to explain the chemical process by which the failure of Ca, deposition in the growing bones, the essential process of rickets, comes about. It has a strong appeal to those who have worked with the rachitic children in industrial districts and who realise that the depressants of metabolism there encountered by these children e.g. overclothing, living in stagnant over-heated, over-crowded atmospheres etc., can hardly fail to exert an influence in producing the disease.

The proof of the theory would be exceedingly difficult, and has not yet been established, although there is some evidence that during active rickets the amount of calcium "available" for neutralisation of acid bodies in the blood is reduced. (Gyorgy)

(b) Distribution and Incidence of Rickets.

Valuable information as to the nature of rickets has been obtained by study of the geographical distribution of the disease, and its incidence in certain populations.

Geographical Distribution.

The Collective Investigation Committee of the British Medical Association published in Jan. 1889 a report upon the geographical distribution of rickets within the British Islands. Two tolerably definite principles of distribution are mentioned

(1) its great frequency in large towns and thickly peopled districts, especially in the region of the coalfields, and its comparative rarity in rural districts

and(2) the greater tendency to rickets in the rural parts of the South of Britain than in those of the north.

In the towns the prevalence of rickets was found to be in direct proportion to the massing together of the population, and there were only eleven towns of over 30,000 population in which rickets was said to be not common, and three of these were health resorts. As regards the rural districts, the wet nature of the soil with much mist and rain in the air seems to be the factor chiefly associated with the disease.

Palm in 1890 recorded how struck he was with the absence of rickets amongst the native population of Japan as compared with the high frequency of the disease in his practice among the poor children in the large cities of England and Scotland. He pointed out that the great difference between these countries as regards climate is the want of sunshine and dull gray skies and frequent fogs in Britain. "This is most intensified in our towns, which are under a perennial pall of smoke, and in which the high houses cut off from narrow streets a large proportion of the rays which struggle through the gloom. It is in the narrow alleys, the haunts and playgrounds of the children of the poor, that this exclusion of sunlight is at its worst, and it is there that the victims of

rickets are to be found in abundance".

This wonderful flash of insight into the nature of the disease was apparently quite neglected for about a generation.

Lawson Dick (1922) collated the literature of the geographical distribution of rickets. He found that rickets to-day was a disease of the northern hemisphere in Europe and N. America existing in the belt between Lat. 42 and 60 N. Outside this area, both in the Arctic and tropical zones, and even in the corresponding southern latitudes which include S. Island of New Zealand, Tasmania and Patagonia, rickets is very scarce. The sole exception is in certain parts of Australia where the disease is incipient and mild.

~~The~~ The affected zone in Europe reaches from the middle of Italy to the North of Scotland. Its chief features may be said to be:

(1) It is the most populous in the world (except India and China) and contains the great industries.

(2) There is much rainfall and cloud; frequent grey skies; little sunlight; winters are long, cold and wet, and there is a smoke pall in the atmosphere.

(3) It is the great grain-growing belt for wheat, oats and rye.

North of the zone e.g. in Greenland, Iceland and the Faroe Isles it rarely occurs.

South of the area, Southern Italy Southern Spain and Greece are immune. Constantinople is an exception. There is much rickets in that city, possibly owing to its wet climate, but rare at the sea coast and at an altitude. It is never seen at 3000 feet and over. In Davos, Volland and Sprengler did not find a single case.

As a general statement, "Rickets is almost non-existant in Egypt Algeria, Morocco, W & E Africa, Central Africa, S. Africa, India, China and Japan". - That is where there is intense sunlight during the greater part of the year. It seems to be an obvious conclusion that such sunlight is sufficient to overcome the rachitic effects of bad feeding and bad hygiene of all kinds, for the latter are doubtless rife in most of these countries.

In Australia, rickets is uncommon except in the large cities of Sydney and Melbourne, where a mild type is fairly common. In these cities it may be said that the advantage of a sunny climate is offset by the crowded state of the population and buildings.

In New Zealand, rickets is extremely rare. The climate is, on the whole, sunny, and there are few slum areas.

In America, rickets abounds in the cities of the Atlantic coast and in densely populated parts of California. Grulee of Chicago states: "In a large experience of negro babies in Chicago, I have never seen one between 6 and 18 months of age, whether breast or artificially fed, that did not have very definite signs of rickets".

Incidence. Hess and Unger (1922) studied the incidence of rickets amongst infants in New York. They point out that with our broadened conception of rickets, using the tests of beading of the ribs, diminished inorganic phosphate in the blood, and the X-Ray, almost all bottle-fed infants, examined in March, give some indication of rickets, Rickets is much more prevalent in the winter, and its incidence increases month by month until April. Even a group of well-nourished breast fed infants, examined clinically and by X-Ray at the end of March showed rickets in more than 50% of the cases.

Schmorl (1914) investigated histologically for rickets 221 infants between 4 and 18 months of age, as they came to autopsy. He found rickets present in 96.7 per cent. In only 7 of these cases were histological changes absent.

Studies of the incidence of rickets amongst young children in large cities e.g. Vienna, Glasgow, London, Boston and New York have been made by Kassowitz, Findlay, Morse, Hess and Unger &c. These observers have generally found an incidence of rickets of 50% to 80% in these cities.

The writer carefully examined for signs of rickets all infants between 6 months and 3 years of age who attended during the years 1924-26 the consultation clinics at the Carnegie Child Welfare Centre, Motherwell. The town is a mining and steel making centre in the Lanarkshire coalfield and has a population of 70,000. No case was considered to be rickets unless it displayed at least two well-recognised osseous signs, e.g. beading of the ribs and enlarged epiphyses. In addition most of the cases showed signs of epiphyseal rickets or at least osteoporosis by X-Ray examination.

The results were as follows:-

Year	Cases examined Age 6 mths to 3 years	Definite Rickets	Percentage.
1924	247	135	54.6%
1925	278	197	70.9%
1926 (1st 4mths)	105	91	87%
Total	630	423	67%

It will be noted that the incidence appeared to undergo a continuous rise. This was apparently due to several factors:- e.g.

(a) the popularity of the Artificial Sunlight Clinic, which commenced to operate in 1924, and which attracted increasing numbers of rickets cases to the Centre.

(b) the figures for 1926 are taken from the months of the year when rickets are most prevalent, viz. Jan. to April.

(c) during the period under review poverty greatly increased in the town owing to long-continued industrial depression.

In order that these figures might be checked, the health visitors were carefully instructed in the clinical signs of rickets and asked to record on a card certain definite measurements of all children visited by them between the ages of 6 months and 5 years. In this way a fair sample of all the children in the town were examined. The examinations were made during May. The results were as follows:-

Health Visitors cards examined (children age 6 mths to 3 yrs.)	Definite Rickets	Probable Rickets	Percentage of Rickets (including probable cases)
631	252	59	49%

Summary

1. Rickets is indigenous in Europe and N. America between Lat. 42 and 60 N. It is most prevalent in the cities of the plains, where climate is wet, buildings are high and crowded together, and there is a smoke pall.
2. If we except a few large cities, rickets may be said to be absent from sunny countries.
3. The incidence of rickets in large manufacturing cities in Britain and America is from 50% to 80%, or even higher if the slighter forms of rickets are counted and examinations made in March, during which month the disease is most prevalent.
4. In a Scottish mining and steel centre of 70,000 population the incidence of clinical rickets with osseous signs amongst children aged 6 months to 3 years, was found to be about 50% or even higher.

(c) Rickets and Environment.

Confinement or "domestication".

The classical data upon which confinement and lack of exercise were believed for a time to be the chief causes of rickets, are now seen in a new light in virtue of the more recent discovery that deprivation of ultra-violet light exerts a powerful rachitic effect upon a young infant.

Hansemann in 1906 considered that rickets developed in children for the same reason that it developed in young wild animals in the Zoo, viz. that they were deprived of exercise and fresh air. He noted that autopsies of children born in Autumn and dying in Spring usually show strongly marked signs of rickets, while those born in Spring and dying in Autumn rarely show signs of the disease.

Findlay (1908) supported this theory on the ground of the distribution and incidence of the disease, and on the basis of experiments on puppies kept confined in cages.

Findlay and Ferguson (1918) on the basis of sociological enquiries in Glasgow slums concluded that "inadequate air and exercise seem to be potent factors in determining the onset of rickets". They found that only 32 to 42.5% of the rachitic children examined, as against 86.5% of the non-rachitic had sufficient outdoor exercise.

Paton, Findlay and Watson (1918) supporting the same idea and opposing the theory of a dietetic anti-rachitic factor, compared pups kept in the laboratory on an ample milk-fat ration with others allowed to run about in the country and kept on a low diet in milk-fat. They found that the former all became rickety, while the latter remained free from rickets.

Moisture Hagen-Torn (1896) observed the great frequency of rickets in those provinces of Russia in which the moisture

exceeds 80%.

Korenchevsky (1922) by comparative animal experiments with rats in "dry" and "wet" cages was unable to obtain any evidence that increased humidity of the atmosphere per se, played an important part in the aetiology of rickets.

Leonard Hill and others have shown that ultra-violet rays are largely prevented from reaching the surface of the earth when there is much mist in the atmosphere.

Conclusions.

While it is probable that all forms of bad or depressant environment exert a rachitic influence, there is abundant evidence that they do so chiefly by depriving the growing animal of ultra-violet light. Experimental evidence in favour of the domestication theory is now seen to suggest that in the prevention of rickets, the value of light to the growing animal is paramount, and exceeds even that of a fully adequate diet.

Rickets and Vitamines.

Although Cod Liver Oil has been used empirically in the treatment of rachitic children for generations, and, as long ago as 1884, Bland Sutton used it to cure rickets in lion cubs in the Zoological Gardens, there was no clear notion as to how it exerted this effect until the modern conception of vitamines in food arose.

In 1906, Gowland Hopkins suggested that rickets might be a deficiency disease associated with the lack of an accessory food factor in the diet. While Hopkins was working at vitamines in this country, the American workers McCollum and Davis, working independently, described the fat soluble accessory factor vitamin A (1914).

Professor Mellanby in 1918 using puppies, produced definite experimental evidence that rickets could be caused by deficiency of fat soluble vitamines in diet, and could be prevented and cured by the addition of such accessory food factors to the standard diet. The chief sources of these vitamines were cod liver oil and milk fat.

Korenchevsky (1922) by experiments with rats corroborated Mellanby's work and concluded that the anti-rachitic factor operated by influencing the capacity of the animal to assimilate calcium. He showed also that where the diet of the mother rat, during pregnancy and lactation, was deficient in the vitamines, the offspring were liable to develop severe rickets on the same diet. Mellanby (1926) showed that this applied also to puppies and that even after a period of good feeding, rickets yet occurred readily in the offspring if the defective diet were introduced.

In human rickets, Schabad (1909-10) showed by biochemical retention experiments that Cod Liver Oil sharply increased the retention of calcium. Howland and Kramer (1921) showed, in 12 cases of rickets, that cod liver oil produced an average rise

of inorganic phosphorus in the blood serum from 1.9 mgm to 5.5 mgm per 100 c.c. and cured the rickets. In Vienna, Chick, Dulsiel, Hume, Mackay, and Bimberger (1922) both prevented and cured rickets in Winter by the use of cod liver oil, whilst control children treated without it, developed rickets. Park and Howland (1921) investigated radiographically, and in some case microscopically the deposition of calcium salts in the skeleton of rickety children before and after cod liver oil treatment. "In two or three months", they state, "so much infiltration with lime salts has taken place, that the extremities of the bones, except for deformities, were practically normal. The authors look upon cod liver oil as a specific for rickets, they have not known it fail in any single instance, and they have known it to cure the rickets even though the children were dying of some other disease". On the other hand, May G. Wilson found that in 47 infants <u>treated as out-patients</u>, daily doses of 1-3 teaspoonfuls of Cod Liver Oil failed to prevent the development of rickets in 68 per cent of the cases.

McCollum and his co-workers (1921-22) showed that there were two fat soluble vitamines: one vitamin A or growth promoting &c., and the other vitamin D or anti-rachitic. Vitamin D is present in milk fat in less proportion than Vitamin A, but it predominates in cod liver oil, 1% of cod liver oil being more highly anti-rachitic than 10% - 20% of butter. Wagner and Bimberger (Lancet 1924) corroborated this work of McCollum's by the use of cod liver oil oxidised and heated to 120° C. They found that the oil so treated had its anti-rachitic power only slightly diminished when tested on children suffering from active rickets. On the other hand, the growth-promoting power of the oil as tested on rats was reduced to one twentieth of its former value.

Again, it has been found that 0.1 gram of spinach leaves is sufficient to restore the power of growth to animals kept on a deficient diet, whereas 3.0 grams will not cure rickets.

Hence fresh green vegetable is a source of vitamin A, but not of vitamin D.

Many attempts have been made to isolate the vitamins as pure substances, but without success. Steenbock and his co-workers (1921) followed by Drummond (1924-25) have traced the vitamins A & D to the unsaponifiable part of cod liver oils and again to the cholesterol-free residue. From this residue the latter have obtained by fractional vacuum distillation certain yellow oils which contain the vitamins in one-thousandth part of the bulk of cod liver oil.

And now the story of rickets and vitamins becomes inevitably intertwined with the story of rickets and ultra-violet light.

E.Hume (B.M.J.22-8-25) repeating experiments of Hess, Unger and Pappenheimer, showed experimentally that in rats fed on a diet deficient in fat soluble vitamins, but exposed to ultra-violet light, rickets did not occur. After a time growth failed, xerophalmia and death occurred, but, post mortem, calcification was found to be normal. This seems to prove that exposure to ultra-violet light produces the same effect as Vitamin D, but not that of Vitamin A. Again this quality was traced to the unsaponifiable fraction of the substance and to the common factor a sterol, possibly cholesterol. It was then found by Drummond and his co-workers at the Lister Institute that, while ordinary cholesterol is not anti-rachitic, cholesterol crystals, but not dry cholesterol can be made highly anti-rachitic by irradiation. From irradiated cholesterol Rosenheim and Webster by the use of digitonin precipitated a substance which in daily doses of $\frac{1}{100}$ mgm. prevents rickets in rats. Their most recent work suggests that ergosterol is the actively rachitic substance which is associated with cholesterol.

The theory has thus been propounded that u.v.light is anti-rachitic in virtue of its production of vitamin D by action on the cholesterol of the skin. That it mobilises the reserves of vitamin D in some way is fairly certain, but since experience

shows that it exerts its anti-rachitic effect for a considerable time after exposures have ceased, it is difficult to understand how the manufacture of vitamin D from the skin can go on during the period when there is no exposure to light. The theory, however attractive, cannot yet be said to be proved. The facts, however, warrant the conclusion that rickets can be prevented and cured both by vitamin D in diet, and by ultra-violet light, and that in practice the latter is more constant in its action than the former.

Further insight into the nature and relationship of vitamins A and D in cow's milk has been obtained by Luce (Biochem. Journ. 18, 716, 1924) and corroborated by Chick and Roscoe (Biochem. Journ. 20, 632, 1926). By irradiation of vegetable oils etc., it has become possible to produce substances containing vitamin D and no vitamin A. On the other hand, as fresh green food contains vitamin A but not D, it thus becomes possible by feeding experiments etc., to test the sources of vitamins A and D in cow's milk. Using this method, the above observers found that the vitamin A of cow's milk is obtained from the cow's food, and is abundant when the cow is fed on fresh green food, and poor, with the winter feeding of cereals and roots. It was also shown that the content of vitamin A in the milk was independent of exposure of the cow to light or darkness. On the other hand, the vitamin D content does chiefly depend upon exposure of the cow to sunlight. Thus the cow does not manufacture vitamin A, but passes on what it has received in its food; while it <u>does</u> manufacture vitamin D according to the degree of exposure of its skin to u.v. light.

These experiments, if applicable to the human mother, suggest how variations in the anti-rachitic power of breast milk may be brought about, viz. according to the degree of exposure of the mother to u.v. light. It would help to explain the well-known fact that many breast-fed infants in industrial districts become rachitic. The observations further suggest

that exposure of the mother to u.v.light is more important than diet in the prevention of rickets, since this is the source of the anti-rachitic substance vitamine D.

There is indeed very recent proof that anti-rachitic properties not previously existing in the milk of a nursing mother were developed in it by exposure of the mother to daily irradiation with the mercury vapour quartz lamp for 1 month; and that these properties resided in the non-saponifiable fraction and were presumably vitamin D. (Hess, Weinstock and Sherman. J.A.M.A. 88 No. 1 1st Jan. 1927. p.24)

There is another, though probably subsidiary, phase of the relationship of rickets and vitamines, which has been brought forward by E. Mellanby, viz. the existence of a rachitic anti-vitamin in certain cereals. He found, by experimental work on puppies, that when excess cereal was given in the diet, it exerted a rachitic effect quite opposite to the anti-rachitic effect of cod liver oil or milk fat. Oatmeal was the worst cereal in this respect but its rachitic action could be easily antagonised by cod-liver oil, egg yolk and milk. Mellanby further traced the source of this rachitic substance to the <u>saponifiable</u> part of the fat of oatmeal. "Thus" as he says, "we may have in the fat of oatmeal an anti-rachitic vitamin and a rachitic anti-vitamin side by side, one associated with the unsaponifiable and one with saponifiable portion of the fat". He adds that "it would appear not improbable that in this country where the average diet is either deficient in or contains a border-line quantity of anti-rachitic vitamin and calcium, and where sunshine is negligible, the ingestion of oatmeal during pregnancy and lactation, and in growing children does much harm.

This work serves again to point to the complexity of the factors which may be involved in the production of rickets, although as the report indicated, all of those factors are by no means of equal value.

Before leaving the subject of vitamins it is right to mention the view of certain biochemists that proper balance of

minerals in a diet is sufficient to explain its anti-rachitic effect, without postulating the existence of vitamins. Thus Orr points out that the deposition of lime salts in the bones is dependent on the concentration of Ca and P in the blood, and that the main factor in maintaining the essential physiologically balanced solution in the blood is the mineral content of the diet. If the diet is so balanced that all the essential minerals pass through the wall of the intestine, it is doubtful if rickets can occur.

"Cod liver oil and U.V.light", he says "have their greatest influence when the mineral content of the diet is defective, since they increase the percentage absorption of the deficient calcium".

On this subject, on the other hand, Pryde quotes McCollum's recent work by which rickets was produced in young rats by diets well-balanced in minerals, but not containing vitamin D. He says "It is now generally admitted that it is possible so to adjust diets that the deciding factor as to whether or not any animal develops rickets becomes the presence or absence from the diet of the anti-rachitic vitamin of which cod liver oil is the most potent source". He quotes the Report of the Medical Research Council (1924) as to the points of general agreement regarding the relative importance of a deficiency of calcium, phosphate, and anti-rachitic vitamin in the causation of rickets, viz:

(I) An anti-rachitic vitamin in the diet corrects improper balance in the calcium and phosphorus intake, and the greater the disproportion or defect in these elements, the more important is the role of the vitamin in the prevention of rickets.

(2) Even when the calcium and phosphorus balance is good and the supply of each adequate the absence of anti-rachitic vitamin from the diet will result in the production of imperfectly calcified bone."

As to the bearing of all this on the practical problem of rickets in industrial districts, the writer would point out that ideally balanced diets either of mothers or children are quite exceptional, and that, therefore, in the present condition of things, some powerful anti-rachitic agent is often required to antagonise the ill effects of rickets-producing diets or constituents of diets. Such agents are cod liver oil and ultra-violet light, but the greatest of these, and the one which has other additional advantages, is, in the experience of the writer, ultra-violet light.

(e) Rickets and Ultra-Violet Light.

The subject of ultra-violet light in relation to rickets is so inextricably intertwined with that of vitamins, that inevitably many of the relevant facts have already been given, and only a brief resumé is now required.

One of the earliest apostles of sunlight in the prevention and cure of rickets known to modern medicine was Theobold Palm, who, in the "Practitioner" in 1890, suggested that the immunity of the Japanese to rickets was due to the intense natural sunlight to which they were constantly exposed, and that the frequency of rickets in British towns was due to lack of sunlight, associated with smoke and mist in the air and crowded tall buildings, shutting off the light from the streets and houses. He indicated that the chemical activity of the sun's rays was more valuable than its heat, and that the light of the sun was a powerful natural stimulus, which the young active growing organism required for healthy function.

There appears to have been no further mention of this idea in medical literature until in 1904 Bucholz reported the curative effect of light on rickets. Again the idea appears to have been forgotten for about 15 years, although Rollier and Bernhard during this period were working out with remarkable success the curative effect of the sun in the high Alps in surgical tuberculosis.

In 1919 Huldschinsky published his classical work on the cure of active rickets by an average of 26 exposures to the rays of the mercury vapour quartz lamp. He was the first to demonstrate this cure clearly by radiograms taken before, during and after treatment.

Freer (1921) drew attention to the improvement in rickets produced by heliotherapy in the Swiss Alps.

Then came the excellent work of the English lady doctors in Vienna in 1922, Chick, Dalzell, Hume, MacKay and Smith, who showed that active rickets in infants was cured by cod liver oil, and

by exposure to sunlight or to the rays of the quartz mercury vapour lamp. In this work, Bimberger demonstrated the healing of active rickets by a series of beautiful radiograms. He showed that radiographic signs of healing occurred about three weeks after the commencement of treatment either by cod liver oil or light, and that healing was in most cases complete in two or three months.

In 1922, Powers, Park, Shipley, McCollum and Simmonds, by experiments with rats reached the conclusion that "the effect of sunlight and of cod liver oil on the growth and calcification of the skeleton, and on the animal as a whole, seem to be similar if not identical".

The latter statement, in the writer's experience, is not borne out in practice. Ultra-violet light has two distinct effects on the rachitic child:

(1) improvement in calcium and phosphorus metabolism and (2) improvement in general health.

In practice it is frequently found that the second effect is even more striking than the first, and this cannot be said of cod liver oil therapy in anything like the same degree. Artificial light therapy usually brings considerable general improvement in the activity, muscle tone and contentment of rachitic infants, whereas cod liver oil has much less general beneficial effect.

Mackay and Shaw (B.M.J. 22-8-25) make the following statement with which the writer agrees: " as regards the retention and metabolism of calcium and phosphorus, cod liver oil, containing the so-called anti-rachitic factor, and ultra-violet light can be regarded as interchangeable; but the same does not hold as regards the effect on the general health".

The anti-rachitic effect of U.V. rays in the form of "artificial sunlight" has been abundantly confirmed. In the Annual Report of the Scottish Board of Health for 1925, it is stated that the Carnegie Clinic, Motherwell, was the first public institution in

Scotland to provide light treatment for rickets, and the present writer's account of that treatment based on 75 completed cases of active rickets cured and greatly benefited in general health by 20 or more exposures to the rays of Carbon Arc and Mercury Vapour lamps is given in the report. In 1927, K.L.Gamgee published her book on the "Artificial Light Treatment of Children in Rickets, Anaemia and Malnutrition", in which she gives an analysis of 200 such cases treated and, like most of those who have done such work is most enthusiastic about results..

The following is a brief statement of the effects so far ascertained of ultra-violet light ~~of~~ on the young rachitic organism:

The rays beyond the spectrum of visible light down to 2,900 Angstrom Units have been described as "biologic" and are of such a nature as to stimulate biological processes. Rays from 2,900 to 2550 are called "abiotic" and are effective chiefly in producing a lethal reaction, e.g. as germicides, and their penetration into the skin is very slight.

1. Effects on Calcium and Phosphorus Metabolism (anti-rachitic)

(a) In Ca-deficient diets, an increased amount of calcium is absorbed from the intestine and retained.

(b) Such increased absorption of Ca leads to increased fixation of phosphate in the skeleton.

(c) Thus u.v. radiation exerts its maximum anti-rachitic effect, when as the result of badly-balanced diet, or some other cause, retention of minerals is low.

(d) The amount of inorganic P in the blood, reduced in active rickets, is increased, and the amount of Ca in the blood, if reduced, is increased.

(e) In practice, these effects bring about the cure of active rickets in children in all but a negligible proportion of cases, e.g. those so ill as to have no remaining power of

response to the stimulus.

2. Effects on General Health.

(a) A marked feeling of physical well-being, evidenced by better appetite and sleep, brightness of disposition, increased muscular activity, loss of head sweating, increase of weight, etc.

(b) Improvement in anaemia, e.g. a slight increase of red blood corpuscles, increase of haemoglobin, temporary leucocytosis mainly lymphocytic and increase of blood platelets.

(c) Great improvement in muscular tone.

(d) Increase on haemo-bactericidal power of the blood, except in an overdose of radiation.

3. Effects on the Skin.

(a) Production of erythema, oedema, and, in an overdose, destruction of tissue cells, by action through the cutaneous nerves.

(b) A powerful antiseptic action and direct curative effect on superficial skin lesions due to bacterial infection.

(c) A marked improvement in many chronic skin diseases, e.g. psoriasis, eczema, etc.

(f) Infection and Heredity in Rickets.

Infection.

Various workers including Mircoli (1898) Morpurgo (1900), Koch (1912) and Korenchevsky (1922) have attempted to produce experimental rickets in animals by injection of various micro-organisms obtained from bones of rachitic subjects, or by feeding with or injection of intestinal anaerobes etc. None of these have succeeded.

Paton (1922) has suggested that unhygienic conditions and defective diet may be simply predisposing causes, and that the exciting cause may be an infection. No direct evidence, however, is adduced.

V.Pirquet (1922) has stated that his previous belief in the infectious nature of human rickets was shattered by the Vienna workers. As a result of their work he says, " The chain of evidence appears to me to be complete that animal experiments upon rickets are applicable also to man, that rickets is a disease of nutrition, and that deficiency of fat soluble vitamins in diet is an essential cause of the disease".

Heredity.

Pfeiffer(1885) Siegert (1903) and others have drawn attention to the frequent occurrence of rickets in several members of the same family, and in children of rachitic parents. Korenchevsky's experiments with rats, and Mellanby's with bitches provide us with some direct evidence on the point and suggest that deficiency of Vitamin D and of Ca salts in the maternal diet during conception, pregnancy and lactation, has a rachitic influence on the offspring. The experiments of Chick and Roscoe on cows, suggest strongly that the factor of greatest influence in this respect is the degree of exposure of the mother to ultra-violet light, during the same period.

Since Schmorl (1909) found by histological examination, rickets present as early as 6 weeks after birth, it is probable

that in the case of such young infants, latent or incipient rickets exists at the time of birth.

Summary.

(1) The available evidence is strongly in favour of rickets being a disease of nutrition, rather than an infectious disease.

(2) Rickets is hereditary only in so far as the various rachitic influences, chiefly lack of light, acting upon the mother during pregnancy and lactation, bring about disturbed nutrition and the "status calciprivus" in the offspring. If such a child is not subjected to intense anti-rachitic influences, clinical rickets may occur at a very early age.

Some complications or associated conditions.

Dental Hypoplasia. Delayed dentition has long been regarded as one of the accompaniments of rickets, but until recently little attention has been paid to hypoplasia or deficient and defective structure and calcification of the deciduous teeth. This is essentially of the same nature as rickets, although many children who exhibit it, may show few, if any, of the classical signs of rickets. It must be regarded as a "minor" manifestation of rickets, but nevertheless it is a most important one. Mrs. Mellanby has recently found by microscopical examination of thin sections of about 1000 deciduous teeth that instead of 3% being structurally defective (as dentists, relying upon naked eye examination have reported) fully 80% of such teeth examined were of defective structure. The architecture of the enamel and of the dentine was not such as to produce "normal" hardness.

Comparing normal and abnormal teeth in a series of 636 she found that 92% were in agreement with the generalisation that defective structure is associated with caries and sound structure with no caries. In those teeth which were found to be structurally defective and yet showed no caries, there was generally found a good deposit of secondary dentine which was probably due to improved calcifying conditions after the teeth had been formed.

This may be said to be the modern view of dental caries. Healthy well-formed teeth do not readily decay irrespective of the use of the tooth brush.

In puppies Mrs. Mellanby has been able to produce well-calcified teeth by

(1) A diet containing vitamin D

and (2) exposure to ultra-violet light.

On the other hand, she has produced teeth structurally defective by giving a diet containing excess of cereal, especially oatmeal.

She has shown also that a group of children brought up on a

diet including milk, egg yolk and cod liver oil did not show dental caries, at least up to the age of 5½ years.

As dental caries leads to oral sepsis, and predisposes to infection, to enlarged glands, enlarged tonsils and adenoids, chest deformities malnutrition etc., it is evident that if it is essentially rachitic in nature, as the above work as well as general experience indicates, then the rachitic condition is one that is exceedingly widespread amongst the children of our nation , and is fraught with more widespread injury to health than has formerly been supposed.

Broncho-pneumonia and diarrhoea. Every medical worker in an industrial district knows that these diseases are very commonly associated with rickets. E. Mellanby and others have shown that animals suffering from experimental rickets, whether produced by defective diets or otherwise, are most liable to suffer from bacterial infections, especially of this nature.

E. Hume as far as experiments with rats are concerned, believes that respiratory disease is associated more with vitamin A deficiency than with lack of vitamin D.

If this is so, broncho-pneumonia and diarrhoea may be associated not so much with rickets as with dietetic deficiency in vitamin A. Such deficiency in practice, is often found in the rachitic child, but not necessarily so.

Fatalities from Measles. The clinical experience of the writer as regards measles in industrial districts in Glasgow and Lanarkshire, strongly suggests that fatal broncho-pneumonia following measles is most frequently associated with rickets. No figures have been kept of earlier experiences in this respect, but a record was kept of cases admitted to Hospital in Motherwell in connection with a small outbreak of measles in the spring of 1926. There were 12 children, whose ages ranged from 7 months to 2½ years, admitted with broncho-pneumonia following measles. All of those had clinical signs of rickets

and 4 of the 12 cases died. The figures are too small for any sound opinion to be based on them, but they are in line with previous experience in this respect. It can at least be said that measles is commonly a much more severe disease in a rachitic than a healthy child.

Enlarged Tonsils and Adenoids.

Some evidence as to the frequent association of this condition with rickets has been brought forward by McGonigle of Stockton. He found in an examination of 2676 school children in the County of Durham, that 83% showed definite osseous signs of rickets, 11% showed doubtful signs of rickets, and only 6% were non-rachitic. In the first group ("Rickets +" children) 27% exhibited signs of enlarged tonsils or mouth-breathing or both: in the second group ("Rickets ?" children) there were 15% such cases and in the third group (non-rachitic) there was not a single case of enlarged tonsils or adenoids.

The osseous deformities adopted by McGonigle as signs of rickets in connection with this work included head bossing, chest deformities, bow legs and knock knee.

He thus found that the children with enlarged tonsils and mouth breathing almost all had had rickets. This work has not so far been confirmed and probably few workers would be inclined to go so far, but the results are not altogether unexpected by those who have worked in districts containing a high proportion of rachitic children.

Summary: Some recent evidence of a preliminary nature is offered in this section as to the frequent close association of rickets with (1) Caries of the temporary teeth, (2) broncho-pneumonia and diarrhoea of young children, especially that following measles, and (3) enlarged tonsils and adenoids. The evidence is sufficiently in line with clinical experience of rickets in industrial districts, to suggest strongly the widespread nature of the damage to health associated with the rachitic condition.

5. Treatment of Rickets at a Child Welfare Centre in an Industrial Area.

The present writer became interested in the problem of rickets whilst working at the Carnegie and other child welfare centres in the industrial town of Motherwell and Wishaw during the years 1919 to 1926. The town is situated in the Lanarkshire coalfield and has a population of about 70.000. Its industries are almost exclusively those of coal and steel production. There is, therefore, a very considerable amount of smoke at all times in the atmosphere. During 1919 and 1920 the town was prosperous and the proportion of cases of active rickets seen at the child welfare centres was low.

From 1920 onwards, the coal and steel industries have been continuously depressed, and the poverty of the inhabitants has increased progressively. Coincident with this increase of poverty there has been in the experience of the writer a very considerable increase in the amount of active rickets observed in the children. It may be mentioned that the town is overcrowded and that the housing conditions of the population are among the worst in Scotland, there being roughly about 20% of the houses consisting of one room, and about 55% consisting of two rooms.

During the period 1919-24 the treatment of rickets carried out at the child welfare centres was on dietetic and hygienic lines, and particularly by the use of cod liver oil. During the latter two years, an extensive experiment on ultra-violet light treatment of the condition was carried out at the Carnegie Model Child Welfare Centre, Motherwell, and careful clinical records were kept and, in many cases, X-Ray examinations made.

A preliminary report on this work was published in several medical journals e.g."Medical Officer" 12/3/26 during 1926, and was printed in full in the Annual Report of the Scottish Board of Health for 1925. A more detailed account of the work is given below.

Early Period (1919-24)

Treatment by Diet, Hygiene and Cod Liver Oil.

During those five years several hundreds of infants and young children were examined, chiefly on account of various forms of "minor ailments". In most of those the rachitic state was present to some degree, and in a fair proportion the signs of rickets were gross. The diagnosis was made by clinical signs only, as the X-Ray was not at that period available at the centres.

These children belonged to the poorest class in the town. They almost all lived in grossly overcrowded houses and were fed on the "mushy" diet commonly given to such infants, and which is now recognised as being an inadequate diet in respect of vitamines and probably also of calcium, phosphorus and iodine.

For example the infant's diet after weaning consisted generally of a combination of diluted cow's milk, bread and milk, bread and margarine, oatflour, milk pudding, tea and milk and sugar. Very rarely was butter or egg or fruit or green vegetable given. The staple diet of the mother was bread, margarine and tea with occasional sausage or pies or bacon and rarely butcher's meat. The infants in many cases were, badly cared for and dirty, as it was impossible for the mothers to give their children reasonable attention under the onditions existing.

The clothing was as a rule dirty, superfluous and constricting and generally included a broad, thick tightly-applied flannel binder. The skin was thus kept in a humid moist unstimulating atmosphere, so that it was more or less constantly covered with visible perspiration. The effect of the clothing was that of a powerful depressant of metabolism. The atmospheric conditions of the town were bad, as regards smoke, mist and sunlight. The houses in which these cases lived were often small, damp and overcrowded and some lacked an internal water supply.

The buildings were not, however, so much crowded together as the slum quarters of the larger cities, most of the houses

being in rows of one or two storeys in height, and not, as a rule, in the form of tenements. This was, perhaps, the one redeeming feature of the conditions under which the children lived.

Under such conditions, where mothers and children were subjected to every kind of rachitic influence it is little wonder that little of value was accomplished. In many cases the mother's chief object in attending the Centre was to obtain free milk, and she was not disposed to depart from traditional methods of feeding, clothing and management especially if it involved some continuous effort and determination. At the same time a minority did make an effort, under the adverse conditions, to respond to the advice which was given as to diet, hygiene and management. This advice included the following:-

(1) Diet.

A more generous allowance of milk for infant and mother. Generally 1 pint of milk daily was given free or at reduced cost according to economic circumstances.

The mothers were advised to make every effort to obtain the following for the baby:- butter, an egg twice or three times weekly, soup, fruit or orange juice, except in cases suffering grom gastro-intestinal derangement, vomiting, diarrhoea etc. For the worst of this latter class, treatment in Hospital was arranged.

(2) Cod Liver Oil.

Those exhibiting definite osseous rachitic signs were given cod liver oil, preferably in the form of the pure oil one teaspoonful three times daily. If this was not well tolerated cod liver oil 33% Emulsion was tried, and, failing that, Malt with Cod Liver Oil 15%.

(3) Hygiene.

Removal of binders and all restrictive clothing was advised with reasonable aeration and cleansing of the skin, and keeping the child, as much as possible, in the open air.

The visit to the Centre was generally followed up by visits to the home by health visitors who endeavoured to persuade the mother to adopt these methods, and also to return to the Centre at intervals.

The success achieved by these methods was very small. There was a relatively small number of those with gross rickets who improved remarkably, and in a few instances towards the end of 1924 it was possible to demonstrate healing of active rickets by X-Ray examination of the epiphyses. The great majority plapsed as regards diet and hygiene, and did not continue the cod liver oil owing to the expense or trouble involved or, evenly more commonly, owing to the fact that, as given under home conditions it was badly tolerated. The following cases were among the most successful:-

Case 1.

M.McK. age 2½ years, when seen on 23/5/24, had gross stigmata, of rickets, e.g. open anterior fontanelle, cranial bossing; beading of ribs, Harrison's sulcus and genu valgum. She had a pale sweaty skin and flabby muscles X-Ray films showed a typical form of active rickets in a growing child, viz. a "frayed out" appearance of the end of the diaphyses, denoting irregular calcification and growth of osteoid tissue, between epiphyses and diaphyses.

There had been several children in this family with rickets, and the mother was anxious to do her best for the child. Cod Liver Oil was taken regularly 12 oz. per month, and diet and hygienic conditions slightly improved.

Result on 22/8/24. childs general condition much improved and X-Ray film showed healed rickets with formation of a good regular provisional zone of calcification at the epiphyses.

Note. This period of 3 months during which healing of rickets took place correspond to the mid-summer months with maximum of sunlight, and this, no doubt, had some effect

Case 2.

M. C. age 2 seen on 16/5/24; gross rickets, e.g. open fontanelle beading of ribs, Harrison's Sulcus, pot belly and bow legs, with pallor, sweating about head, and intermittent diarrhoea.

X-Ray - "fraying" of epiphyseal line.

Treatment:- Pure Cod Oil not tolerated, 8 lbs. Malt and Cod Oil 15% given in 3 months.

Result on 22/8/24. condition improved.

X-Ray shows healing rickets, the zone of provisional calcification being fairly well formed.

(Note:- Healing in this case also took place during summer).

Case 3.

R.S. age 6 months when seen on 29/1/24: weight 16 lbs. 11½ ozs. a fairly large soft flabby child with osseous signs of rickets viz. craniotables: parietal bossing: beading of ribs, and Harrison's Sulcus. Wholly breast fed. Mother very poor and had been deserted by her husband.

Treatment. Cod Oil Emulsion ½ drachm, t.i.d.

Progress after two months: general condition improved, muscle tone much better: no teeth yet erupted. X-Ray film shows slight "fraying" of epiphyseal lines at wrists.

Result on 27/5/24. after 4 months treatment: muscle tone now good and activity increased. 4 teeth just erupted at age 10 mos. Osseous stigmata e.g. widely open fontanelle, persist. 2 years later, X-Ray on 8/1/26 shows transverse lines of old healed rickets near the epiphyses, and also some curvature of tibia and fibula which had become considerably straightened by increase in the thickness of the periosteal new bone in the concave portion.

Case 4

H.R. age 1 year and 1 month, weight 17 lbs. 4½ ozs. when first seen on 9/10/23, severe rickets, Pallor: sweating about head: large open fontanelle: parietal bossing: greatly enlarged radial epiphysis etc: beading of ribs: only 3 teeth erupted.

Muscle tone poor: child unable to stand or walk. Suffers from intermittent diarrhoea, and is cross and peevish: has numerous ulcers on mouth and tongue and discharging ears.

Diet. Wholly breast fed for last 4 months.

Progress: The child was treated in Hospital under good hygienic conditions for 4 months (during the winter).

Gastro intestinal disturbance precluded the administration of much Cod Oil.

X-Ray on 7/12/23 and again on 22/2/24 showed evidence of florid rickets and no appearance of healing i.e. osteoporosis and extreme "fraying" of epiphyseal lines at wrist and elbow; that is, Hospital treatment during the winter resulted in no improvement of the rachitic condition.

Subsequent History:- From April 1924 to Jan. 1925 was treated at home and had equivalent of 52 oz. cod liver oil, as well as open air treatment during the summer.

X-Ray 18/12/25 showed healed rickets with well formed epiphyseal lines and marked curvatures of tibia and fibula, which had been well "straightened out" by formation of a broad zone of periosteal bone in the concavities. Stigmata of rickets still present e.g. parietal bossing: enlarged epiphyses and genu valgum.

This case shows the failure of Hospital indoor treatment during the winter months to cure rickets, and its subsequent slow cure by cod liver oil combined with open air treatment during the following summer.

Case 5

M.H. age 8/12 when seen on 28/3/24. Signs of rickets present e.g. pallor, sweating: widely open fontanelle, beading of ribs and enlarged epiphyses.

X-Ray showed the fraying of epiphyseal lines at wrist, but calcification was fairly dense as when rickets is healing.

Treatment: Malt and Cod Liver Oil 1 lb. fortnightly i.e. equivalent of 8 oz. Cod Liver Oil in 2 months.

Result- X-Ray 16/5/24 showed healing rickets: epiphyseal lines were much more densely calcified and only slight fraying of distal ulnar epiphyses remained.

In the above, the best cases of the "Cod Liver Oil" series, the healing process took place chiefly in the summer or later spring months, and,no doubt, exposure to sunlight was an important contributory factor. Even so, the results cannot be called brilliant. In contrast with these there were very many cases of active rickets to whom the mothers were unable or unwilling to give cod liver oil. These were the greatest failures. They mostly healed slowly in the course or one or more years, doubtless under the influence of increasing exposure to sunlight as the child grew older and was able to be most out of doors. They exhibited however the penalty of slow healing of rickets viz. persistence of marked stigmata and deformity and severe intercurrent acute disease, especially enteritis and bronchitis.

The following two cases show that active rickets can be long continued with little signs of healing, in the absence of specific treatment.

case 6.

J.P. age 1 6/12 when seen on 16/4/23. Severe Rickets. Unable to stand or walk.

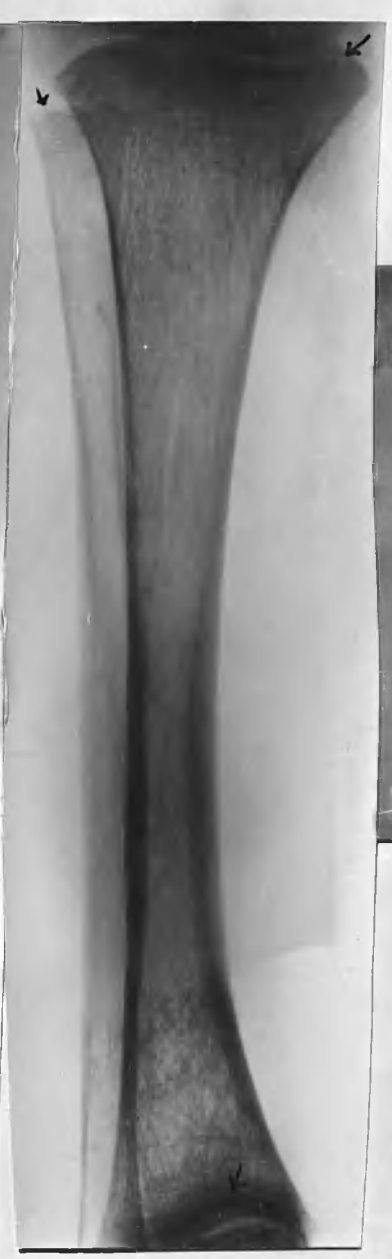
Treatment: child would not take pure cod oil emulsion, nor even malt and cod oil. Virol was given regularly.

Progress: One year later, April 1924, rickets still very severe, with gross deformity of lower limbs, and inability to walk without assistance (age 2½ years).

Jan. 1926. (age 4¼ years) X-Ray showed healed rickets, but with serpentine curvatures of tibia and fibula.

Case 7

J.McK. age 2 7/12 when seen on 6/6/24. Had severe active rickets with pallor, head sweating, flabby muscles etc., and



No. 77.

I.

J.McK. age 2yrs. 10 months.
Date. 6-6-24
Condition. Active Rickets.
Note. fraying, spreading and hollowing of metaphyseal lines with osteoporosis of shaft.
The double line at upper metaphysis of tibia denotes some attempt at healing.

J.McK. 18 mos. later.
Date. 12-12-25.
Condition. Active Rickets not yet healed.
Note. No treatment applied: cod liver oil not tolerated.

J.McK. after treatment with ultra-violet light.
Date. 24-3-26.
Condition. healed Rickets.
Note. Rickets definitely healed 3 mos. after ultra-violet light applied.

J.McK. age 4½ yrs
Date. Feb. 1926.
Note. Knock Knee flat foot &c. and mouth breathing associated with long-standing rickets.

marked genu valgum. X-Ray showed extensive "fraying" of epiphyseal lines and osteo-porosis.

Treatment: none: child would not "take" cod liver oil.

Progress: on 12/12/25, i.e. 18 months later, rickets clinically had not improved. X-Ray showed rickets still active, but with evidence of attempts at healing, the calcification being more dense than at previous examination, there was an old greenstick fracture in lower third of fibula; very slight periosteal calcification.

This case in the absence of specific treatment showed clinically no improvement and by X-Ray, only slight improvement in the rachitic condition over a period of 18 months; the period including two summers. Subsequently he was treated by exposures to ultra-violet light with rapid and complete healing and fairly good progress as regards deformities and general condition.

The following case shows gradual healing of active rickets and greenstick fractures with deformity. Partial straightening of the limbs occurred by thick deposition of new bones in the concavities of the deformities.

Healing in this case appeared to be aided by cod liver oil given irregularly and in uncertain amounts. There was, however, little corresponding improvement in the general condition.

Case 8

M.A. age 2 years when seen on 23/5/24: gross case of rickets: anterior fontanelle still admitted two fingers: beading of ribs: fiddle-shaped chest: parietal and frontal bossing: much enlarged epiphyses: genu valgum. Chlorotic pallor and marked sweating about head: irritability and fretfulness and soft flabby muscles: she was unable to walk.

X-Ray showed severe osteo-porosis and fraying at all epiphyseal lines at knee joint: greenstick fracture with deformity in lower third of fibula.

Progress: 3 months later, X-Ray showed signs of healing in the epiphyses, probably due to effect of good summer weather in 1924, aided by small amounts of Cod liver oil. Child was still sweaty and pale and showed little improvement in general condition.

Child seen one year and 4 months later; the stigmata of rickets were still most marked. The anterior fontanelle was still open (at age $3\frac{1}{2}$ years): genu valgum was marked.

X-Ray 12/12/25 shows healed rickets, with broad zones of periosteal calcification in the concavities of tibia and fibula, thus tending to straighten the limb.

As compared with the constant and rapid healing and the great general improvement noted in cases of rickets treated by ultra-violet light from lamps this slow uncertain healing action on the bones, indifferent action on the general condition and many failures of the "cod oil period" contrasts very unfavourably. Neverthe less it may be mentioned here that exceptionally good results have been reported in the treatment of rachitic conditions among the poor of an industrial district, by dietetic methods aided by cod liver oil, e.g. by McGonigle "Medical Officer" 6/11/26 of Stockton. The latter, working in an industrial district somewhat similar in character to that of Motherwell, obtained many good results by using diets containing apparently a sufficiency of vitamines e.g. the following:-

Diet for Rickets as used, at Stockton-on-Tees (McGonigle)

Nursing Mothers Diet: Whole meal bread and beef dripping are substituted for the usual white bread and margarine.

Fresh vegetables and fruit are added, if the mother can be persuaded to take them.

Diet of Weaning: at 8 or 9 months, additional food is given consisting of brown bread, butter or beef dripping, soup or stew with vegetables, meat

juice, half a raw egg in milk and fruit juice. When fruit is unobtainable, raw turnip juice is recommended. At 9 months, a very little potato is allowed. White bread, rice, sago, arrowroot, tapioco, biscuits, cornflour, oatmeal and all similar foods are absolutely forbidden. In some cases, cod liver oil emulsion is given (particularly those with a tendency to bronchial catarrh).

Dr. McGonigle continues:-

"The results are very satisfactory. The mothers are unanimous that with such a diet weaning causes no trouble. No vomiting or diarrhoea is caused and the frequently seen decrease in the weight curve at this age is eliminated. Babies usually walk before 12 months of age on this diet, and dentition is free from trouble.

In those cases in which weaning has been carried out by giving starchy foods, the baby is usually flabby and anaemic and head sweating is common. A change to the diet recommended (usually with a little cod liver oil emulsion) is followed by rapid improvement. The head sweating ceases in a week or ten days, and the general condition rapidly improves.

Many children are brought to the Centres for the first time. between the ages of 14 months and 2 years who have been fed on a starchy diet since weaning. The condition of these toddlers is typical. They are anaemic , flabby, cross and peevish, with marked head-sweating, big bellies, eversion of lower ribs, beading of ribs, outward bending of tibia and, in those children who have walked varying degrees of bow legs. Many have a distinct tendency to Bronchitis. In some, all these signs are present; in others, only some of them.

The improvement which follows a change of diet to that recommended for weaning with complete prohibition of starchy foods except wholemeal bread and some potatoes is usually rapid. The head sweating ceases; the anaemia is replaced by a healthy colour, the muscular tone rises and the child quickly gains weight.

It is no unusual thing for a child of 18 months who has never walked to walk quite well in three months".

Dr. McGonigle quotes a number of actual cases of which the following is an example:-

"Boy age 2 years. Gained 3 lbs 14 ozs. in 15 weeks; walked at one year but did not walk from 18 months to 2 years. Walked again within 3 weeks of attending Centre at 2 years of age. Had knock knees when first attended. Legs straight in 15 weeks".

As will be seen from the following pages, these are practically the results which the writer obtained by the use of "artificial sunlight" and without giving very particular attention to diet. That such results should have obtained by dietetic means alone (supplemented by a little Cod Liver Oil) in the patients of the class under consideration, appears to the writer to be an extraordinary success and must only have been achieved by the expenditure of great personal energy and by great devotion on the part of those in charge.

(b) Intermediate Period 1924-1925 Treatment by Cod Liver Oil first and Ultra.violet light subsequently

Case 9

J.S. age 3 3/12. first seen 23/9/24. Severe rickets with deformities e.g. extreme cranial bossing, fiddle chest; genu valgum, and enlarged epiphyses, Child pale, sweated easily, was cross and peevish, walked very badly and was easily tired. X-Ray showed frayed out, concave zone replacing the normal epiphyseal line: osteo-porosis and greenstick fracture at lower end of femur, and lower end of tibia and fibula.

Treatment Cod Liver Oil 30 oz. over 3 months period Sept. to Dec. 1924.

Result Fair degree of healing of rickets radiographically, but no marked improvement in general condition.

Further Treatment. U.V. Light 36 exposures Jan. - April 1925.

Result. Marked increase in brightness and feeling of well-being. Was heard to sing for first time after having 5 exposures, and was observed then to be walking strongly and actively. Gained 2 lbs weight in 4 months. Legs much straighter; fiddle chest and pot belly hardly noticeable, and was running about easily.

Case 10

J.C. age 11/12 on 12/8/24. Had moderate degree of rickets, with much enlarged anterior fontanelle, cranial bossing, rosary, and enlarged epiphyses. Child was pale, had flabby muscles, and was unable to stand.

1st Course of Treatment. Cod Liver Oil intermittently Aug. - Dec.1924.

Result Practically no improvement noted in general condition, and functional activity. X-Ray showed rickets healed radiographically, with several transverse linear shadows near the epiphyses, denoting intermittent attempts at healing.

2nd Course of Treatment U.V.Light Jan. - March 1925, 29 exposures.

Result Marked improvement in general condition and function.

Further history: Was found to have relapsed after the winter when seen again on 29/1/26. age 2 11/12. Fontanelle still open, rosary and Harrison's Sulcus; sweating: tendency to croup.

3rd Course of Treatment:- U.V.Light 21 exposures Feb.- to March 1926.

Result, Fontanelle closed: no sweating: running about actively and in good condition, although gain in weight was only 4 oz during the 2 months. X-Ray showed healed rickets.

Case 11

Case 11

J.D. age 3 3/12 on 1/5/23. Extreme rickets with gross deformities e.g. fontanelle still open: cranial bossing ++: fiddle-shaped chest: radial epiphyses of the size of small tomatoes: marked genu valgum. Child pale, sweated at night, had soft muscles, and walked very badly with waddling gait.

First Treatment: Hygienic treatment in Hosp. May to July 1923.

Result no improvement.

Seen again 9/9/24. X-Ray showed florid rickets with frayed out epiphyseal lines, and osteoporosis: no sign of healing.

Second Treatment: Malt and Cod Liver Oil, Sept. to Nov. 1924.

Result: X-Ray shows no healing in epiphyses of wrist, but some healing in the phalanges only.

Third Treatment: U.V.Light, 33 exposures Dec. 1924 to Feb. 1925.

Result X-Ray shows rickets healed at epiphyses of wrist and ankle.

Child running about: well and active: gained average of 11 oz. in weight per month during course.

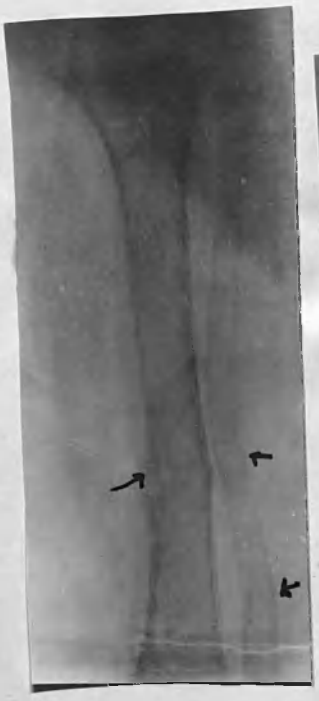
Case 12

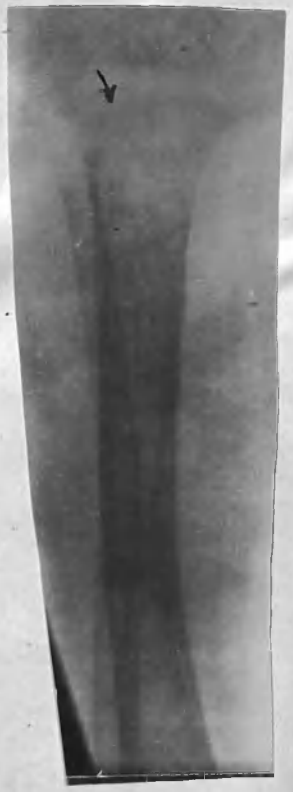
E.S. age 1 year when seen on 30/5/24. Extreme rickets with marked craniotabes, exceedingly soft musculature and unable to stand. X-Ray shows extreme osteoporosis, numerous greenstick fractures and frayed out epiphyseal zone.

First Treatment: Cod Liver Oil 33% emulsion 60 oz. May 1924 to March 1925. 10 months period.

Result: no improvement either clinically or radiographically. Rickets still active, with open fontanelle, fiddle chest, rosary, much enlarged epiphyses, pot belly and S - shaped tibial and fibular curves: head-sweating, pallor, muscles soft and unalbe to walk. (age 1 10/12)-

Second Treatment: U.V.Light 26 exposures March to June 1925.





1 & 2.

E.S. age 1 year.
Date. 30-5-24.
Condition: Extreme Active Rickets.
Note. Multiple green-stick fractures: phantom bones containing very little lime: fraying of metaphyses.

3.

E.S. 10 mos. later.
Date 20-3-25.
Condition: Rickets still active.
Treatment, Cod Liver Oil Emulsion.

E.S. after ultra-violet light treatment.
Date 22-5-25.
Condition. Healed Rickets.
Note. Rapid result of u.v.r. treatment as compared with cod liver oil.

Result: Enormous change: child bright and running about actively: gained average of 18 oz. in weight per month during course: X-Ray showed healed rickets with well-formed epiphyseal lines, bones well calcified and all greenstick fractures healed.

Case 13

M.McC. age 1 5/12 When seen in Jan. 1924. Florid Rickets, with head bossing, beading, enlarged epiphyses, bow legs, + muscles soft and child unable to walk. X-Ray showed frayed out and concave epiphyseal margins.

First Treatment: Jan. - April 1924 general hygiene: massage: passive movements and Malt and Cod Oil, (cod liver oil alone not being tolerated).

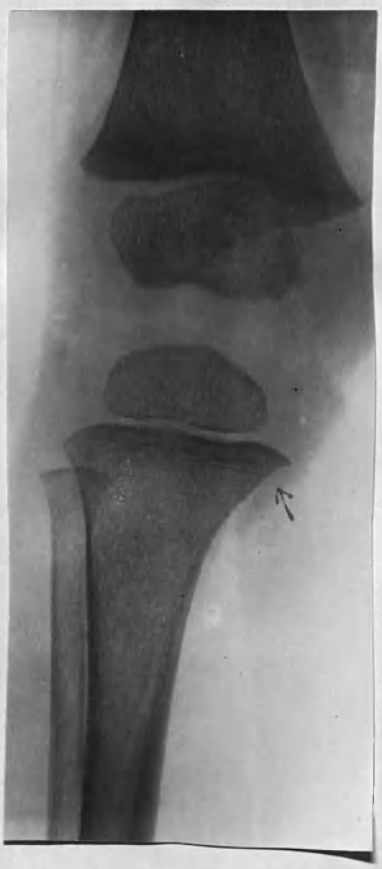
Result: Negative: X-Ray showed slightly increased calcification at epiphyseal line, but rickets still very active.

Second Treatment: (15 months later, by which time bow legs were +++ U.V.Light 44 exposures Aug. - Nov. 1925.

Result: Running about actively: legs straight and child in excellent condition, gained average of 27 oz. in weight per month during course. X-Ray shows healed but irregular metaphyseal line with dense transverse linear shadow ¼ inch up the shaft, i.e. evidence of previous well-marked rickets.

Case 14

M.A.C. age 1 5/12 had had Cod Oil about 48 oz. intermittently when seen on 22/10/25. Anterior Fontanelle admitted two fingers; beading of ribs marked; dentition delayed; a fat flabby child just beginning to walk and tottered on her feet. Was unable to stand till 14 months old. Had frequent enteritis and bronchitis. X-Ray showed typical intermittent and healed rickets, there being several transverse linear shadows near proximal epiphysis of tibia.



I.

M.A.C. 1 yr. & 5 months. 9-10-25. Evidence of previous rickets in case treated by Cod Oil at home, probably administered intermittently. The horizontal lines of shadow at the upper end of tibia denote several successive attempts at healing within short intervals of one another.

II.

M.A.C. 4-12-25. After 12 exposures, and Cod Oil given more regularly, The condition of the bones remains much as before, but the child is walking very much better and general health greatly improved.

Treatment: U.V.Light 20 exposures over 2½ months, plus Cod Oil.

Great General improvement: running about actively and muscle tone much improved. Gained 2½ lbs weight and Haemoglobin increased by 10%. X-Ray showed as before healed rickets, with evidence of previous intermittent rickets.

Case 15

A.C. age 1 year, when seen on 30/12/24. Severe rickets, with bossing of head, large rosary, Harrison's Sulcus, and enlarged epiphyses. Two teeth erupted. Pale: sweated about head: has soft muscles and unable to stand: tendency to enteritis. No X-Ray examination.

First Treatment: Cod Oil 12 oz. during Jan. 1925.

Result: Practically no improvement: enteritis continues:

Second Treatment: U.V.Light 26 exposures without Cod Oil. Feb. to April 1925.

Result: Immensely improved: gained 2½ lbs in weight in 2½ months: no diarrhoea. Child bright and active and able to walk and run about. This child walked for first time after 3rd exposure to u.v.light.

Case 16

E.T. age 1 6/12 when seen on 9/9/24. Anterior fontanelle admitted two fingers, beading of ribs marked, and epiphyses enlarged. Child very pale, had soft muscles, and was just beginning to walk in a tottering fashion.

First Treatment: Cod Liver Oil about 20 oz. Sept - Dec. 1924.

Result: little improvement.

Second Treatment: 39 exposures to u.v.light Feb - June 1925. plus Malt and Cod Oil equivalent to 20 oz. Cod Oil

Result: Gained 2 lbs weight. Immense general improvement: bright, active and running about easily.

Anterior Fontanelle filled in with hard bony membrane.

X-Ray shows epiphyseal lines well formed.

These cases show the great superiority of results obtained by light treatment over treatment by cod liver oil. The mothers were much more enthusiastic over light treatment and willingly put up with the inconvenience of coming to the clinic two or three times weekly for about 3 months. The improvement in the children's general condition was very striking: brightness, happiness and well-being were conspicuous features. There was in all cases rapid repair of the calcification defects in the bones as shown by X-Ray. Muscle tone was greatly improved. Complications such as enteritis and bronchitis disappeared. Relapses (as in case 10) were exceedingly rare and were dealt with successfully by a further course of u.v.light treatment.

(c) Third Period 1925-26.

Treatment by Ultra-Violet Light.

This, in the experience of the writer, is the treatment par excellence, for active rickets, in all cases where natural heliotherapy is unobtainable. In the appended tables, an attempt has been made to summarise particulars of age, weight, symptoms, clinical and radiographic signs, treatment and result in 121 cases of rickets which were treated as out-patients by artificial sunlight, at the Carnegie Child Welfare Centre, Motherwell.

Diagnosis: The methods of diagnosis employed were clinical and radiographic only.

Biochemical tests for calcium retention, and blood examinations for diminution of inorganic phosphate could not be made. Hence many of the slighter cases of rickets were almost probably overlooked and only the grosser cases, exhibiting definite clinical and radiographic signs were treated.

Records: A card was made out for each patient at the commencement of the course setting forth the name etc. age, weight, clinical and X-Ray findings and in some cases the haemoglobin percentage.

At monthly intervals, or thereby during the course of treatment, particulars were entered on the card as to the number of exposures made to ultra-violet light, the weight, haemoglobin percentage, and clinical and radiographic progress.

The following were the chief clinical findings at the outset in the 121 cases treated:-

Clinical Findings. Head Bossing was present in all but 4 cases. It was minimal in 7 cases; very pronounced in 7 others, and moderate or considerable in 104.

Anterior Fontanelle: of 52 cases whose ages ranged from 18 months to 3 years, 45 had the fontanelle still open, and only in 7 cases was it closed.

Craniotabes: This was found generally in the younger infants between 4 and 12 months of age who exhibited a severe degree of rickets. Of the 121 cases, 24 showed craniotabes and only 4 of these were more than 1 year old.

Teeth: A record was kept of the condition of the teeth in 62 cases, and of these 34 had delayed dentition, and caries was already present in 27.

Beading of Ribs: This sign was found to be very constant, being absent in only five cases of the series.

Enlarged radial epiphyses: was noted in every case but four.

Harrison's Sulcus: This sign was found present in varying degree in 88 of the cases.

Pot Belly: Protuberance of the abdominal wall to this extent was found in 76 of the cases, all of which exhibited a Harrison's Sulcus in addition.

Deformity of Limbs: Only the grosser deformities were recorded. Bow Legs were exhibited in 31 cases: Genu Valgum in 20: Antero-posterior tibial curves in 12: and marked flat foot in 6 cases.

Head Sweating and Pallor: are symptoms commonly present in active rickets, but as they are common to many diseases, they are only of significance when associated with osseous changes or radiographic signs. Head Sweating was a feature in no less than 76 of the cases, and pallor in 83. Pallor was of the extreme waxy or greenish (chlorotic) type in 17 cases: and in some of those in whom haemoglobin estimations were made, the haemoglobin averaged about 35% on the Tallquist Scale. (normal on this scale = 80%)

Standing and Walking:- Fifteen of the cases aged 1 year or more were unable to stand at the beginning of treatment, and no less than 32 of such age were unable to walk.

Bronchitis and Enteritis:- 17 cases showed evidence of bronchitis and no less than 40 suffered from intermittent diarrhoea.

Radiographic Findings. A preliminary radiogram of the wrist or ankle was made in 90 of the 121 cases.

The earliest radiographic sign of active rickets is a slightly "frayed out" appearance of the line of junction (metaphysis) between the diaphysis and epiphysis of the more quickly growing bones e.g. radial and ulnar epiphyses of the wrist. Normally this metaphyseal line should be of sharply cut distinct outline and of regular straight or slightly convex form.

The convex line becomes concave as the epiphysis enlarges under the influence of continuously active rickets, especially in cases where there is considerable movement and stress on the limb. Healing is denoted by a dense line of shadow appearing, transversely in the metaphysis, and by increase of periosteal calcification.

An attempt has been made to differentiate roughly the

radiographic findings into active and healing grades or stages as follows:-

Radiographic signs of Rickets.

Active.

Grade A.1. Slightly "frayed" appearance with or without slight concavity of metaphyseal line. (Radiogram No. 53)

" A.2. Slightly "frayed" metaphysis, plus osteoporosis of shaft.

" A.3. Gross fraying. concave shape or cupping and spreading out of the metaphyseal line: osteoporosis of shaft:in addition, there are commonly greenstick fractures of shaft and curvatures. (Radiogram Nos 16, 38, 45 etc)

Healing.

Grade H.1. Irregularity of form of metaphyseal line due to dense shadow of calcium deposition more in one part than another of a previously concave metaphyseal line. (Radiogram No 16, 83)

(N.B. This is the first sign of healing in form A.3).

" H.2. Several densely-calcified lines extending partially or wholly across the end of the shaft and separated from one another by minute lengths of spongy bone.(N.B.These are evidence of previous intermittent partial attempts at healing active rickets). (Radiogram Nos 16, 62.)

" H.3. Densely calcified metaphyseal line with close texture of the bone immediately above it in the diaphysis. The line is regular or irregular according to the degree of previous disturbance of calcification and the course of the healing process.(N.B.This denotes healed rickets). (Radiogram No 5) 18 (11), 38 (11), 45, 53, 62 (11) etc.)

In any given case, intermediate grades or combinations of these grades may be found.

Other signs of healing rickets which may be present along with any of the above are(1) an increase of periosteal calcification along the shaft(2) dense calcification of greenstick fracture.(Radiogram No 113) (Radiogram Nos 89, 102.)

Straightening of Deformities.

The straightening of curvatures of the long bones occurs by great increase in thickness of periosteal bone in the concavities formed in the shaft, and remoulding of the whole shaft by continuous ossification in the new line of the shaft through which the weight or stress passes.

Of the 90 cases which were X-Rayed at the commencement of treatment 66 were classified as active rickets as follows:-

Radiographic Classification.	No. of Cases	
A1	16	(Two of these clearly exhibited greenstick fractures).
A2	3	
A3	45	(3 of these clearly exhibited greenstick fractures at time of examination)
A1 H2	2	(These were active rickets and they had previously suffered at intervals from attacks of active rickets which had become partially or possibly completely healed for the time being).

23 were classified as healing rickets as follows:-

H1	6	(These had all recently been very active, but at the time of examination showed some radiographic sign of healing).
H2	11	(These exhibited signs of intermittent rickets apparently healed at the time of examination, although clinically they appeared to stand in need of treatment).
H3	1	(radiographically healed rickets but clinically requiring treatment).
H1 H2	5	(These were cases of intermittent rickets which had again become active and were showing some signs of healing at the time of examination).

1 case presented an appearance of apparently normal bones.

Treatment.

Of the 121 cases, 98 had the full course of 20 or more exposures to the ultra-violet light; 21 cases had between 10 and 20 exposures, and only 2 cases had less than 10 exposures.

A few had considerably more than 20 exposures generally at the urgent request of th mother who was so much gratified by the progress which her child was making.

The exposures were generally made twice weekly and the average length of the course was 2½ months. In a number of cases, the course was interrupted for a period owing to the mother being unable to bring the child for various reasons, e.g. pregnancy, pressure of domestic affairs, outbreak of infectious disease, acute illness etc. (Many cases, of course, dropped their attendance altogether for these

reasons and did not complete the course, but these are excluded from consideration in this paper.)

During the earlier part of this period, the mothers were advised to give cod liver oil concurrently with the light treatment. Later this practice was stopped as it was abundantly proved that ultra-violet light was sufficiently powerful in itself to effect a cure. As far as could be ascertained only 18 cases in this series had cod liver oil in addition to ultra-violet light, in an amount varying from 6 to 20 oz. during the observation period: and 8 others had some small unknown quantity of cod liver oil during the period. There were thus 95 cases in the series treated by ultra-violet light alone.

Diet

The diet was in general, the high carbohydrate diet common amongst the poorer working classes. No special instructions were given as to diet. To the very poorest households 1 pint of milk was given as a routine in respect of children under 2 years. It is not known to what extent each child received this milk, and, in any case, most of the households to which it was given had had it for long periods before light treatment was commenced.

Technique of Ultra-violet light exposures.

The lamps used were 30 - Ampere Carbon Arcs burning ordinary cored carbons, and two Mercury Vapour Quartz lamps of the vacuum type. The children sat round the lamps naked at a distance of $2\frac{1}{2}$ to 3 feet from the lamps. The commencing period of exposure was generally 10 to 15 minutes of the Carbon Arcs, and, in addition, 1 - 3 minutes of the quartz lamp, according to the age and condition of the burner in the latter, and other factors. The irradiation period was gradually increased to 30 minutes of the Carbon Arc and 10 minutes of the quartz lamp, thus making the full session one of 40 minutes duration. Generally, 15 - 20 children were thus accomodated round the lamps in one session, though occasionally larger numbers had to be admitted, and this caused the children to be placed further away from the lamps and to receive correspondingly less irradiation.

The mother of the child attended each session, undressed the child in a waiting room, and after the irradiation, she bathed and dressed the child in an adjoining bath-room.

Results.

Increase of Weight.

The weight of each patient was recorded at the beginning and end of the course of treatment and, as far as possible, at monthly intervals during the course. From these data the average gain (or loss) in weight per month for each patient during the irradiation period has been calculated, and is shown in the table.

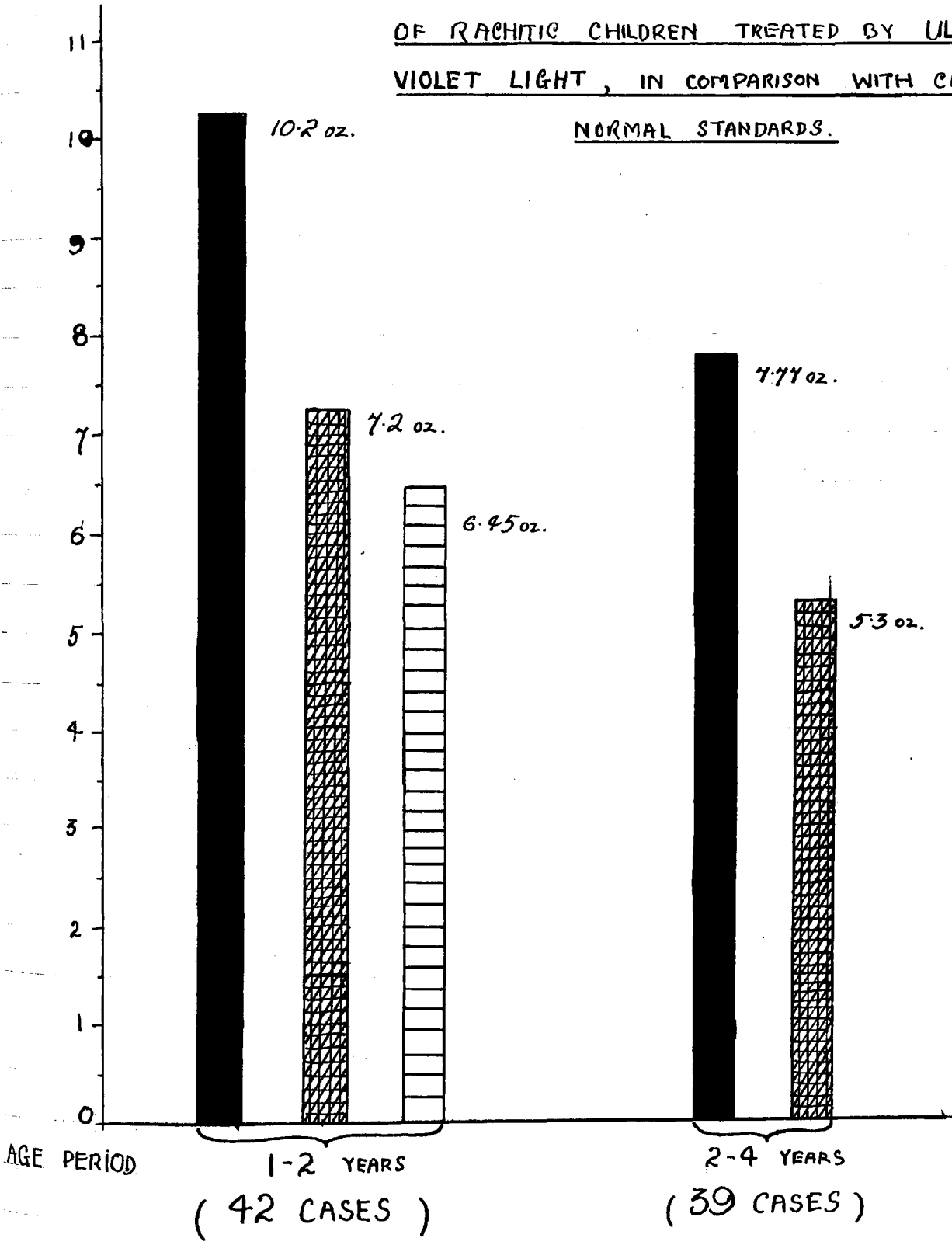
For purposes of comparison with gain in weight of normal children, these monthly increments of weight have been averaged up for the age periods (a) 6-12 months, (b) 1-2 years and (c) 2-4years, with the following results:-

(a) Age Period 6 - 12 months. (14 Cases)

Children whose age at the commencement of the course of treatment was between 6 and 9 months had the great bulk of their treatment during this period.

OUNCES.

GRAPH SHOWING AVERAGE GAIN IN WEIGHT (PER MONTH) OF RACHITIC CHILDREN TREATED BY ULTRA-VIOLET LIGHT, IN COMPARISON WITH CERTAIN NORMAL STANDARDS.



(42 CASES) (39 CASES)

RACHITIC CHILDREN TREATED BY ULTRA-VIOLET LIGHT.

NORMAL CHILDREN (AFTER STILL)

ALL CHILDREN ATTENDING CHILD WELFARE CENTRES AT WOOLWICH (FROM MACMILLAN).

Total of average monthly increments of weight	141½ oz.
Average gain in weight per month	10.1 oz
cf. do. for normal infants (Pfeiffer)	16.5 oz

(b) Age Period 1-2 years. (42 cases)

for this period the children whose ages range from 11 months to 1 year and 10 months at the beginning of the course have been selected.

Total of average monthly increments of weight	428 oz
Average gain in weight per month	10.2 oz
cf. do. for normal infants (G.F.Still)	7.2 oz

(c) Age Period 2-4 years. (39 cases)

Total of average monthly increments	302 oz
Average monthly gain in weight	7.77 oz
cf. do. for normal infants (G.F.Still)	5.3 oz

In each of those groups the gain in weight has been considerably reduced by the inclusion of certain cases which, as shown in the table, lost weight during the course. It must be remembered that the cases dealt with included many who suffered from enteritis, malnutrition and tuberculosis as well as rickets, and, in such cases, no great gain in weight could be expected. Again, many of the infants were of the heavy fat flabby type with protuberant abdomen, and, in general, this type did not make much gain in weight during the first month or so of treatment, but often lost weight as muscle tone improved and the abdomen became more normal. When these facts are taken into consideration the gain in weight noted for the age periods 1-2 years and 2-4 years is surprisingly good. With regard to the young infants aged 6-12 months, these were mostly suffering from severe nutritional disturbance, and the immediate gain in weight, is of course, lower than the normal for the period. Since also, the number of cases is too small for statistical purposes, this age period has been excluded from the graph on p. 624

For the age-period 1 - 2 years, the figures quoted by McMillan may be used as a fairly accurate comparison since they concern the same class of child. He found that the average monthly gain in weight in the second year of life of 447 male babies and 450 female babies who attended the Child Welfare Centres at Woolwich was for males 6·25 oz. and for females 6·67 oz.

Judged by this standard the average monthly gain in weight of 10·2 oz. as recorded above seems to be considerably in excess of the normal. The number of cases is, of course, rather small for accurate comparison, but the figures may nevertheless be considered of some value.

Increase of Haemoglobin: In 17 cases the haemoglobin was measured by the Tallquist Scale at the beginning and end of the course of treatment, and was found on the average to have increased by about 9%. The method of the Tallquist Scale is admittedly a rough one, but it was found by Gamgee in a similar series of cases to be reasonably accurate when tested against other methods. The normal of the scale for young children should be regarded as 80%, not 100% Most of the cases at the outset were found to have about 55% haemoglobin on this scale, and in a few cases, notably those with chlorotic or waxy pallor, the haemoglobin was as low as 35%.

Increase of Muscular Activity.

This was one of the most striking features of the treatment. With the single exception of one child aged 12½ months who had tuberculous glands in addition to rickets (No.62), every child of the age of one year or over at the conclusion of treatment was able to stand and to walk. The increase in activity was remarkable particularly in children, aged about 18 months, who had gone "off their feet". All of these were walking and running about actively, generally in 3 to 6 weeks after irradiation was commenced. One remarkable case (No.112) aged 8 years, an extreme case of rickets with gross deformity in head, trunk and every limb, who had never

walked, was able to rise to her feet in three weeks, to walk about with assistance in six weeks, and at the conclusion was able to run about and play with normal children.

Muscle tone invariably improved enormously except in a few cases suffering from intercurrent disease.

Bronchitis and Enteritis.

I6 cases were noted as having bronchial catarrh before treatment, and only in 3 was it still present after treatment. One of those 3 cases had an incomplete course of only 10 exposures to u.v.light; one lived under inconceivably wretched hygienic conditions in a small room, which was occupied by 9 persons; and the third though not cured was improved.

No less than 39 of the 121 cases were suffering from intermittent or acute diarrhoea when first observed, and when treatment was completed this number was reduced to 7 cases. Of these, five children had less than the full course of 20 exposures to light, and the other two had abdominal tuberculosis with chronic intermittent diarrhoea.

The children with active rickets and enteritis not infrequently had to be suspended on certain occasions for a few days from attendance at the clinic, on account of acute diarrhoea. In time, however, this troublesome complication was generally mastered.

Effect on Rachitic Stigmata.

The full effect of the treatment on stigmata could not of course, be observed within the short period occupied by the irradiations. In many cases indeed there was not much improvement in this respect to be observed for many months. Cases with bow legs, especially those under 3 years, showed the most striking improvement, and not infrequently were found to have their legs almost straight and able to touch each other at the knees, if observed after 9 - 12 months. A photograph of a typical case of this kind is shown on p. ✓ The method of straightening of curvatures can be seen by study of radiograms nos. ✓

The new bone laid down under the periosteum in the concavities

of the bone was gradually increased in thickness, and at the same time new struts of bone were laid down in the shaft in the new line of weight of the limb (see radiogram no. ✓)

At the same time, absorption of the redundant bone outside of the new line of weight took place. In this way a long bone, originally curved, gradually became straightened.

Enlargements of the radial epiphysis can be seen in the radiograms to be greatly reduced as cure proceeded. As the original concave and spread out metaphyseal line, which marked the circumferance of the epiphyseal enlargement became diminished in width and formed a straight line, (see radiogram no. ✓) the epiphysis with which it was joined obviously also became reduced in size.

Radiographic Results.

A terminal radiograph was made in 104 cases of the series. These were classified as follows:-

H1.	5	cases
H2	25	"
H3	73	"
Normal	1	"

Of the 5 cases classified as H1, 2 had less than the full course of treatment, and the other three all exhibited great general improvement, although radiographically healing was slow. In one of the latter cases, a radiogram taken 3 months later was classified as H3.

The H2 cases were mainly those which at the beginning of the treatment showed evidence of intermittent rickets, with or without activity. There were some, however, in which the transverse lines of shadow which indicate intermittent healing became evident only in the terminal radiogram, having presumably been obscured by the broad zone of fraying in the earlier radiogram.

The H3 cases were in a number of instances difficult to distinguish from the normal, but generally one or other of the following distinctive marks determined the classification:-

(1) some degree of irregularity of outline of the metaphyseal line.

(2) closeness of texture of shadow in the shaft immediately above this line.

(3) density of shadow at a point in the shaft corresponding with a previous greenstick fracture.

(4) abnormally thick periosteal ossification in the concavity of a curvature of the shaft.

Thus from the radiographic point of view, the evidence of cure of rickets by this treatment was overwhelming.

Every case in the series which was radiographed showed evidence of healing in some degree and in most cases the healing was complete.

Effect on General Health.

Although this is a condition which does not lend itself to exact classification, the fact was clear and outstanding that the vast majority of the children in this series were vastly improved in their general health. Generally they slept and ate better after a few exposures. Nervous irritability was allayed or disappeared. Spasmophilia, tetany and convulsions were seen no more in every case which had previously exhibited them. Activity, liveliness and brightness became marked characteristics.

The mere fact that the mothers brought the children for treatment so many times in spite of much hardship and great inconvenience showed that they were convinced of the great value of the treatment to their children. Some of them expressed their verdict in the following terms:-

" a different child, ever so much livelier"; "better in every way"; "a wonderful change"; "a great difference"; "more intelligent looking"; "can run like the mischief now"; "got on marvellously"; "can walk much further without taking rests"; "very active - I cannot keep him in now"; "smart on his feet now"; "it has put more life in him";

Careful scrutiny of the records shows the following effects on general health in the 121 cases:-

very well and greatly improved	102 or 84%
Improved but not quite well	19 or 16%
	121

Of the 19 cases which were classified as "improved but not quite well" 8 had tuberculosis of osseous or glandular systems in addition to rickets, 6 were very severe cases in whom the course was too short to effect the full improvement, 2 had inter-current whooping cough, 1 had extreme anaemia, 1 had threadworms, and 1 lived under indescribably bad hygienic conditions.

Considering that these children were mostly the products of the

slums in a very poor and congested industrial area, the restoration to health of 84% of them in the course of two or three month's treatment appears to be a very considerable achievement. Indeed, it is not too much to say that it caused something of a sensation in the districts.

After History. Although these cases were constantly being supervised in their homes by health visitors and referred to the clinic in case of need, not more than three or four of the 1924 - 25 cases were found to require further light treatment in the following winter. These few cases had become pale and toneless again, but none of them showed recurrence of radiographic signs of active rickets.

General Summary and Conclusions.

1. The conception of the disease rickets has, in recent years been widened to include the "status calciprivus", which may exist in varying degree, and for varying periods and which may alternate in a series of waves with phases of improving or normal calcium metabolism. Biochemical work in particular, suggests that the essential metabolic disturbance may be present without obvious osseous changes in the conditions known as latent or incipient rickets. Low calcium retention and diminished inorganic phosphate in the blood may be used as tests of this condition.

2. Calcium starvation may in some cases play a part in the causation of human rickets, but, generally, the causes of the disturbance of calcification in rickets are much more profound, than mere deficiency of calcium in the diet.

3. A diet ill-balanced in mineral content, particularly calcium and phosphorus, tends to defective retention of both these elements, but retention is greatly improved, even on such defective diets, by exposure of the skin to ultra-violet light, or by inclusion of cod liver oil in the diet.

4. Inorganic phosphorus in the blood has been found to be reduced in active rickets, while calcium in the blood may be normal or slightly reduced. The exact bearing of these facts on the full story of calcium and phosphorus metabolism has not yet, however, been elucidated.

5. It has been suggested that a condition of acidosis produced by various depressants of food metabolism, e.g. excessive heat or moisture, air stagnation, darkness, confinement, lack of vitamine, precedes the onset of rickets. The theory is attractive, although the accepted biochemical facts as to the shifting of the acid-base balance of the blood do not yet include a clear account of the rolle of the calcium salts in the process.

6. Study of the world distribution of rickets may be said to show that generally the sunny countries of the south, and the extreme northern or arctic regions are almost free from rickets. Rickets is chiefly found in overcrowded industrial districts and cities, especially where there is much smoke and mist in the air.

7. The incidence of rickets in such cities has been estimated at 50% to 80% and even higher if the slighter forms of rickets are included, and the examination made in March, during which month the disease is most prevalent.

In a Scottish mining and steel town of 70,000 population, the incidence of clinical rickets with osseous signs amongst children aged 6 months to 3 years, was found by the writer to be about 50% or even higher.

8. The confinement or domestication theory of the causation of rickets must be modified by the discovery of the potent effect of sunlight and ultra-violet light in the cure of rickets. While the essential metabolic disturbance involved may be contributed to by various factors connected with confinement, the most potent of these factors appears to be lack of ultra-violet light.

8. There is much experimental evidence that rickets may be caused in animals by feeding them on diets deficient in a fat-soluble accessory food factor to which the name vitamin D has been given. The disease does not develop, however, if the animals are exposed during the same period to sunlight or ultra-violet light. Cod liver oil is the substance known to be richest in vitamin D. and rickets has been prevented and cured both in infants and young animals under experimental conditions by the exhibition of cod liver oil. On the other hand, in out-patients, both the prevention and cure of rickets by cod liver oil has been attended with varying success, and even, in many cases with comparative failure.

Recent experimental evidence suggests that the content of vitamin D in cow's milk depends upon exposure of the cow to sunlight, and not upon its diet.

It has also been shown that human milk is not uncommonly devoid of anti-rachitic properties and that these can be developed in it by exposure of the mother to ultra-violet light.

Evidence has been adduced that the fat of certain cereals, oatmeal in particular, contains an anti-vitamin which tends to produce rickets

Adequacy and good balance of calcium and phosphorus in the diet is insufficient to produce well calcified bone in the absence of vitamin D or of exposure of the young animal to ultra-violet light; and the greater the disproportion or defect of these minerals, the more important is the role of the vitamin or of ultra-violet light.

There is a considerable weight of evidence that adequate exposure of the young animal to sunlight or ultra-violet light both prevents and cures rickets, even under the wide variety of adverse dietetic and hygienic conditions to which young children are subjected in practice in all parts of the world. In addition, exposure to ultra-violet light has generally a much more beneficial influence on the general health of rachitic infants than dosage with cod liver oil, although the latter may cure the rickets.

The available evidence is strongly in favour of rickets being a disease of nutrition rather than an infectious disease.

It is probable that under different conditions many different hygienic defects may conduce to the rachitic state, but in practice, in industrial areas in this country, the chief determining factors appear to be lack of exposure either of mother or child to actinic light, and lack of well-balanced diet rich in vitamin D. The former appears to be the more powerful factor, although each can to some extent compensate for the other.

Rickets is hereditary only in so far as the various rachitic influences, chiefly lack of ultra-violet light and possibly deficiency of vitamin in diet, acting upon the mother during pregnancy and lactation, bring about disturbed mineral metabolism

and the "status calciprivus" in the offspring. If such a child is not subjected to intense anti-rachitic influences, clinical rickets may occur at a very early age.

13. Recent evidence shows that fully 80% of deciduous teeth in children, when microscopically examined, are of defective structure, and that these teeth are particularly liable to caries. As dental hypoplasia is essentially of the same nature as rickets, it becomes evident that the rachitic condition may be responsible for the widespread damage to health associated with dental caries in children.

14. Broncho-pneumonia and diarrhoea in children, especially of the severe and often fatal type which follows measles is, in the limited experience of the writer, very frequently associated with rickets.

15. There is some evidence that enlarged tonsils and mouth breathing in school children are almost exclusively confined to those who exhibit signs of old rickets.

16. The present writer as a result of experience in treatment of many cases of rickets attending as out-patients at a Child Welfare Centre in a populous Scottish industrial area during the last seven years has arrived at the following conclusions:-

(a) Cases in which treatment was attempted by instruction in diet, general hygiene, and dosage with cod liver oil exhibited few successes, and there was rarely striking improvement in general health. In many cases cod liver oil was not well tolerated or, at any rate, was not, for various reasons regularly given in practice, although prescribed.

In contrast with this experience, there is some evidence that when a diet very rich in vitamines and low in cereal content is recommended, together with cod liver oil, a much greater proportion of successes can be obtained.

(b) Cases treated first by the above methods and latterly by exposure to ultra-violet light, showed the great superiority of the latter method to the former. In particular, the improvement in general health became most marked, after light treatment had been adopted.

(c) In a detailed investigation of 121 young children treated by ultra-violet light to a conclusion, and of which full clinical and radiographic records were kept as shown in the table, the results indicated conclusively that ultra-violet light practically never fails to cure rickets in out-patients provided they attend sufficiently; and, in general, weight is put on at a greater rate than the normal, especially in the second year of life, haemoglobin is increased, muscular tone and activity is greatly improved, nervous irritability is allayed, bronchitis and enteritis are often cured, or ameliorated, and general health is so improved that the patients become like different children.

If rickets is extreme and the infant very young, ultra-violet light generally effects an almost immediate improvement in the child's condition, but radiographic signs of healing of active rickets are not evident until about three weeks after irradiation is commenced. Generally 20 exposures to light over a period of 2 - 3 months is sufficient for cure. Older children with active rickets require more irradiation, generally 30-40 exposures, or even more for complete healing. A much shorter irradiation period, however, serves to set the healing process in motion, and, in ordinary circumstances, healing continues to advance after irradiation has been stopped. Curvatures of the long bones, due to minute greenstick fractures in the active period of rickets, are greatly straightened out in the process of cure, by the deposition of broad zones of new bone in the concavities of the shafts, and throughout the bone in the new line through which the weight of the body or stress of the limb is borne.

Very bad conditions of feeding, especially lack of vitamin D and ill-balanced diet, and also very poor hygienic conditions, such as overclothing, overcrowding, lack of ventilation and light, delay but do not prevent the cure of active rickets within a reasonable period by irradiation with ultra-violet light. Intercurrent acute disease is a temporary contra-indication to

light treatment, but generally, irradiation can be safely resumed after the acute stage has passed, and such conditions as whooping cough, measles, bronchitis, enteritis and even tuberculosis in children are, perhaps, best treated in this way.

Radiograms and Photographs

A series of radiograms and photographs are included in the appendix to demonstrate the degree of rickets from which many of these children were suffering and the way in which calcium became deposited in the growing ends of the bones as the treatment progressed and healing occurred. The radiograms were made under difficulties with an apparatus which required 3-5 secs. exposure for the wrists, and no intensifying screens were available. Hence, in many cases, the series of radiograms was spoilt owing to the child moving during this long exposure.

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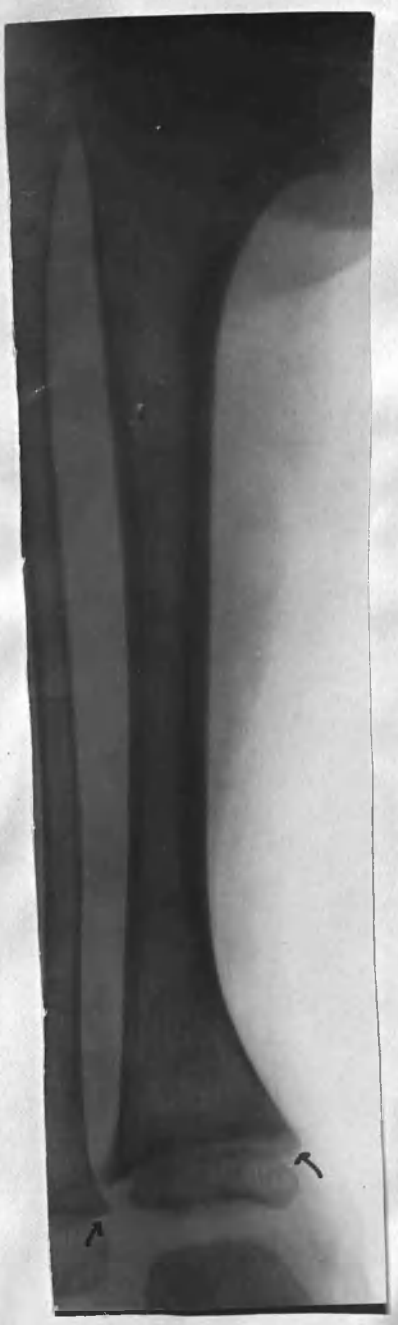
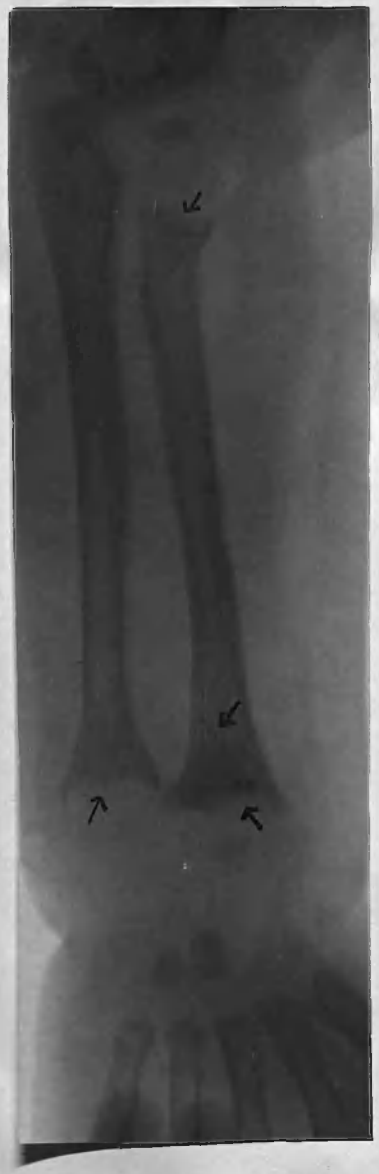
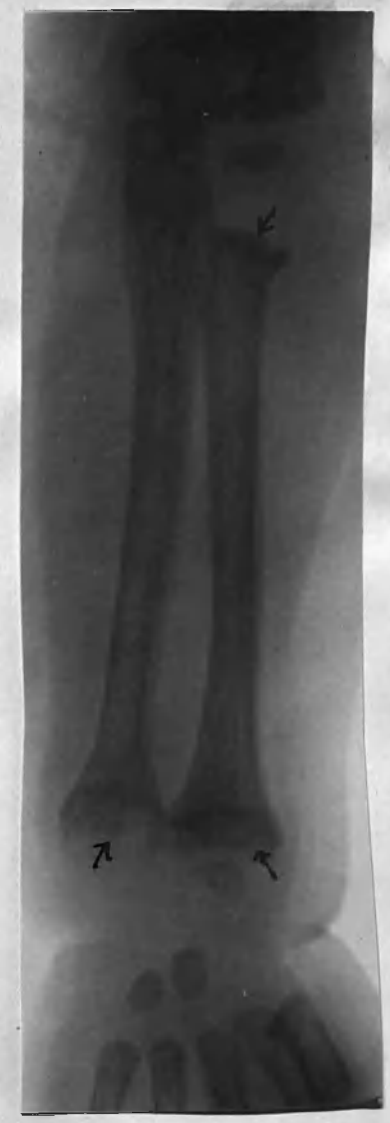
Children with rickets under treatment
by Carbon Arc lamps.

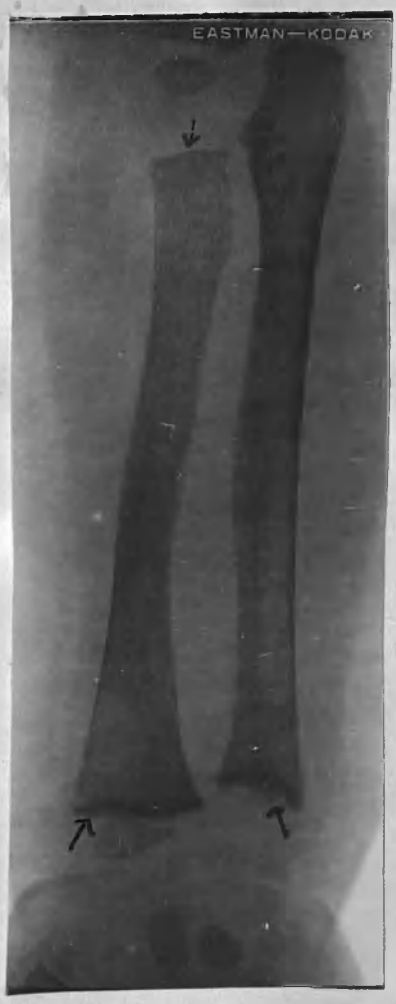


No.16. E.B. Age 2 yrs. 3 months.
Date. 3.4.25.
Classification. A 3.
Note extreme spreading, fraying and hollowing at all metaphyses: osteoporosis marked: periosteal calcification poor.

E.B. 1 year later, She had 9 exposures in April 1925. Then was unable to come for treatment till Dec. 1925, when she commenced a further course of 26 exposures.
Date. 31.3.26.
Classification. H2,
The metaphyses are densely calcified and in the tibia two faint transverse lines are seen ¼" above metaphysis: denoting previous rickets with patial attempts at healing. Much periosteal bone has been laid down, thus tending to straighten the curvature.







I.

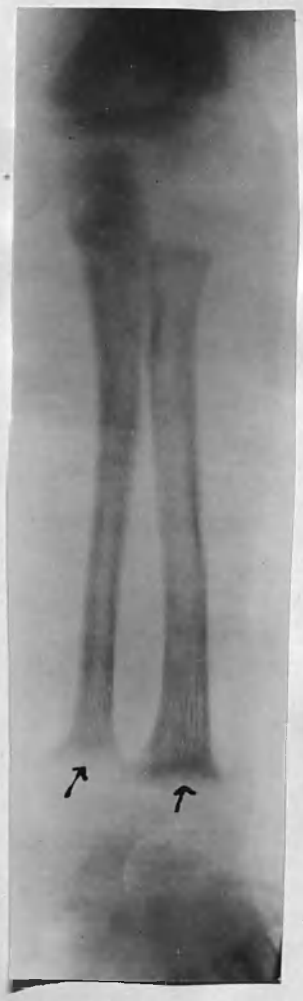
No.18 E.B. age 2 yrs, 4 months.
Date. 5.3.26.

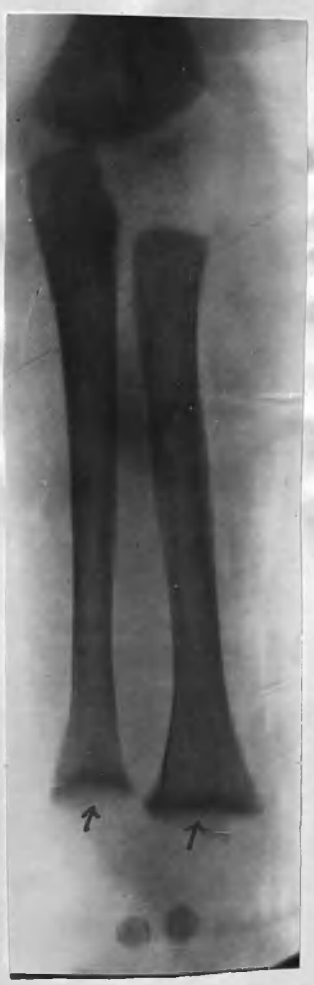
II.

E.B. after 13 exposures.
Date. 14.4.26.
Classification. H1.
Note dense line of shadow appearing in all metaphyseal lines.

III.

E.B. after 27 exposures.
Date. 12.8.26.
Classification. H3.
Note fairly clear but slightly irregular outline of all metaphyses. "spreading" and "concavity" has bee been almost abolished. Periosteal calcificatio has greatly thickened the shafts and there is no ostepoorosis. Centres of ossification are larger and well calcified.





I.

No. 38. T.D. age 9 mths.
Date. 5.3.26.
Classification. A3,
Note extreme spreading and fraying of metaphyseal lines of radius and ulna: ostepporosis of shafts: ill-defined centres of ossification in carpus.

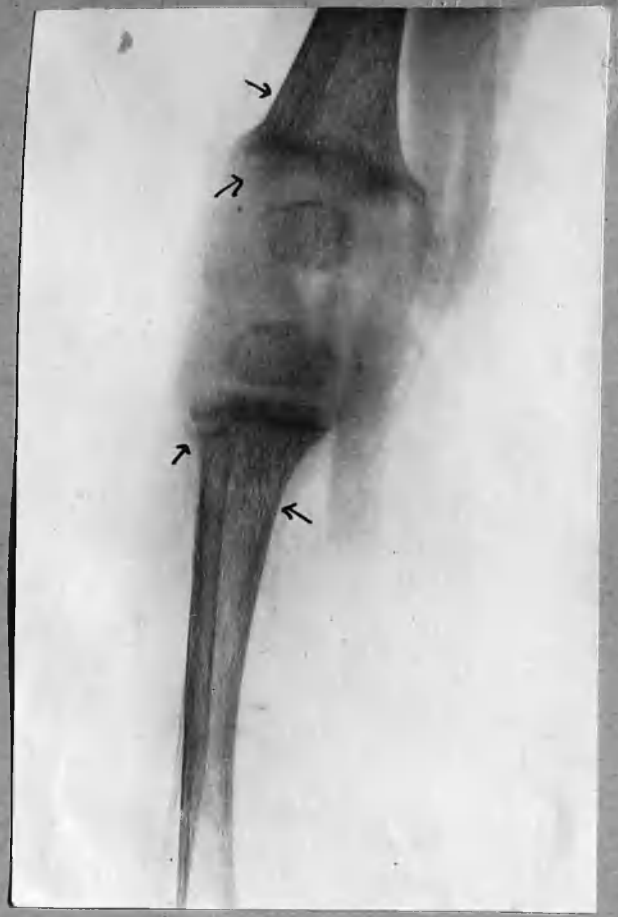
5/12

II.

T.D. 2½ months later, after 32 exposures.
Date. 19.5.26.
Classification H 3.
The metaphyses are fairly well-calcified: Shafts are thickened by increase of periosteal calcification

III.

T.D. 5 months after commencing treatment. (37 exposures).
Date. 12.8.26.
Classification. H3.
The bones have become almost normal in appearance. The metaphyseal lines are, however, denser than normal.
Carpal centres are well ossified.



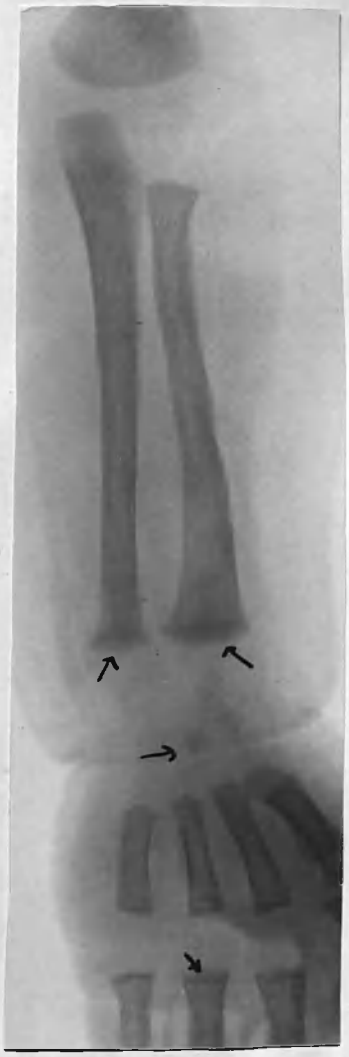
Nº 45

I.

F.G. Age 9 months. 25-5-25. This was a fat flabby child with marked signs of rickets, including craniotabes, and in addition there was a history of 22 convulsion fits. The X-Ray film shews the typical fraying of the growing ends of femur, tibia and fibula, as well as deficiency of calcification of the bones generally (osteoporpsis) The centres of ossification of the epiphysis at the knee joint are very poorly calcified.

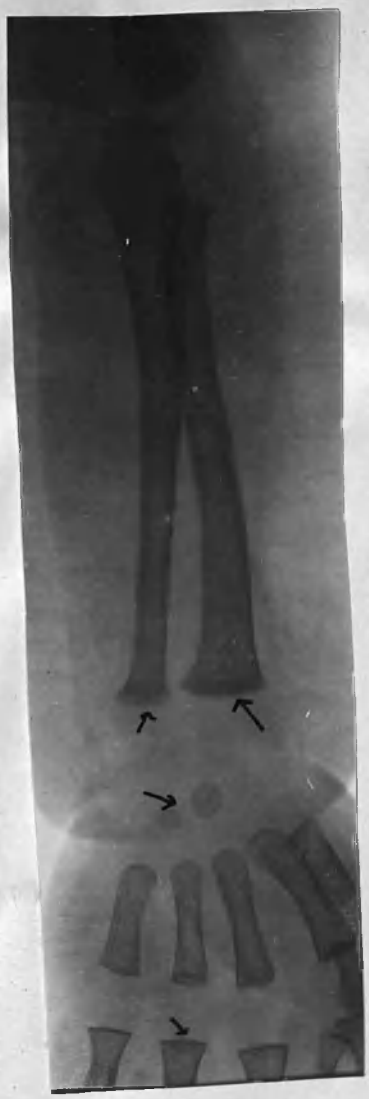
II.

F.G. 2-10-25. After 30 exposures. No exposures during June and July owing to Whooping Cough intervening. No further fits occurred after light treatment was commenced. The X-Ray film is typical of healed rickets with densely calcified line at the growing ends of the bones, good calcification throughout the bones and in the centre of ossification of the epiphysis.



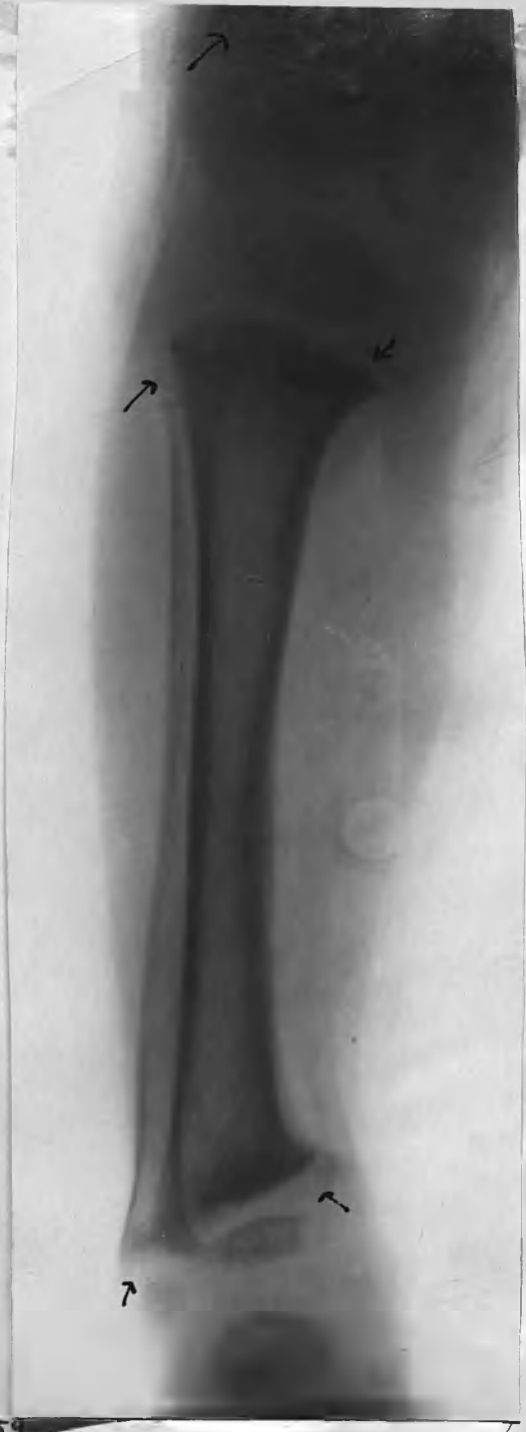
I.

No. 53. J.H. age 4 months.
Date. 30.4.26.
Classification. A1.
Note fraying and spreading of distal radial and ulnar and of proximal phalangeal metaphyseal lines: some osteoporosis: and ill-defined carpal centre of ossification.

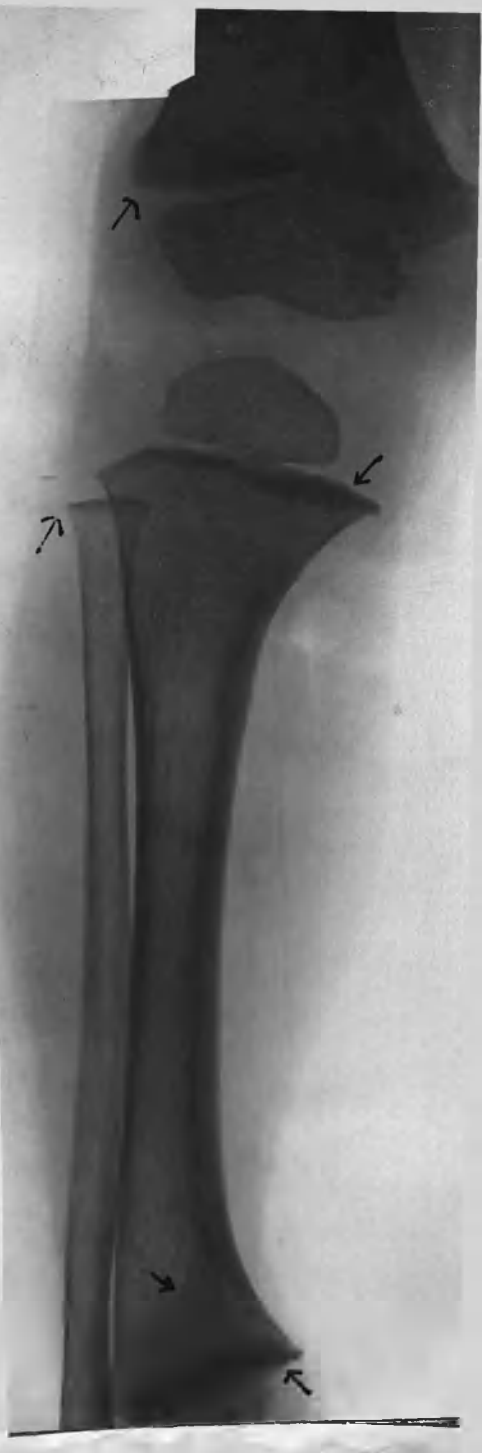


II.

J.H. after 16 exposures.
Date. 9.8.26.
Classification. H3.
Dense shadow of healing at metaphyses of radius, ulna and phalanges (that of ulna not being quite complete). Fine meshing of shadow of shaft. (no osteoporosis) and well-defined carpal centres of ossification.



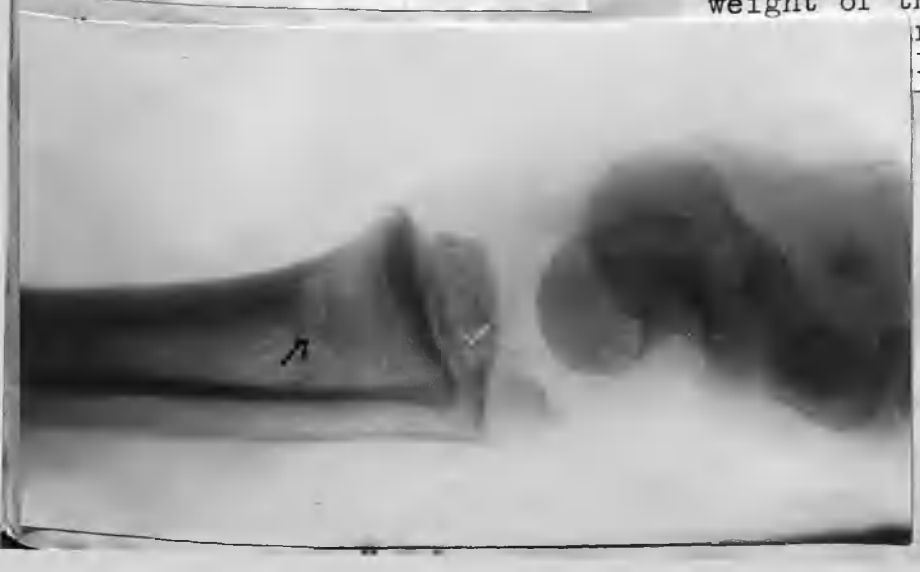
E.L.1 yr. after treatment to show degree of straightening of bow legs.



Nº 5

E.L. Age 1 year & 8 months. 19-6-25.
Florid or active rickets in a child of fair nutrition and muscular activity.
Note the frayed out growing ends of femur, tibia and fibula. Note abnormal curvature of tibia with formation of osteoid tissue in the hollow of the shaft. This child had bow legs.

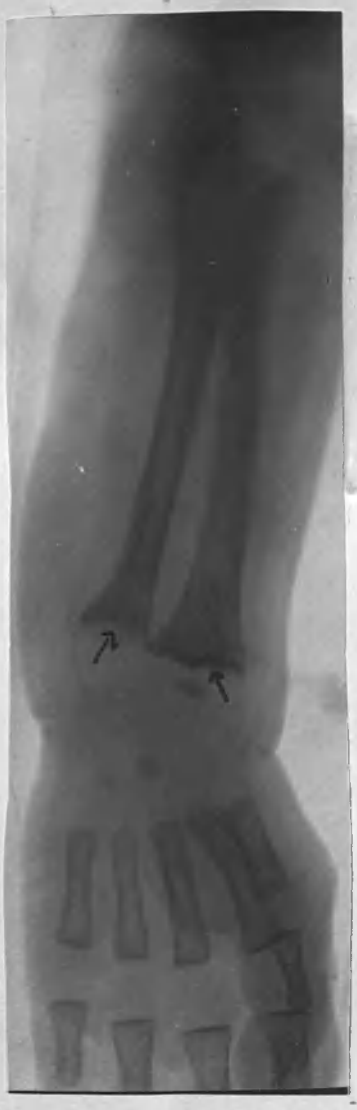
II.
E.L.13.11.25. After 47 exposures. Healed rickets. Note line of dense shadow at all the growing ends - femur, tibia and fibula. Not the general increase of density of the bones and of the centres of ossification. Note also the dense thick zone of subperiosteal bone in the tibia, and near the lower end the struts of new bone being formed along the line of the weight of the body. This illustrates ...res in bone become straightling advances.



III.
E.L.12.3.26. Shews straightening of tibia proceeding by:-
(1) increase of thickness of periosteal bone in the concave aspect.
and
(2) new struts of bone formed along the line of weight.

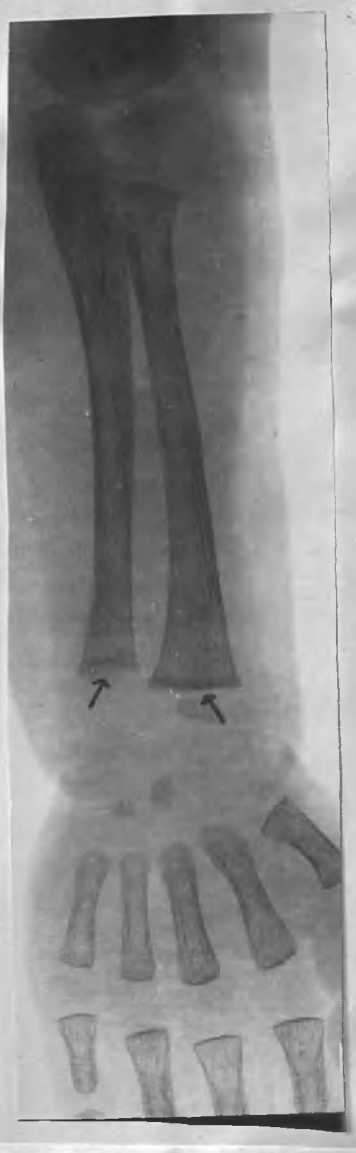
I.

No.62.
J.K. age 10 mos. wt. 10¼ lbs.
Date. 30.4.26.
Classification. A3
Note extreme osteo-porosis: fraying. hollowing and spreading of metaphyseallines in radius, ulna and phalanges: ill-defined centre of ossification in carpus. A severe degree of rickets for a mal-nourished infant.



II.

J.K. after 8 exposures as outpatient, followed by heliotherapy in Hospital.
Date. 4.6.26.
Classification. H2.
There is dense irregular deposition of calcium in metaphyseal lines, and above this, in the radius a second transverse line showing evidence of previous rickets with an attempt at healing.

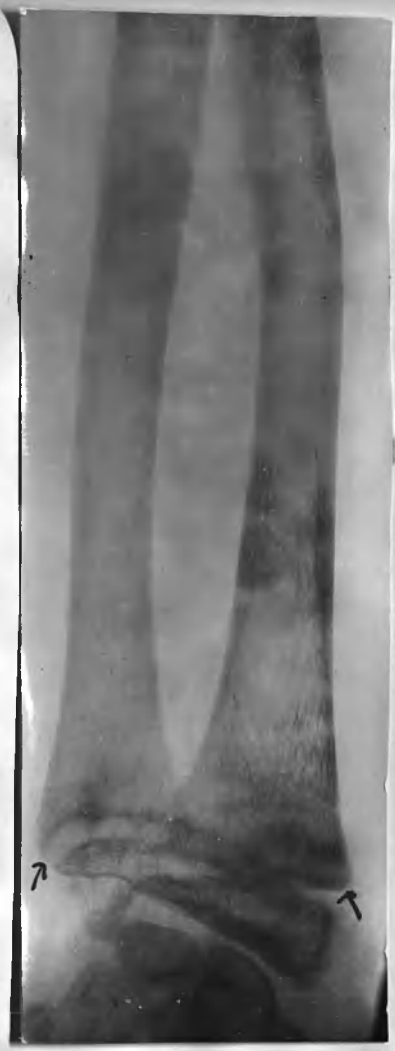


III.

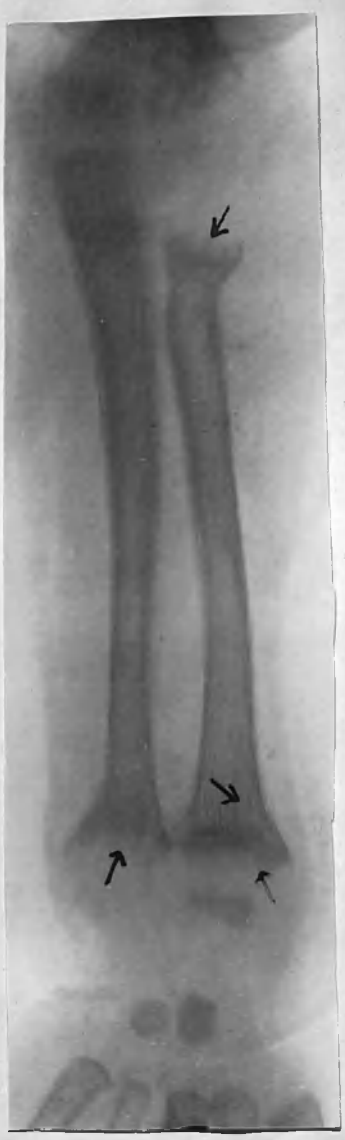
J.K. after 13 exposures and heliotherapy.
Date. 9.8.26.
Classification. H3.
Healed rickets with dense regular meta-physeal lines in all bones: no osteoporosis.

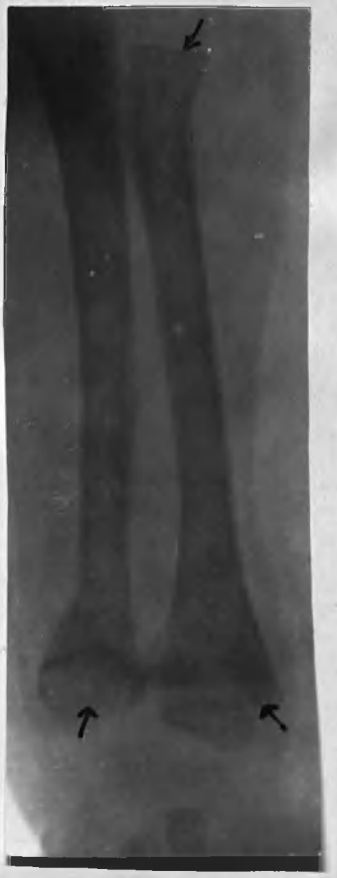
№ 74.
W.M. Age 14 years. - Late Rickets still active.

№ 74. II.
W.M. 5-5-26. X.Ray film before treatment was commenced shews active rickets at growing lower ends of radius and ulna, particularly at the ulna. The latter is expanded, cupped and frayed frayed out and there is no well-formed metaphyseal line.



III.
W.M.16-6-26. After 18 exposures. X-Ray film shews healing well advanced at the growing lower end of ulna with a new metaphyseal line fairly well formed.





I.

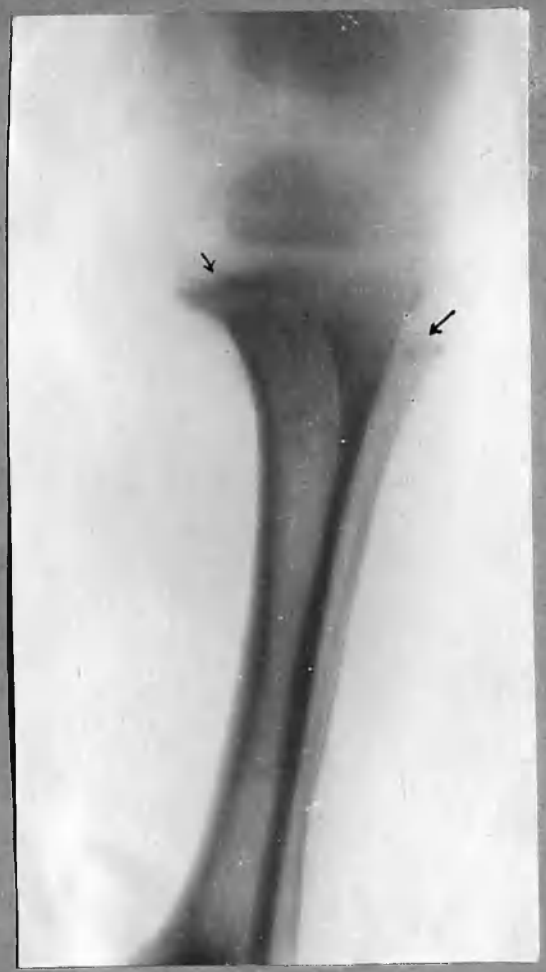
No. 75. D.M. age 2½ years.
Date. 21-5-26.
Classification. A3.

Note extreme degree of active rickets: fraying: spreading: hollowing of all metaphyseal lines, even that of head of metacarpal of thumb: marked osteoporosis: greenstick fracture near head of radius, and near lower end of ulna.

II.

D.M. 6 weeks later, after 11 exposures.
Date. 6.8.26.
Classification. H1.

Dense calcification is beginning to take place in all metaphyses, these are now better defined: osteoporosis much diminished, and carpal centres well ossified. Periosteal calcification proceeding actively at site of greenstick fractures, in order to straighten the bones.



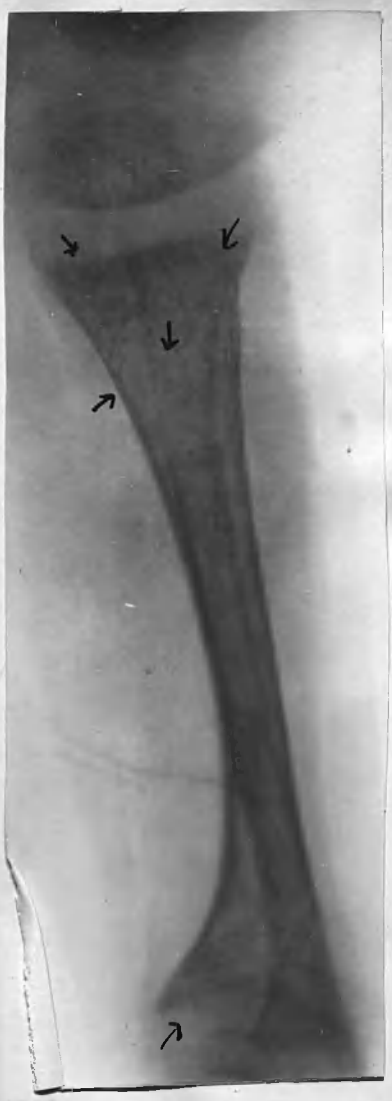
Nº 81

I.

B.McL. Age 1 yr. & 9 months. 3-1-25. X-Ray shews typical lesions of active rickets, with poor calcification ~~of the~~ bowing tibia and fraying out of the growing ends of the shaft.

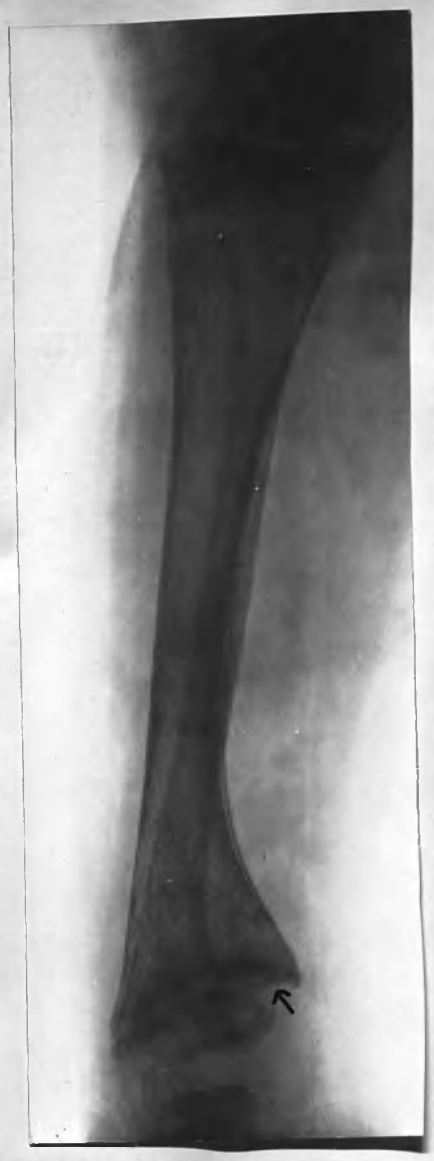
II.

B.McL. 31-3-25. After 24 exposures. X-Ray film shews healed rickets. A new dense line of calcification has been laid down at the growing ends of the bones, and the sub-periosteal osteoid tissue has become calcified. No Cod Liver Oil was given in this case.



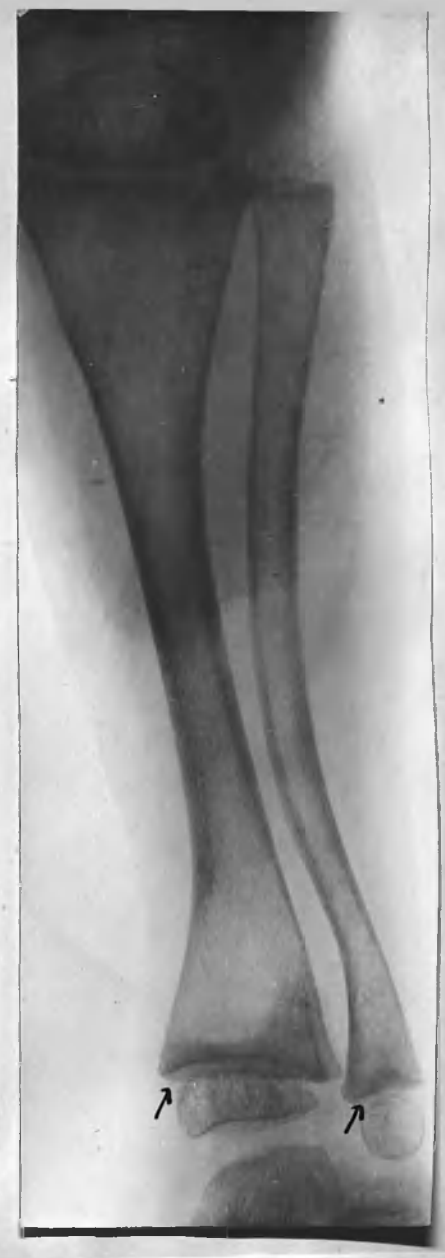
I.

3.M.McF. age 4 yrs.
. 22-1-26.
sification. A.3.
Note extreme fraying
lowing and spreading
ll metaphyses: extreme
rporosis: gross
aturesoof shafts of
us and ulna.



II.

M.McF. after 30 exposures
date. 14-4-26.
Classification. H.1.
Dense shadow at all
metaphyseal lines denotes
healing.



III.

M.McF. after 49 exposures.
date 6-8-26.
Classification. H.3.
all metaphyseal lines are
fairly regular and well
calcified. Straightening
of the shafts is proceeding
by increase of periosteal
calcification in the
concave aspects.
Note the longer course of
treatment required here in
an extreme and prolonged
case of active rickets.

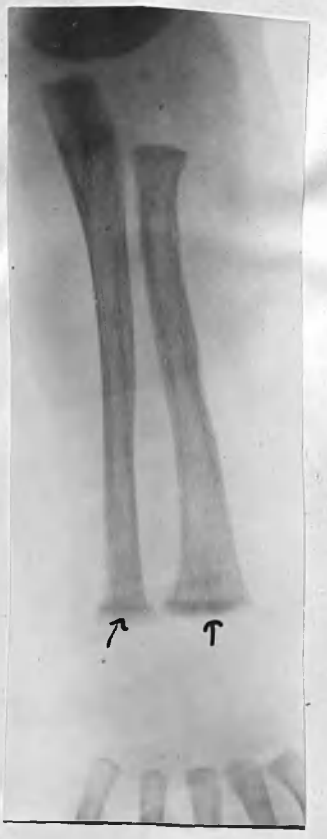
4.

M.McF. Before treatment.

Unsteady on her feet:
requires to be supported
for photograph.

V.

M.McF. after treatment.
Stands steadily on a
high stool for photograph
(time exposure, indoors).

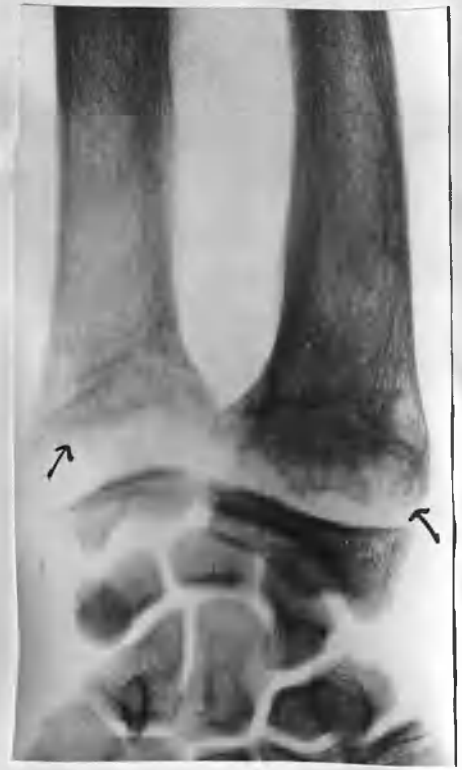


I.

No.84. M.McM. age 4 mos. after 5 exposures.
Date. 2.4.26(3 weeks after commencing treatment).
Classification. A3.
Rickets and Tetany.
Note. Fraying, spreading and some hollowing of distal metaphyses of radius and ulnar osteoporosis of shafts. A faint linear shadow is appearing in the metaphyses, the first evidence of healing.

II.

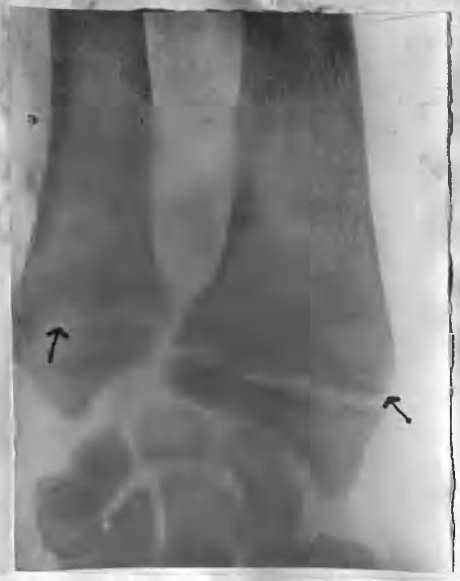
M.McM. after 20 exposures.
Date. 6.8.26.
Classification. H3.
Dense calcification at all metaphyses (the distal ulnar showing least healing). No osteoporosis.



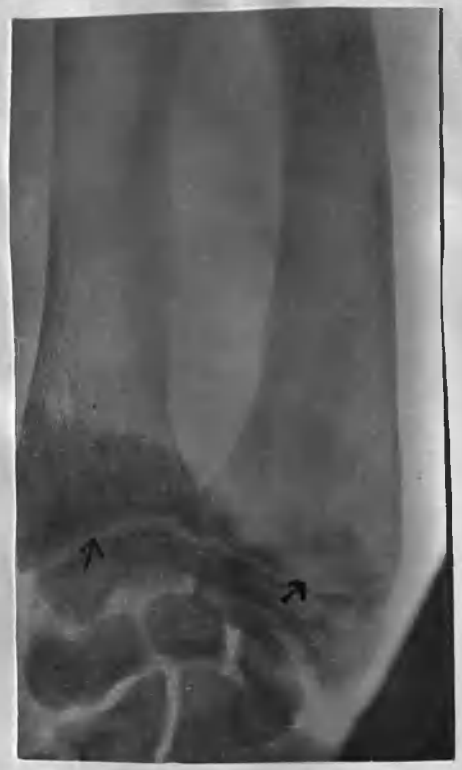
No.94.

I.

B.Q. Age 15 years. 12/6/25. Late Rickets. Shewn by X-Ray to be still active. Note irregular lime salt deposition at the growing ends of radius and ulna. The metaphysis or end of shaft of these bones is cupped and frayed shewing threads and islets of shadow caused by irregularity deposited calcium. This is typical of florid or active rickets where there has been good growth and considerable muscular movement. "The active form of florid rickets".



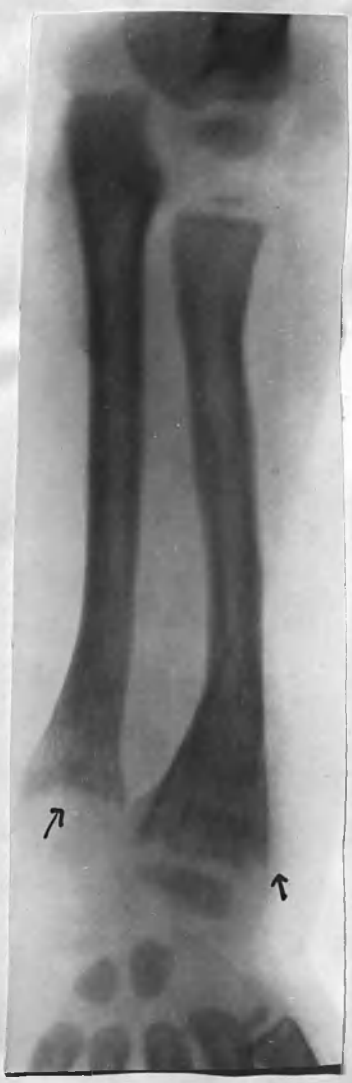
B.Q. 13.11.25. Intermediate stage shewing advanced healing after 51 exposures.

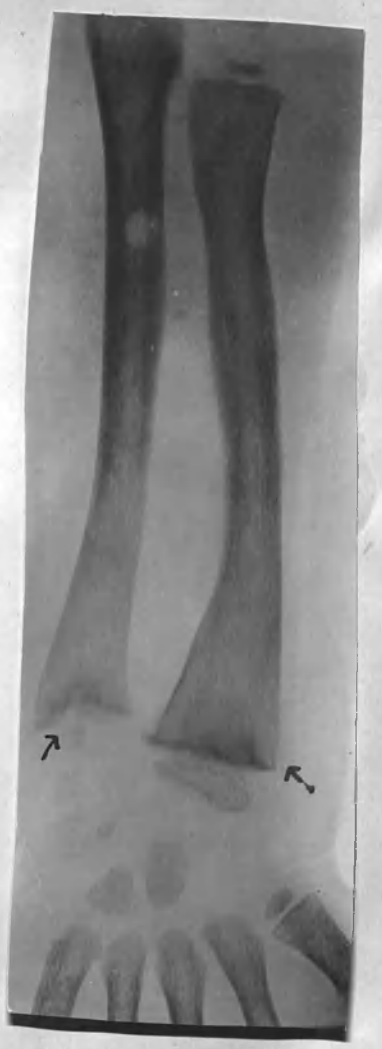


III.

B.Q. 29-1-26. Healed rickets

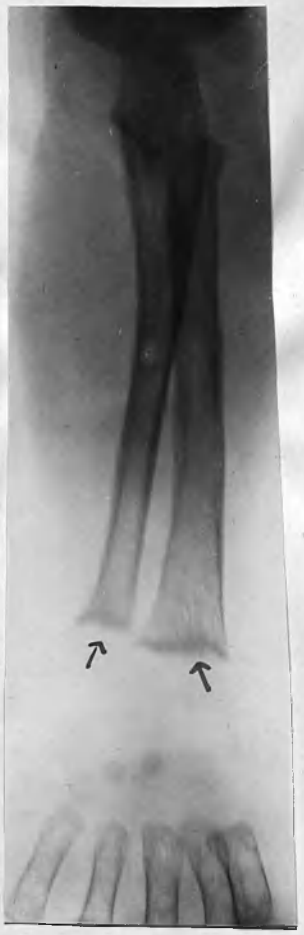
B.Q. 29-1-26. Healed rickets after 69 exposures. Note the irregular and densely calcified line at the growing ends of the bone between the Epiphysis and Metaphysis. Note also increase of calcium deposition in the sub-periosteal bone particularly in ulna.





No.102. M.R. age 4 yrs and 1 month.
Date. 30-4-26.
Classification. A3.
Note marked fraying, spreading and hollowing of metaphyses, especially distal ulnar: osteoporosis marked: curvature of both bones in lower third from greenstick fractures.

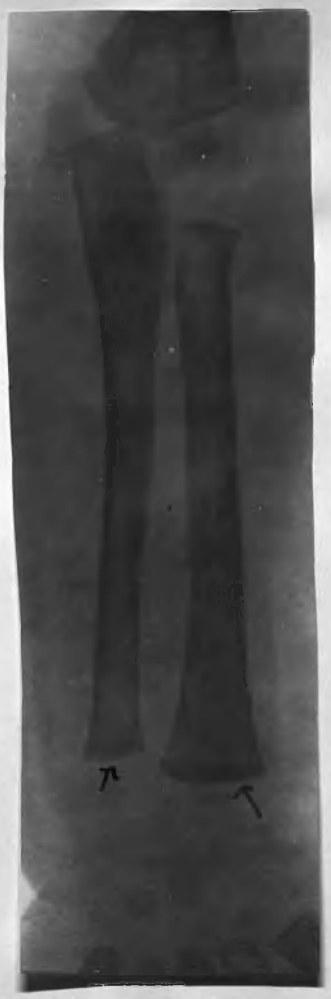
M.R. $3\frac{1}{2}$ months later, after 23 exposures.
Date. 12-8-26.
Classification. H I - 3.
There is dense shadow of healing at all metaphyseal lines, which are still somewhat irregular as healing is not yet complete. Periosteal calcification has increased and the shafts show finer meshing of bone trabeculæ.



I.

No.108. J.S. age 1 year.
Date 26.3.26.
Classification. A3.

Note fraying, some spreading and hollowing of lower metaphyses of radius and ulna: osteoporosis of shafts and poor calcification of centres of ossification.

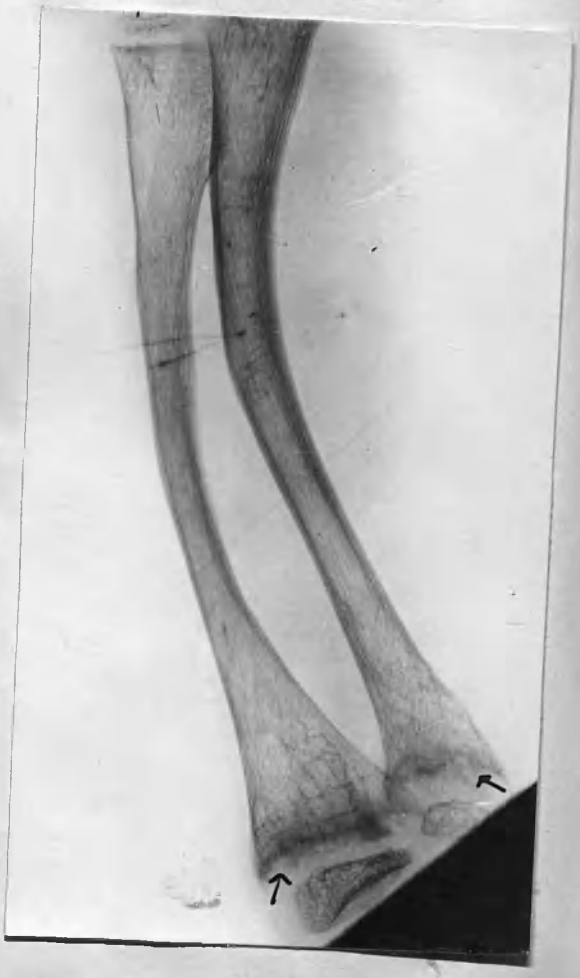


II.

J.S. after 27 exposures
Date 9.8.26.
Classification H 3

Note dense shadow of healed ricket at all metaphyses: trabeculæ of shafts normal: increased periosteal. calcification, and well-calcified centres of ossification.

THIS ONE

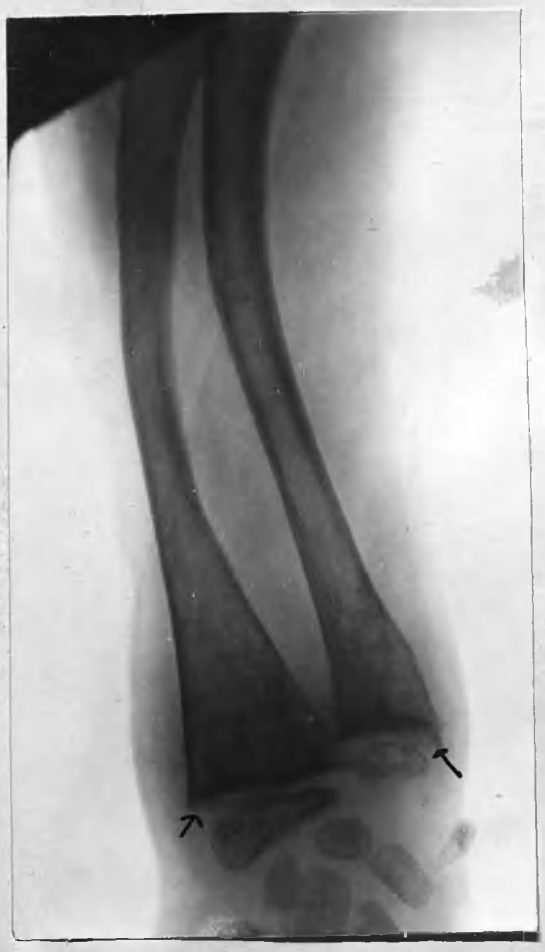


No.113. I.

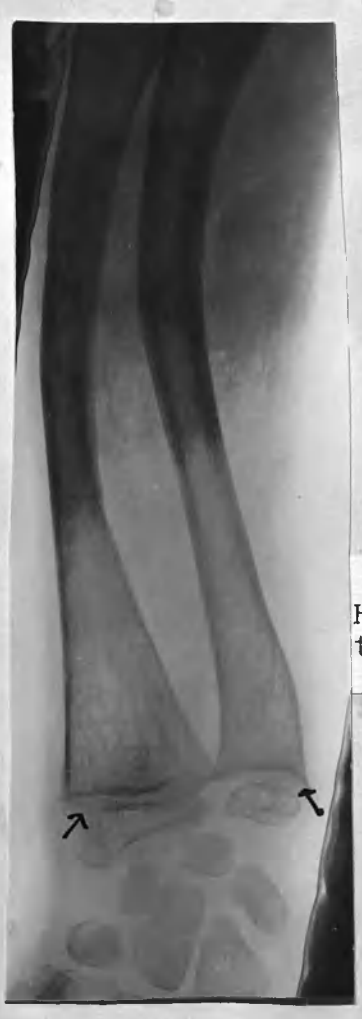
H.T. Age 8 years. Gross Rickets. Shewn by X-Ray to be still active. 2-11-25. Practically every bone in limbs and trunk twisted. This child had never been able to walk at 8 years of age, nor was she able to creep, having bent the arm bones in attempting to do so. This child was able to walk with assistance a fortnight after treatment was commenced, and in 6 weeks was able to walk without assistance.

II.

H.T. 2-11-25. Shews condition of radius and ulna before treatment. Note cupping and irregular deposition of lime salts at the growing ends of the bones. The bones generally shew deficiency of calcium (osteoporosis). There are several greenstick fractures and the sub-periosteal bone is poorly calcified.



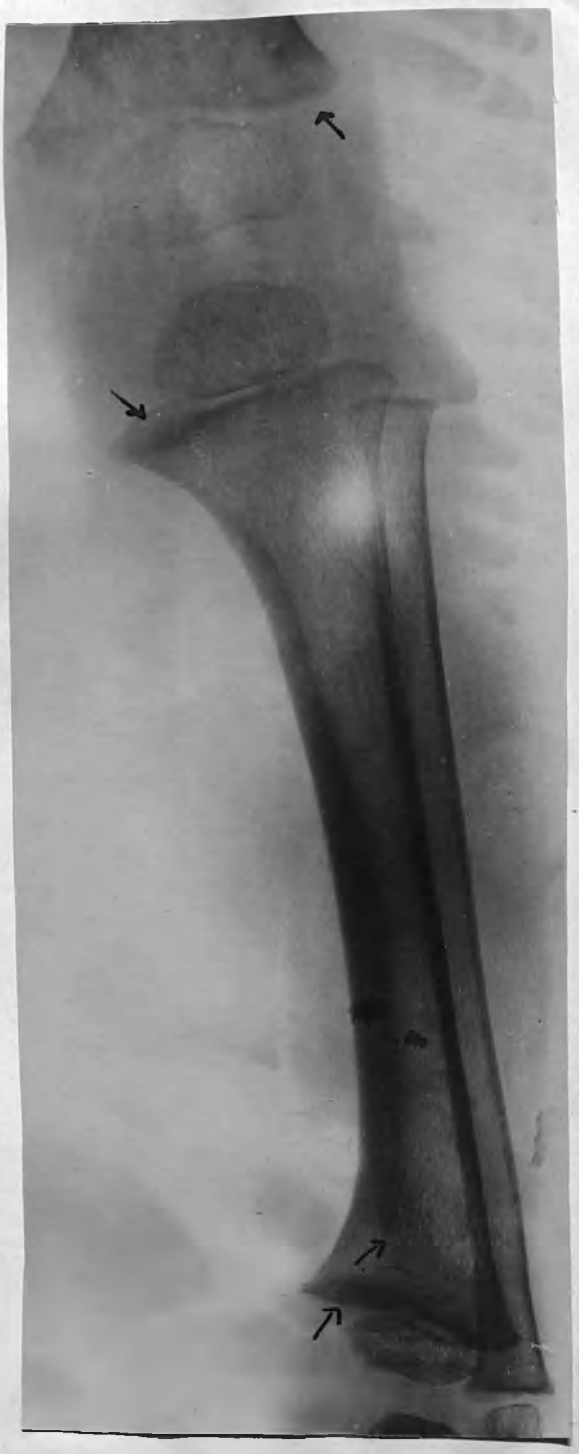
III.
12-3-26. After 41 exposures.
d Rickets. Note dense and
y regular line of calcification
e growing ends of the bones.
ther faint horizontal lines
near the end of the radius are
nce of previous parital and
cessful attempts at healing.



H.T. 8 months after
treatment commenced.

IV

H.T. 5-8-26.
Showing that process of healing
and reconstruction of the bones
has proceeded after treatment
was stopped.



No.114. 1.

I.G.T. Age 1 yrs.5 months. 1-5-25. This was a heavy fat child with bow legs and other bony signs of rickets. He was pale, he sweated about the head, had very soft muscles, was unable to walk and appetite and sleep ~~was~~ were poor. The X-Ray film shows deficiency of calcification and marked bowing of the tibia; all the metaphyseal lines are irregular, frayed out and expanded, shewing large bulbous epiphyses, Centres of ossification in the epiphysis at the knee joint are very poorly calcified.

II.

I.G.T. 18-9-25. After 40 exposures to U.V.Light and 8 ozs. Cod Oil. All metaphyses show signs of healed rickets with densely calcified metaphyseal lines. Centres of ossification in the epiphysis of the knee joint are well calcified. The bowing of the tibia is diminished owing to the increase in thickness of the periosteal bone, and there are new struts of bone forming in line of weight of the limb at the lower end of the tibia.

I.G.T. 9-4-26. Shewing the improvement in the bow legs 11 months after treatment was commenced. At this the legs are seen to be now very slightly bowed.

Evidence of improvement
to [illegible]
reader. [illegible]

III.

.T. 9-4-26. Shewing state of bones 9 months
er treatment was completed. Note that the
ving of the tibia is greatly improved by (1)
crease of thickness of periosteal new bone and
new struts of bone forming in the line of
ight at the lower end of the tibia with
orption of the projecting circumference of
metaphysis.