The Blackader Lecture

ON

SOME ASPECTS OF RICKETS*

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I AM fully conscious of the honour conferred on me in being chosen to inaugurate the Blackader Lectures before the Canadian Medical Association. I take delight at the opportunity to speak before you, but more particularly to pay this initial tribute to Doctor Blackader, that pioneer in pædiatrics in North America, distinguished physician, teacher, author and editor, that delightful host, firm friend, and ever courteous gentleman, whose generous thoughts and tireless zeal for the welfare of others and the cause which seemed to him right have called forth so splendid a testimonial of respect and devotion.

I shall not make the attempt to cover rickets, or even any main division of it, since the knowledge concerning the disease has completely outgrown the confines of a lecture. Accordingly, I shall discuss as briefly as possible four aspects of the disease which, though unrelated, have interested me personally.

Calcification Versus Bone Growth

As Pommer[1] originally pointed out, the ends of the various long bones are affected unequally in rickets, and the degree of involvement is in direct proportion to the rapidity of growth of the epiphyseal cartilage. Schmorl[2] says that rickets first develops at the costochondral junctions of the middle ribs, next at the lower

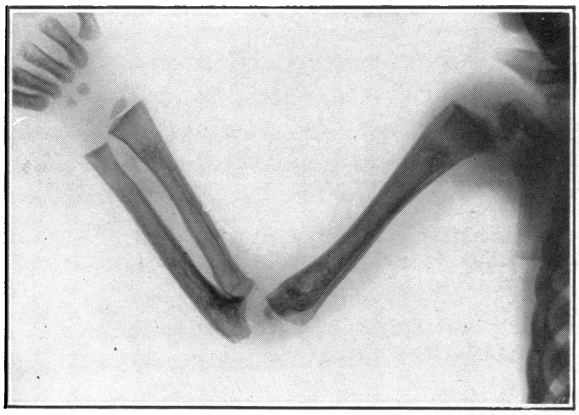

Fig. 1.—J. S. X-ray photograph of arm in a case of healed rickets. History unknown. The outlines of the shafts at the time the rickets began are clearly visible. The new bone formed under the influence of rickets has a ground-glass appearance as the result of a fine trabeculation. The demarcation between anti-rachitic and post-rachitic bone is so striking that the case affords an excellent illustration of the relative rates of growth. One can see that the magnitude of growth has been in the following order: upper end of humerus; lower end of ulna; lower end of radius; lower end of humerus; upper end of radius; upper end of ulna. The difference in the rate of growth at the lower end of ulna and at the lower end of radius is very slight. Growth at the lower end of the humerus takes place about half as fast as at the upper end and at the upper end of the radius about one-third as fast as at the lower end. Growth at the upper end of the ulna is very slow. Rickets should show itself at the ends of these bones in the order of the rates of growth.

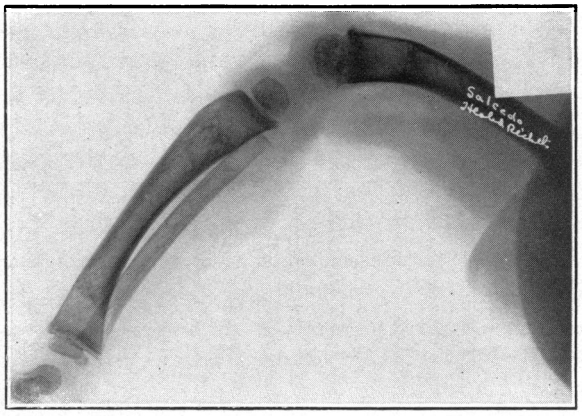

Fig. 2.—X-ray photograph of the leg of same child as in Fig. 1 (focal distance the same). One can see that the rate of growth of the lower end of the femur is greatest and the rates at the upper and lower ends of tibia and fibula are approximately the same but definitely less than that at the lower end of the femur. The bend backwards of the lower end of the femur at the junction of anti-rachitic and post-rachitic bone means that the epiphysis was dislocated backwards on the diaphysis at the time the rickets was at its height, producing an angular deformity which has mounted higher and higher in the bone with continued growth. If one will compare x-ray pictures of the arm and leg it is possible to see that the rate of growth at the lower end of the femur has been the fastest and at the ends of the tibia and fibula and the upper end of the humerus the rates of growth correspond. At the lower ends of the radius and ulna they are very slightly less.

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end of the femur, next at the upper end of the humerus and at both ends of the tibia and fibula, and next at the lower ends of the radius and ulna, the upper end of the femur, the metatarsal and metacarpal bones and the phalanges. At the more slowly growing endochondral centres he found no disturbance until the disease elsewhere had reached an advanced development. Cases in which it is possible to measure the rates of growth at the ends of the various long bones by means of the x-ray confirm Schmorl's observations (Figs. 1, 2, 3, 4). Of the bones of the extremities, the lower end of the femur exhibits the most rapid growth, the upper end of the humerus and both ends of the tibia and fibula follow next, and then the lower ends of radius and ulna. Eliot and Souther[3] found that the lower end of the ulna grows slightly faster than the lower end of the radius. The lower end of the humerus and the upper end of the radius seem to grow about one-third to one-half as fast as the opposite ends. The upper end of the ulna grows most slowly of all. An appreciation of these facts has a practical importance.

The principle just discussed applies also to the shaft in rickets, namely, that at any point the degree of involvement is proportionate to the rate of osteoblastic growth. For that reason in searching the shaft for the characteristic osteoid formations of rickets one looks especially at the surfaces of the newly forming trabeculæ near the cartilage-shaft junction or at some point in the cortex which is being reinforced with new bone or in a callus.

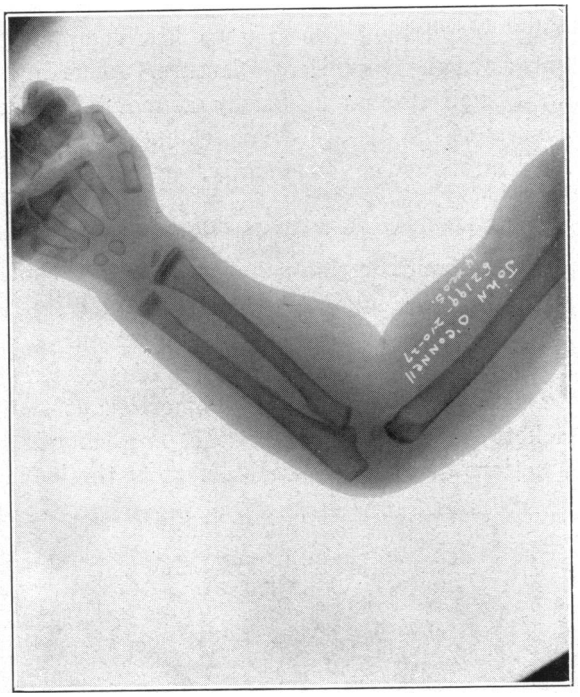

FIG. 3.—J. O., aged 14 months. X-ray photograph of arm after the administration of cod liver oil and phosphorus. As first pointed out by Phemister, inorganic phosphorus causes transverse bands to appear in the x-ray pictures of the bones. The phosphorus produces an intensely thick trabeculation which is responsible for the shadows. Only the parts of the bone in process of growth at the time of the phosphorus administration are affected. In this case the banding produced by the phosphorus gives a beautiful demonstration that the lower end of the ulna grows slightly faster than the lower end of the radius, that the lower end of the humerus and upper end of the radius grow much more slowly, and that the upper end of the ulna grows most slowly of all.

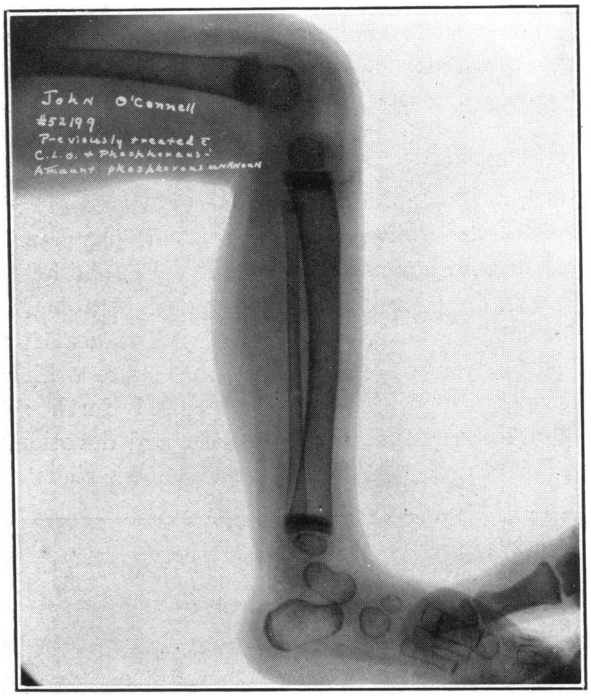

FIG. 4.—J. O. X-ray photograph of leg of same child as in Fig. 3 (focal distance the same). One can see that the rate of growth is fastest at the lower end of femur, less fast at the ends of the tibia and, by comparing leg and arm, that the growth at the ends of the tibia is slightly in excess of that at the lower ends of the radius and ulna.

The dependence of rickets on growth has been imperfectly appreciated. For rickets to develop; first, the skeleton must grow; secondly, lime salt deposition must be deficient in the new bone formed. Rickets cannot occur without growth, because lime salts cannot fail to deposit unless newly formed tissue exists for them to fail to deposit in. Rickets is present or absent according to the relationship which prevails between the rate of lime salt deposition and the rate of bone growth. If the latter

just corresponds to the former, complete calcification obtains. If the latter exceeds the former, rickets develops. If we express this relationship mathematically, as the easiest way in which to make the conception clear, then

$$\frac{\text{Rate of lime salt deposition}}{\text{Rate of bone growth}} = K_1$$

Since the rate of lime salt deposition is determined chiefly, if not entirely, by some, at present unknown, solubility product in the body fluids, which we represent by Ca x P, not because the expression is the correct one but because as the result of the teachings of Howland and Kramer[4] it is the one used practically, then

the rate of lime salt deposition (a) = Ca x P in the blood serum (b). If we now substitute (b) for (a), then

$$\frac{\text{Ca x P}}{\text{Rate of bone growth}} = K_2$$

The point is that, if the rate of bone growth (the denominator) increases, the solubility product, Ca x P (the numerator), must also increase. The product, Ca x P, therefore, cannot be thought of as having a constant value for the development or non-development of rickets, but must increase or decrease with the rate of bone growth.

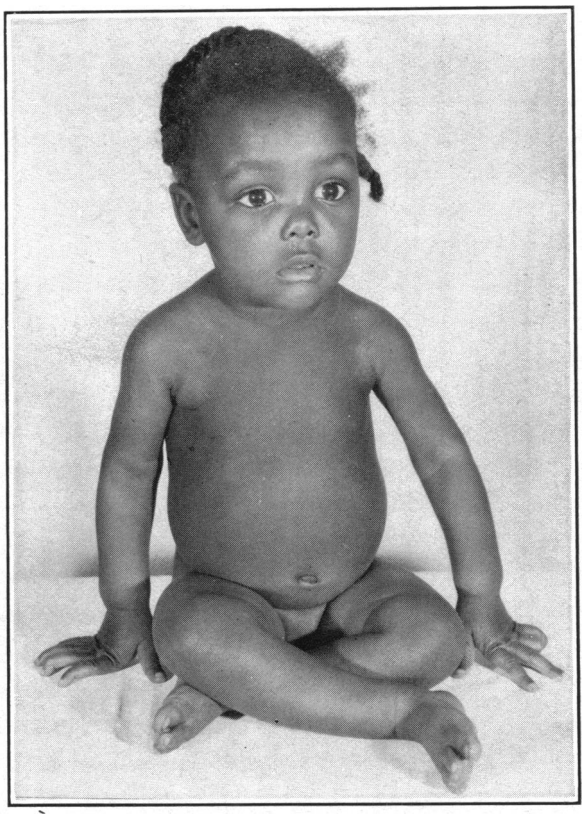

FIG. 5.—V. R., aged 16 months. Photograph illustrating the habitual sitting posture in severe rickets. The child leans slightly forward supporting the weight on outstretched fingers and palms. The legs are always crossed.

The truth of this idea is borne out by several sets of facts. The calcium remains at a more or less constant value of 10 + 1 mg. per 100 c.c. of serum throughout the life of the healthy individual, but the phosphorus varies with age. In the very young it has an average of 5 ± 0.5 mg.; in the adult, however, it is from 2 to 3.7 mg. Many adults should have rickets if the phosphorus level in the plasma or the calcium times phosphorus product were the sole determining factor. Hess[5] states that among 68 infants less than 4 months old, all of whom showed craniotabes, only one had a concentration of inorganic phosphorus less than 3.75 mg. per 100 c.c. of serum, and Wilson and Kramer,[6] in the study of 13 infants with craniotabes, varying in age from 2 to 8½ months, found that in no case was the blood phosphorus lower than 4.27 mg. and the product lower than 45. Hess reports a premature baby, 4½ months old, who developed well marked rickets when the serum calcium was 10.1 and the inorganic phosphorus 6.3 mg. (Ca x P = 61), and another premature infant, 3 months old, who showed progressive rickets in spite of the fact that the calcium of the blood was 13.6 mg. and the inorganic phosphorus 4.2 mg. (Ca x P = 57). Such data show that the concentrations of phosphorus and calcium required to prevent rickets in young babies, in particular young premature babies, may be greater than at subsequent life periods.

Proof of this conception, which seems to have entirely escaped attention, follows from the fact already stated, that in one and the same individual the degree of rickets at the cartilage-shaft junctions varies with the rate of growth. Suppose a baby has a serum calcium of 10 and a phosphorus of 3 mg. (Ca x P = 30) and the distribution of rickets is as follows:— costochondral junctions +++, lower end of femur +, lower end of humerus –, upper end of ulna –. Obviously the calcium and phosphorus concentrations in the blood have been sufficient to prevent the development of rickets at the slowly growing upper end of the ulna and the lower end of the humerus, but unable to prevent it at other fast growing cartilage-shaft junctions. The costochondral junctions, (corresponding to the premature baby) show rickets; the upper

end of the ulna (corresponding to the older child) fails to show rickets, though all are nourished by the same blood.

An appreciation that the growth factor causes the requirement of the skeleton for lime salts to vary removes the stumbling block which has annoyed students of rickets for some time. This stumbling block was introduced when Howland

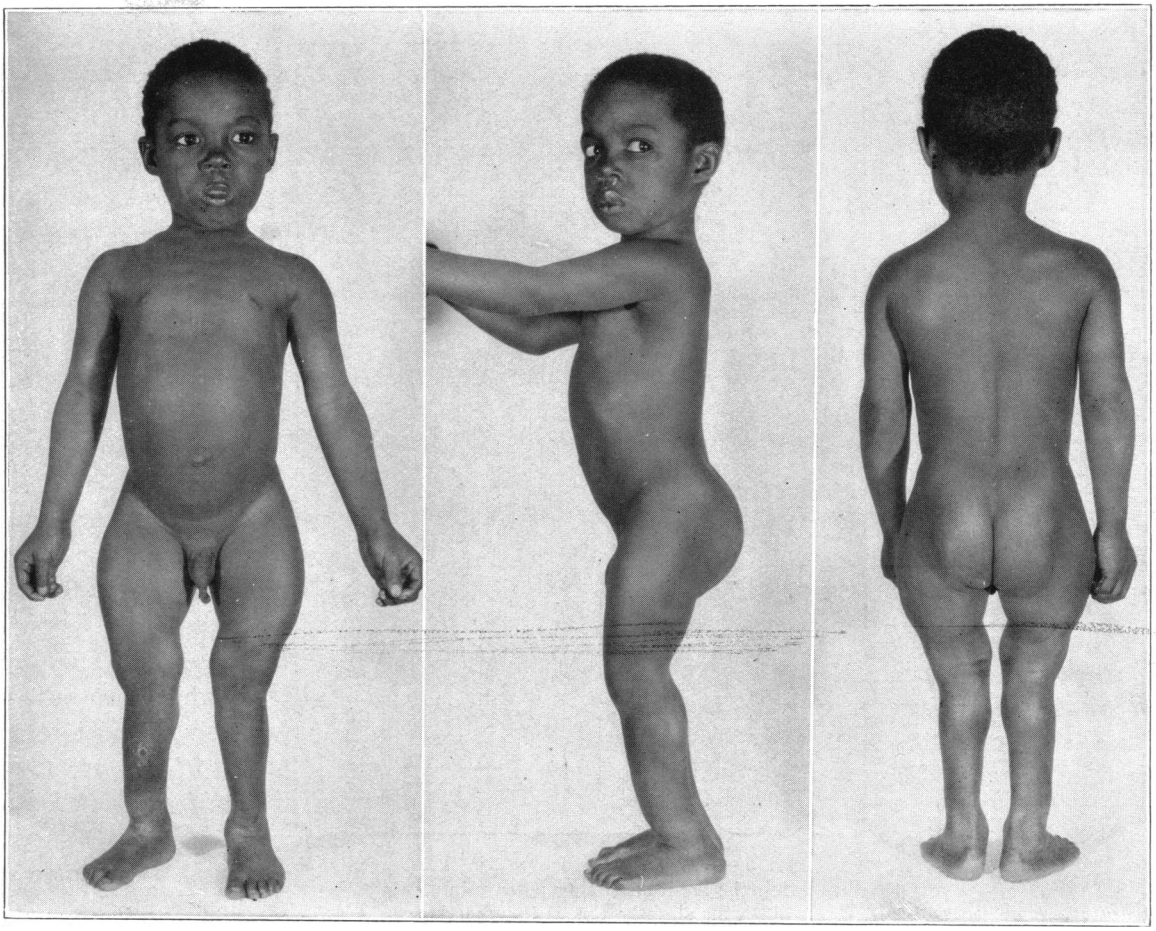

FIG. 6.—E. J., aged 5 years. Photograph illustrating the typical erect posture of child with rachitic deformities acquired from habitual sitting. Note the sabre-shin deformity just above the ankle and the bowing of the femur in the anterior and external directions. The posture is semi-crouching with buttocks protruding and compensatory lordosis. The deformities of the arms are not apparent.

and Kramer[4] advanced the idea that the Ca x P product alone was a reliable criterion for the existence or non-existence of rickets and assigned for it arbitrary limits above which, 40, the disease could not occur and below which, 30, its occurrence was inevitable.

In the criticism of the validity of the calcium x phosphorus product as a means of determining rickets I do not wish to give the impression that the formula is not useful. It is most useful, but does not have an absolute value, and must not be considered by itself but always in conjunction with the age (rate of growth), and, indeed, all the other signs of rickets. If the patient is an infant or young child, a calcium-times phosphorus product under 30 means rickets and usually severe rickets. If the patient is an adult, the calcium x phosphorus product under 30 does not have special significance. If the patient is a prematurely born infant a calcium x phosphorus product of more than 40 does not preclude the possibility of rickets.

In our turn we must give warning not to take the equation

$$\frac{\text{Ca x P}}{\text{Rate of bone growth}} = K,$$

with too great literalness. The numerator, Ca x P, is used merely to symbolize the, at present unknown, equilibrium product in the

blood serum which determines lime salt deposition in bone and cartilage. Moreover, the equation leaves out of consideration the cartilage and bone cell which may be factors in the phenomenon of lime salt precipitation. Finally, the "rate of bone growth" is intangible and not susceptible of measurement. There is not the slightest doubt, however, that the equation expresses the truth, in indicating that the Ca x P product or the true solubility

THE ENDS OF THE LONG BONES LISTED ACCORDING TO SPEED OF GROWTH AND SUSCEPTIBILITY TO RICKETS

Middle ribs, costochondral junctions	++++++
Femur, lower end	++++++
Tibia, both ends	++++
Fibula, both ends	++++
Humerus, upper end	++++
Femur, upper end	++++
Ulna, lower end	+++
Radius, lower end	+++
Humerus, lower end	++
Radius upper end	++
Ulna, upper end	+

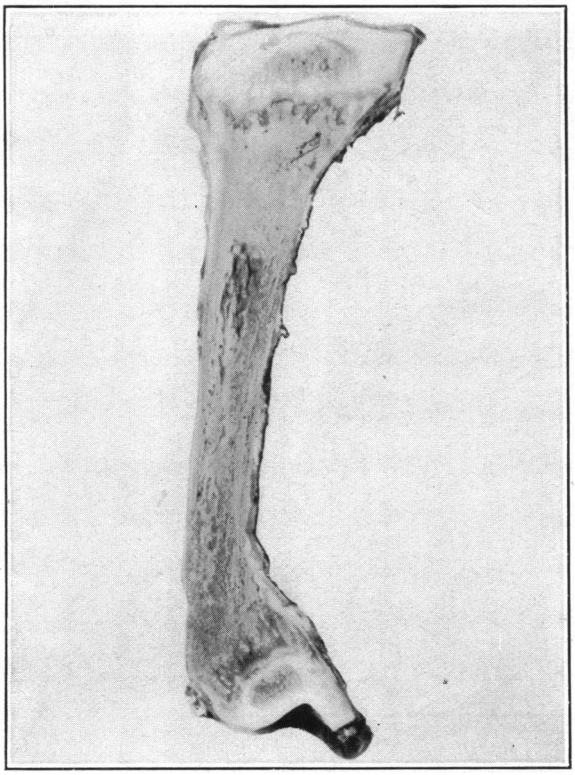

FIG. 7.—Photograph of the cut surface of the tibia split longitudinally in a case of severe rickets, showing the dislocation backwards of the lower epiphysis on diaphysis. The dislocation is made possible as the result of the stretching of the soft rachitic intermediate zone. As growth proceeds, the new bone which is generated by the lower epiphysis will take the direction of the latter with the result that the angular deformity will rise higher and higher in the leg. The photograph illustrates the pathological condition responsible for the sabre-shin deformity in its incipient stage.

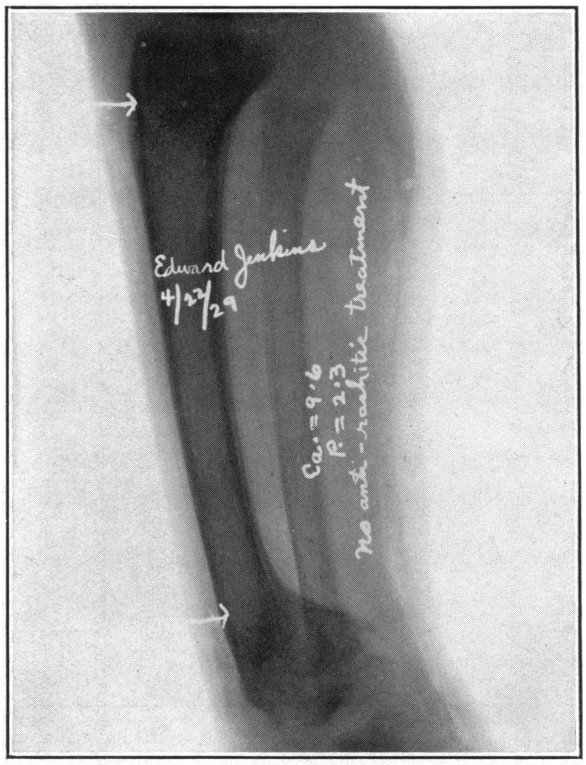

FIG. 8.—E. J., aged 3 years. The same child as in Fig. 6. X-ray photograph of leg showing origin of the sabre-shin deformity. The lower epiphysis of the tibia has been displaced backwards on the diaphysis. The deformity is situated just above the ankle.

product required for calcification of the skeleton is not fixed, as might be expected to be the case if the body were a collection of dead substances, but varies with the demands for growth. The equation is important from another point of view because it illustrates how the etiology of rickets has two sides, so to speak, and some factors operate by affecting calcification and others by influencing growth.

DEGREES OF RICKETS

We must grasp another fact in order to understand rickets, namely, that the disease manifests itself with every gradation, from one in which no lime salts deposit in the newly formed cartilage and bone to one in which only a small fraction is lacking. Rickets often goes on for months or years partially developed, for example, with 20 per cent of the optimal lime salt deposition occurring and 80 per cent not occurring, or with 60 per cent taking place and 40 per cent not taking place. When we discover by the x-ray film fragmentary lime salt deposits in the proliferative part of the epi-

physeal cartilage we often think that recovery is in progress, when really the condition has never changed and the lime salt deposits have been forming all along but have never been complete. If enlargements of the cartilage-shaft junctions persist in spite of treatment, how common it is to conclude that the treatment has been a total failure, whereas in reality it has changed a condition from one in which little lime salt was being deposited to one in which perhaps 70 per cent of that required was being attained. Probably a totally rachitic state, that is one in which no lime salts deposit anywhere does not ever occur. In all cases of rickets one ought to think of lime salt deposition, not as being absent but as being disturbed, in some cases slightly, in others severely. Probably in most cases of rickets the lime salt deposition fluctuates from time to time, depending on variations in the diet, chance exposure to ultra-violet light, the presence of complicating disease, etc. If the fluctuations are marked enough to express themselves in lines of lime salt deposit in the bone, we speak of "intermittent" or relapsing rickets.

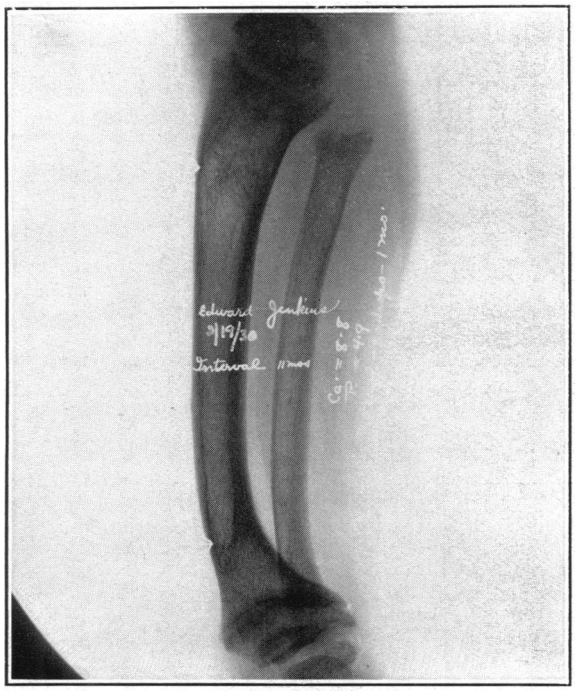

FIG. 9.—E. J. X-ray photograph of leg of same child as in Fig. 8 after an interval of 11 months. Note that this deformity is at higher level as the result of growth at the lower epiphysis. The deformity has really been pushed upwards and is now definitely above the ankle. The lower termination of the shaft of the tibia at the time when the rickets began is clearly outlined.

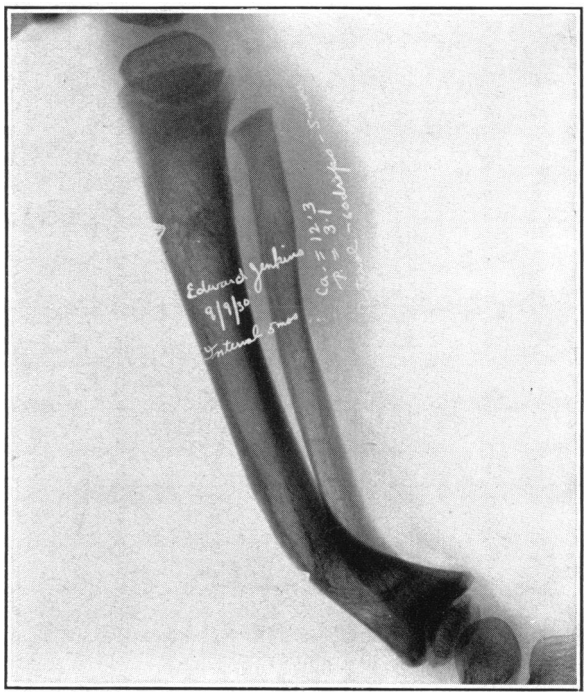

FIG. 10.—E. J. X-ray photograph of leg of same child as in Fig. 8 five months later than in Fig. 9. The sabre-shin deformity has mounted still higher as the result of continued growth at the lower epiphysis. The bending has increased. The lower end of the tibia at the time the rickets began is clearly outlined but now ends in a sharp point. Characteristic curved lines of stress extending downwards and backwards have made their appearance in the tibia below the deformity.

SITTING DEFORMITIES WITH PARTICULAR REFERENCE TO THE ORIGIN OF THE SABRE-SHIN

In extensive rickets every bone subjected to strain is affected to some extent and in the case of some bones, notably the long bones of the extremities and ribs, cranial vault and pelvis, the changes may be extreme. I wish to point out that the deformities caused by rickets follow different patterns at different age periods depending entirely on the time in the child's life when the rickets develops and flourishes. The reasons are, first, that stress and strain are differently applied, as the posture and activity change with increasing age, and, secondly, that rickets affects different parts of the skeleton at different ages according to the changing rates of growth. During the first six months the head is especially affected. In the next two and one-half years the cartilage-shaft junctions and the shafts vie in showing the greatest deformity. During the

third and fourth years the cartilage-shaft junctions and the shafts are equally involved, but after the fourth year the deformities of the cartilage-shaft junctions become less and less and those of the shaft more and more conspicuous. Indeed, from the fifth year on rickets bears an increasingly close resemblance to osteomalacia. According to Schmidt[7] the last cartilage-shaft junctions to show deformity are the costochondral junctions, because growth at them continues when it has virtually ended at the others.

I shall describe briefly the chief deformities of the arms and legs which are produced by sitting, and in a little more detail the origin of the sabre-shin deformity. The infant with severe rickets often sits all day long for months with legs crossed, tailor-fashion, leaning slightly forward and supporting the body on the outstretched hands (Fig. 5). As the result of this unvarying posture both arms and legs become deformed. The humerus bows outwards or outwards and forwards, slightly above the insertion of the deltoid; the curvatures, normal to the radius and ulna, are exaggerated and the lower epiphyses of the two bones are bent forwards and at the same time deflected either internally or externally according as the hands "toe in" or "toe out". The deformities of the arms are interesting and characteristic but do not have great practical importance. I have seen the bends of the radius and ulna disappear entirely within a year after the arms have been released from weight bearing. The bow in the humerus may be permanent, but is never conspicuous and does not interfere with the usefulness of the arm. The deformities of the leg, on the other hand, are most important. Though they may disappear, they often remain throughout life, are most disfiguring, and require orthopædic intervention. If one examines the legs in a typical case, one finds that the lower end of the femur, the lower end of the tibia, and, sometimes also, the upper end of the tibia are bent backwards or, perhaps, more often, backwards and inwards, and if one straightens out the legs and then returns them to their original position, one finds that the legs exactly fit into each other. When the child with typical sitting rachitic deformities finally stands he is forced to assume a characteristic semi-crouching position, with the buttocks thrust backwards and extreme lumbar lordosis (Fig. 6). If the rickets comes on after the child has learned to stand, the bends in the legs always occur sideways. The presence of the curvatures in the antero-posterior plane dates the rickets back to infancy.

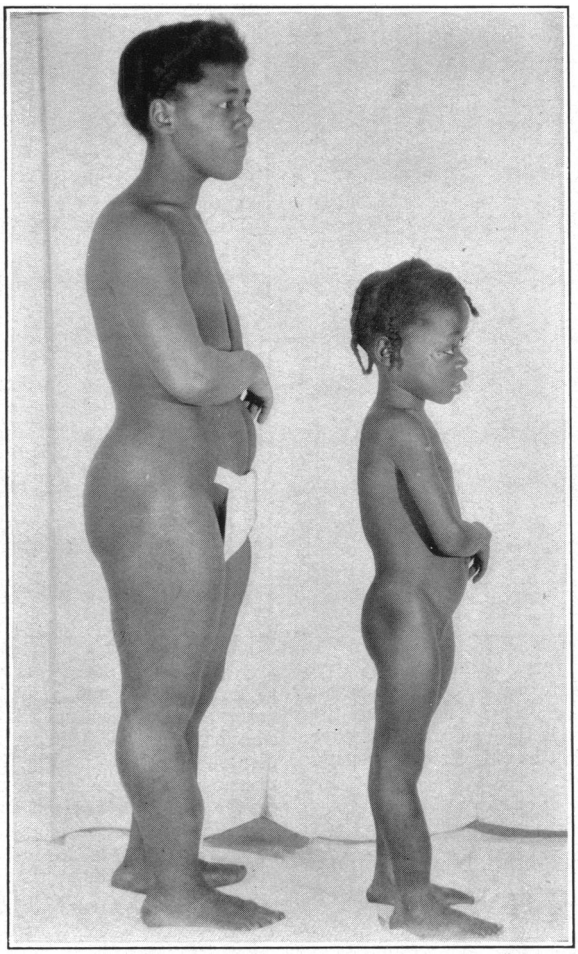

FIG. 11.—Typical sabre-shin deformity in mother and daughter. The daughter is seven years old. Note that the sabre-shin is higher in the case of the mother than the daughter. In both the anterior bowing of the femur can be recognized by the shape of the thigh. One knows from the characteristics of the deformities that the rickets was acquired in infancy from sitting.

The deformity of the tibia is the celebrated sabre-shin. Though the sabre-shin is usually a sitting deformity it can probably also be produced as the result of continued lying on the back with feet relaxed. The exact origin of the sabre-shin is interesting. It is produced by the pull of the tendo Achillis which carries the foot backwards. The displacement of the foot is made possible because the soft tissues composing the rachitic intermediate zone between the lower epiphysis and diaphysis of the tibia stretch and allow the epiphysis to bend backwards, so as to form an obtuse angle with the

shaft (Figs. 7, 8, 9, 10). Since the growth at the end of a long bone takes place entirely from the proliferative zone of the epiphyseal cartilage, the new bone generated at the lower end of the tibia comes entirely from the epiphysis and, therefore, takes the direction of the latter and perpetuates the obtuse angle. As growth continues, that is, as the epiphysis yields more and more shaft, the bend rises higher and higher until it reaches a point at or just above the lower third of the tibia. The reason that the bend in the sabre-shin never lies above the middle of the leg is that it begins always at the lower epiphyseo-diaphyseal junction. The sabre-shin deformity is a most common sight in the negro children playing on the Baltimore streets. In those who are very young the bend is just above the ankle, in those a year or two older it can be seen just above the shoetop, and in adults at or just above the junction of the middle and lower thirds of the leg (Fig. 11). Of course if the rickets continues after the child with these sitting deformities stands and walks, the bends characteristic of the rickets of later periods may be superimposed and the sitting deformities modified. For example, the characteristic anterior curvatures may be found in company with extreme knock-knees or bow-legs and it is often possible to recognize from the nature of the deformities that the rickets has continued to affect the rigidity of the skeleton after the sitting period is over. As time goes on, the sabre-tibia thins from side to side and thickens from before backwards. The thickening is due to new bone formation on the concave side. But the result is to intensify the sabre characteristics.

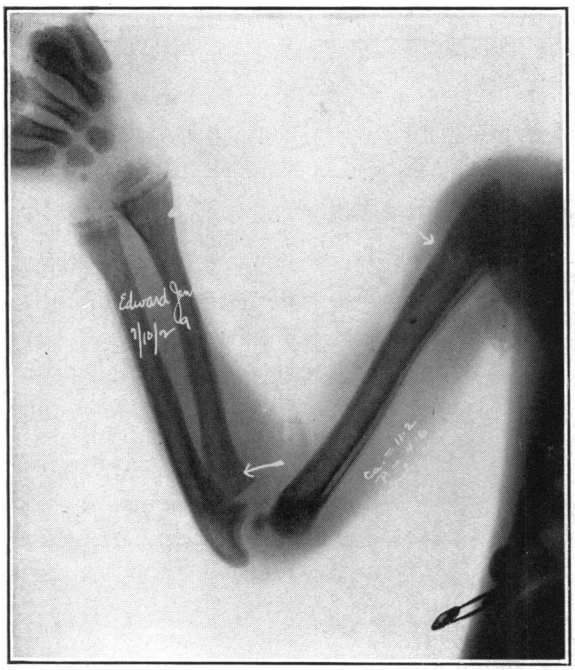

FIG. 12.—E. J., aged 3 years 4 months. X-ray photograph of arm showing deformity of the humerus acquired from sitting in severe rickets. The part of the humerus indicated by the arrow is the one at which later the bowing developed. Note that at this time no bowing exists and no fracture is present.

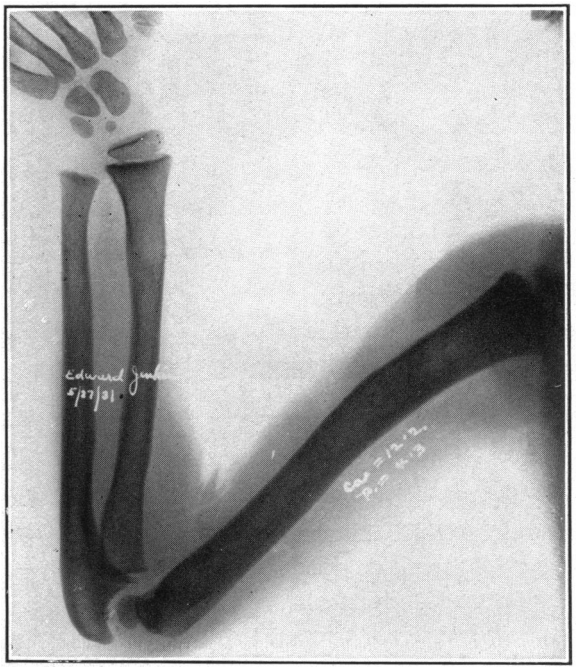

FIG. 13.—Photograph of the arm of the same child as in Fig. 12, one year and ten months later. The anterior (and external) bowing of humerus is evident. The bowing has occurred in the part of the bone indicated by arrow in Fig. 9, as has been determined by measurement. Inasmuch as growth at the upper end of the humerus occurs more rapidly than at the lower end, the point at which the bowing took place has been carried downwards, so that it now occupies a much lower position in the bone than when it first began. This deformity of the humerus has a totally different origin from that of the sabre-shin. As pointed out it results from yielding of the substance of the shaft and the point of yielding is probably determined by the pull of the deltoid, to the insertion of which it has a more or less constant relationship.

Shifting inwards on the diaphysis of the upper epiphysis of the humerus is very common also in rickets and the outward bend of the upper end of the humerus may be the product of the shift of the epiphysis and the yielding of the shaft above the deltoid.

The curvatures of the long bones in rickets have different origins. Many of them, like the sabre-shin and the backward bends at the lower end of the femur and upper end of the tibia, have their origin in displacements of the epiphysis on the diaphysis, but others are the result of simple bends in the substance of the

shaft or of fractures. The bowing of the humerus mentioned is an example of simple bending of the shaft (Figs. 12 and 13). If one takes x-ray pictures at frequent intervals of the humerus of the sitting rachitic child, one finds that the bend begins gradually a short distance above the insertion of the deltoid, probably as the result of the traction of that muscle, and progressively increases. Since the growth of the humerus at the upper epiphysis occurs twice as rapidly as at the lower, the bend gradually descends, but always remains the same distance above the insertion of the deltoid. Shifting inwards on the diaphysis of the upper epiphysis of the humerus is very common also in rickets and the outward bend of the upper end of the humerus may be the product of the shift of the epiphysis and the yielding of the shaft above the deltoid.

LIGHT IN RELATIONSHIP TO RICKETS*

The anti-rachitic radiations in sunlight and artificial light.—Sunlight consists of visible rays varying in length from 380 to 760 mμ (39 per cent); invisible infra-red rays from 760 to 50,000 mμ (60 per cent), those longer than 4,000 mμ amounting to only 1 per cent of the total; and invisible ultra-violet rays, from 380 to 290 mμ (1 per cent). The distribution of the energy in the solar spectrum is continuous.

The quartz mercury-vapour lamp gives off a percentage of infra-red (52 per cent) comparable to that in solar radiation, a much smaller percentage of visible light (20 to 33 per cent), and a wealth of ultra-violet light of wave lengths ranging between 310 and 255 mμ. Twenty-eight per cent of the total energy lies in the ultra-violet, and of this 6 per cent in wave lengths shorter than 290 mμ and hence beyond the limits of the solar spectrum. The spectrum of the quartz mercury-vapour lamp for practical purposes is discontinuous. Strong lines lie at 255, 297, 302 and 313 mμ, and weaker lines at 265, 270 and 310 mμ.

The carbon arc lamps give different kinds of emissions, according as certain metals, *e.g.*, cerium, fluorine, iron, nickel, cobalt, strontium, are incorporated in the carbons, either singly or in combinations. If iron is used, the radiations become strong in the ultra-violet between 230 and 320 mμ and, if strontium, in the near infra-red. The arc lamps whose carbons are combined with the idea of furnishing light of percentage composition similar to solar radiation are fairly successful in that regard. The wave lengths between 310 and 290 mμ are somewhat better represented than in sunlight, and those shorter than 290 do not possess much energy.

It is by means of the artificial sources of light used in combination with various filters that the rays active in rickets have been isolated. Hess and Anderson[8] found that wave lengths of 310 and 313 mμ are slightly effective, and Sonne and Rekling[9] that wave lengths of about 300 are very effective, those around 280 the most effective, and those around 260 mμ valueless. Window glass, which removes ultra-violet rays shorter than 315 mμ, deprives light of all its anti-rachitic power. The ultra-violet radiations active in rickets, then, lie between 315 and 260 mμ, corresponding in their range to the absorption spectrum of ergosterol.

The seasonal variation in ultra-violet light and in rickets. — The solar radiations shorter than 200 mμ convert the oxygen of the air into ozone, while those between 200 and 300 mμ change the ozone back again into oxygen. Through the action of the short ultra-violet radiations of the sun, a blanket of ozone, in continual process of formation and dissolution, is maintained in the outer layers of the atmosphere (Dobson,[10] Buisson[11]). This blanket acts as a great screen which is *totally* impermeable to radiations shorter than 290 mμ and through its exclusion of them makes life on the earth possible. To the anti-rachitic radiations between 290 and 315 mμ the blanket is only *partially* permeable, removing, as one might expect, the shorter more effectively than the longer. The movement of the earth on an oblique axis around the sun causes the sun's rays to impinge on the earth's atmosphere at different angles at different times of the year, and hence compels the rays to traverse a greater thickness of atmosphere at some periods than at others. The interposition of the atmosphere between the earth and the sun, and the movements of the earth around the sun, therefore, combine to

* In the lecture as delivered in Vancouver one of the main subjects was "The Age Incidence of Rickets". The presentation of the subject rested largely on the data which Eliot and Souther had collected in the course of the rickets investigation conducted by the United States Children's Bureau in New Haven, Connecticut, and Porto Rico. Inasmuch as Eliot and Souther have not yet published the results of their work, "Light in Relationship to Rickets" has been substituted.

cause a seasonal variation in both the quantity and quality of the anti-rachitic radiations which reach the earth's surface. For example, if the altitude of the sun is high, so that these anti-rachitic short radiations which can be partially absorbed strike the earth nearly perpendicularly, they encounter a minimal thickness of atmosphere, and the loss which they sustain is minimal. At the same time their density per unit area is maximal. On the other hand, if the altitude of the sun is low, so that the short radiations strike the earth obliquely, they traverse a greatly increased thickness of atmosphere and suffer a correspondingly great loss, and at the same time, as the result of their obliquity, their density per unit area is diminished. At Marseilles at midday, under the most favourable conditions of midsummer, Fabry and Buisson[12] found the shortest radiations to be 290 mμ; in winter at Davos, Switzerland, Dorno[13] determined the shortest rays to be 306 mμ. The energy of the short rays at the extreme end of the spectrum, whether in winter or in summer, is very small. According to Fabry and Buisson, the intensity of the summer radiation at 290 mμ is reduced from its passage through the atmosphere 2,000,000 times, and possesses only one-millionth the strength of the radiation at 315 mμ. According to Clark's[14] measurements at Baltimore, the total energy of the solar radiations between 290 and 315 mμ is 12 to 14 times greater in summer than in winter. One must think, then, of the solar spectrum outside the equatorial zone as undergoing a periodic expansion and retraction at the ultra-violet end, the expansion reaching its maximum at the summer solstice and the retraction at the winter solstice, or, to put the matter another way, of a winter period of famine in the anti-rachitic ultra-violet alternating with a summer period of plenty. The higher the latitude, the longer is this famine period, until at the polar regions it is complete for the greater part of the year. Conversely, the lower the latitude, the shorter the famine period, until as the equatorial zone is approached it ceases altogether, since the lowest altitude of the sun has become sufficient to ensure throughout the year adequate anti-rachitic radiation. By actual measurement Clark found that for Baltimore the anti-rachitic ultra-violet in the sunlight was least in December, began rapidly to increase in February, reached its height in late July or early August, and then rapidly declined to its lowest point in December. The quantity of anti-rachitic radiations in March and October corresponded. The reason that the peak in the anti-rachitic ultra-violet radiation occurred in late July and early August, instead of on June 21st, is that the blanket of ozone in the outer atmosphere remained unchanged until that time.

Tisdall and Brown,[15] at Toronto, have approached the problem experimentally by exposing rachitic rats to sunlight at different times of the year. They discovered that during December, January and February sunlight had a slight but definite anti-rachitic action; a sharp increase in activity occurred about February 15th, which reached the maximum in mid-summer, and an equally sharp decrease began about October 15th. The anti-rachitic power of sunlight in midsummer was eight times greater than in mid-winter. For the latitude and altitude of Toronto, the crucial inclination of the sun, so far as the anti-rachitic property of its light is concerned, seemed to be 35 degrees. Above this angle a pronounced anti-rachitic effect was produced, but below, only a very slight one.

All students of rickets are aware that the disease manifests itself with great frequency during the winter and early spring, and with much less frequency during the summer. This seasonal variation in rickets, which was first described by Kassowitz,[16] in 1884, has its explanation, as already indicated, in the seasonal variation in the ultra-violet radiations of the sun, which during the winter famine period in the northern part of the temperate zone are at best barely sufficient to afford protection, even if fully utilized under the best atmospheric conditions. Since it usually takes about two to three months for rickets to show itself clinically, the disease is found with the greatest frequency wherever the famine in ultra-violet light exists, in late February, March and April. The length of the rickets season, then, and the severity of the disease vary undoubtedly with the latitude, subject to modification by other factors which cannot be discussed here. According to Tisdall,[17] in Glasgow the sun is below 35 degrees for six months of the year,

in London five months, in Toronto and Boston four months, in Baltimore three months. The seasons for the development of rickets in the centres mentioned should be six, five, four and three months, respectively. In New Orleans at midday the sun is above 35 degrees throughout the year.

The diurnal variations in the anti-rachitic ultra-violet radiations show their effect only through summation in the seasonal. During the early morning and late afternoon, when the altitude of the sun is low, sunlight may not have any anti-rachitic activity. Naturally, the period of the day, when sunlight is effective is longer during summer than winter. For example, at Toronto, in June, the sun exerted an anti-rachitic effect from 7.55 a.m. to 4.30 p.m., but on March 6 this marked anti-rachitic action was present only from 10.37 a.m. to 2.18 p.m. (Tisdall and Brown,[18] Tisdall[19]). Clark[14] found that in Baltimore in winter there were no anti-rachitic rays in direct sunshine after 3.00 p.m.

Reflected sunshine and direct sunshine.—It is most important to realize that on meeting the atmosphere the solar radiations active in rickets are in part reflected and scattered, so that at the surface of the earth they are of two varieties, those coming indirectly from the sky, "sky shine" and those coming directly from the sun, "direct shine". An object placed on the shady side of the street is bathed only in sky shine. If placed on the sunny side under a long cylinder pointed at the sun, the sky shine is cut off and only direct rays reach it. Usually, one receives either sky shine and direct shine together, as when sitting in the sun, or sky shine alone, as when in the shade, but, exceptionally, one may receive only the direct rays, as when in a room in the sunlight at a distance from the open window. To receive all the sky shine, no obstruction can exist between the recipient and any part of the hemispherical dome of the sky; *e.g.*, a child seated in the shade against a wall receives only 50 per cent of the sky shine and, if at the back of a room, practically none. The walls of buildings in cities greatly reduce sky shine as well as direct sunshine.

Unfortunately, knowledge concerning sky shine is not so exact as one might wish. At Baltimore, in midsummer at noon on clear days, Clark[14] found that sky shine between 290 and 315 mμ possessed about two-thirds the value of the corresponding direct ultra-violet radiations; in early spring and fall it had about an equal value and in winter a greater value. The ultra-violet radiation in sky shine is far more constant than that from the direct sun shine. Since the reflected ultra-violet rays come from the drops of water in the atmosphere, their intensity may be increased by light clouds or haze. On a cloudy day, therefore, the total ultra-violet intensity of sky shine may be far greater than that of direct shine. On the other hand, if the clouds are dense the ultra-violet rays from sky shine, also, may be greatly reduced. Tisdall and Brown[20] estimated that the curative power of noon sky shine from January to May was two-thirds that of direct sunlight plus sky shine. When the altitude of the sun is high, the ultra-violet radiation of sky shine is greatest, and hence the value of sky shine is greatest in summer and at noon. But, because sky shine is reflected light, its ultra-violet rays are transmitted to the earth when the altitude of the sun is too low for the direct radiations to penetrate effectively. Hence, in the early morning and late afternoon the ultra-violet in sky shine may be greatly in excess of direct shine, as is the case in December. Sky shine is said not to tan but undoubtedly *can* produce tanning. It is the chief source of protection against rickets in parts of the world in which, relatively speaking, little sunlight exists, for example, England.

Ultra-violet radiations, both direct and indirect, may be reflected from favourable surfaces on the earth. For example, Earp[21] found that the ultra-violet light was greater in Denver in November, when the earth was covered with snow, than in September. Doubtless, in the polar regions the anti-rachitic ultra-violet radiation during the summer season is exceedingly weak, but it is present the entire twenty-four hours and its effectiveness is increased because of the continual reflection from the snow. The therapeutic ultra-violet radiations are undoubtedly reflected from the surfaces of water and sand of the seashore and desert regions.

Altitude.—Not only latitude but altitude is important with reference to the transmissibility of the anti-rachitic ultra-violet. The higher the altitude the thinner the covering of atmosphere which can absorb the short radiations. To show how altitude and latitude are interrelated, Tisdall and Brown's[22] comparison of

the anti-rachitic conditions at Denver and Toronto may be cited. These investigators computed that on account of the more southern latitude of Denver the sun should attain an angle of 35 degrees by February 8th instead of March 15th, as at Toronto, but because of the altitude of 5,300 feet of Denver the angle of inclination of the sun required for a pronounced anti-rachitic effect was reduced from 35 degrees to 29 degrees. At Denver, then, the theoretical rickets period begins on December 1st and ends on January 17th, whereas, in Toronto it begins on October 15th and ends on February 15th.

Dust and smoke.—Foreign particles in the air, such as dust and smoke, act as absorbents of ultra-violet light. The extent to which they obstruct the anti-rachitic radiations is not known, but is doubtless considerable.

Clothing.—Little of the anti-rachitic radiation is able to penetrate clothing material as was indicated by the study of Sambon[23] in 1907. According to Hess, the cotton and woollen stocking material worn by infants admits but a small fraction of ultra-violet light unless a very large dosage is used. Rayon material may permit the passage of anti-rachitic radiations.

Intensity versus time.—The therapeutic effect of ultra-violet light in rickets depends on an intensity factor and a time factor, though the product is not a constant. Under poorer conditions a long exposure may be the equivalent of a short one under good conditions. For example, Clark[14] has estimated that in Toronto a daily exposure of approximately twenty minutes to the noon sun in June is equivalent to three or four hours in January.

The area of skin exposed and pigmentation.—A surface area factor, doubtless, ought also to be included in considering the therapeutic effect of ultra-violet light in rickets. Huldschinsky[24] showed that exposure of an arm alone in rickets is sufficient to cause a general deposition of lime salts. Undoubtedly, the exposure of the entire body or a large part to ultra-violet radiation is more effective than the exposure of a small part. Nevertheless, the habitual exposure of a small part, for example, the face and hands, is the way in which protection is ordinarily attained.

The pigmentation of the skin which results from exposure to ultra-violet light is, according to Miescher,[25] nature's method of protecting the dermis. If all pigment is lacking, irradiation, if sufficiently intense and prolonged, will cause ulceration of the deeper layers. Miescher's studies indicate that the horny layer of the epidermis thickens under ultra-violet irradiation and that it possesses the property of absorbing ultra-violet rays. As Miescher puts it:—"The pigment layer is the sunshade for the dermis, as the horny layer is the sunshade for the epidermis. Apparently, the horny layer of the epidermis is a first line of defense against ultra-violet light, as against other injurious agents, and the pigment is a second. The mechanism which brings about tanning is unknown. Tanning is not essential in order that ultra-violet light have an anti-rachitic effect.

Radiation of the skin of the negro is curative of rickets in spite of the heavy pigmentation, a fact which indicates that the seat of action of ultra-violet light must be very superficial. According to Anderson and Macht,[26] ultra-violet light can penetrate the living skin of the rabbit a distance of 1 to 2 mm.

Window glass and its substitutes.—We have already mentioned that ordinary window glass removes anti-rachitic radiations. A variety of translucent substitutes have been developed. Tisdall and Brown,[27] by experiment, estimated that these glasses transmitted only 25 to 50 per cent of the anti-rachitic radiations. The glasses, however, have since been improved and now are said to transmit at least 50 per cent. The anti-rachitic effect of sky shine, except immediately adjacent to the glasses, Tisdall and Brown found, was almost negligible. The reason is that as one moves further and further from a window, the area of sky which can reflect through it becomes so reduced that the quantity of ultra-violet rays it can give off is only a minute fraction of the total. In order to obtain much benefit from rays through ordinary windows glazed with special glass, it is necessary to receive the direct rays of the sun. In the winter, when ultra-violet light is most needed, these glasses, for practical purposes, are almost useless.

The quantity of sunlight.—An interesting consideration is the relationship of the quantity of actual sunlight to rickets. Hess[5] finds that of New York, Paris, Berlin, Stockholm and London, New York has by far the greatest number of hours of actual sunshine, and London the

smallest. New York has approximately the same number of hours during February and March as has London during the months of June, July and August. As Hess points out, rickets develops during the winter in New York, whereas it undergoes cure in London during the summer. Glasgow, notorious for its rickets, has about the same number of hours of sunshine as London. A saving factor in cloudy parts of the world, such as England, must be the reflected ultra-violet light from the sky.

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