THE RECOGNITION OF SCURVY WITH ESPECIAL REFERENCE TO THE EARLY X-RAY CHANGES

BY

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Twelve years ago, under the auspices of the United States Children’s Bureau, Dr. Martha Eliot undertook the prevention of rickets in a district of New Haven, Connecticut, numbering 12,000 inhabitants. At once it became necessary to have more accurate knowledge than was available concerning the criteria for the diagnosis of early rickets in the x-ray films. Accordingly comparative x-ray and histological studies of the bones of all children dying from any cause in the paediatric service of the New Haven Hospital were begun, and have been continued at the Harriet Lane Home, Johns Hopkins Hospital, Baltimore. Altogether examinations have been made of the bones of 532 children between the ages of two months and two years. In the course of the work scurvy was discovered in a number of patients in whom the disease had not been suspected during life, and in this way it was learned that scurvy was escaping diagnosis. Moreover, as the result of the comparisons of the x-ray films of individual bones with their histological counterparts certain early x-ray manifestations of scurvy were observed, which had either entirely escaped attention previously or else had never received adequate notice in the literature. The case records of scurvy in the Harriet Lane files were then examined in the hope of obtaining further information. Altogether, the clinical and x-ray records in 125 cases of scurvy have been studied, and the present paper is the result. Its main emphasis is on the early x-ray signs of scurvy and their histological interpretation, but it also contains scattered observations of clinical interest and importance.

* Miss Bond is the head of the Social Service Department of the Harriet Lane Home and was kind enough to conduct the investigations in the homes of the cases of scurvy reported in this paper.
The first signs of scurvy in the x-ray films of the bones of the extremities.

The signs of advanced scurvy in the x-ray film have been described by Wimberger, McLean and McIntosh, Kato, and many others. The discussion here is confined to the early changes. The illustrations give far better ideas of them than can be conveyed in words (fig. 1, 2 and 3) and these, with all others later referred to, will be found at the end of this paper. The description now to be given serves both as explanation and legend.

Wrists (fig. 1). (The x-ray films were taken with the forearm supinated.) At the wrists the most common early sign of the disease was a defect of the outer corner of the lower end of the radius. In its earliest development it appeared as scarcely more than a fuzziness of the cortex and the slightest rarefaction of the neighbouring cancellous tissue. The cortex was so thin that it was almost indistinguishable or seemed to be lacking altogether. The general effect was to make the corner appear indistinct (fig. 1 a and k). When the lesion was further developed the defect became definite. It then most commonly took the form of a cleft or crevice just underneath the lattice.* The cleft included the cortex as well as the shaft. It showed various degrees of development in different cases. In some the cleft was so slight as to escape observation until especially looked for (fig. 1 b). In others it extended a short distance into the substance of the bone or even half-way across (fig. 1 m). In still others, the corner had the appearance of having been torn away from the body of the shaft (fig. 1 e, f, g and h). The torn part was the lattice; the tear had occurred through a rarefied zone which could be seen extending beyond the detached fragment. Instead of a cleft, the outer corner was marked in some bones by a triangular area of rarefaction. The cortex adjacent to this appeared extremely thin. The normal contour of the bone was preserved (fig. 1 i and k). In some cases the outer corner appeared rounded and thrust out beyond the outer end of the lattice and greatly rarefied. We have called this phenomenon 'bagging' (fig. 1 l).

The inner angle of the radius was affected similarly to the outer angle (fig. 1 i), but seems much less liable to injury.

The changes in the ulna were more varied than in the radius. Either corner or both were involved (fig. 1 i, g, and k). The cortex appeared thin or absent and the adjacent cancellous tissue rarefied. The rarefaction was slight and limited to the corner, or assumed a triangular shape and extended some distance into the substance of the shaft (fig. 1 k). A crack or cleft was often present (fig. 1 c and j); or the corner appeared torn off (fig. 1 a and g). The lower end of the ulna was frequently cupped (fig. 1 d, e, g, i, l, m and n); if the cortex which forms the sides of the

* We discuss the lattice at length in connexion with the histology of scurvy. It is sufficient to state here that the lattice is the framework of calcified matrix substance of the cartilage which is responsible for the dense shadow at the end of the shaft. 'Underneath the lattice' means underneath the dense scorbutic band of shadow which lies across the end of the shaft in the x-ray film.
cup was thin, the appearance in the x-ray film was that of a halo (fig. 1 d and h). Halo formation is certainly not characteristic of scurvy, though it was well marked in a number of our cases. In one case the lower end of the ulna showed a spicule shaped like a thorn protruding from the rim of the cup (fig. 1 o).

When the radius and ulna were viewed together, relationships were noted which seem worth mentioning. In some instances the lattice was torn off at the outer corners of both bones (fig. 1 f). In others it was torn off at the outer corner of the radius and the inner corner of the ulna. In still others triangular zones of rarefaction were present at the outer corners of both bones (fig. 1 i), and in yet others the outer corner of the radius and the inner corner of the ulna were both affected in this way (fig. 1 k). In a number of cases in which the scorbatic process was probably of longer standing, the end of the ulna and the adjacent part of the end of the radius appeared compressed, whereas the outer part of the radius showed a cleft as if pulled apart (fig. 1 d, e, m and n). In an extreme case the lower end of the ulna had obviously been crushed to such an extent that the lattice had been absorbed (fig. 1 e). In another case the lattice had been shifted inwards so that it overlapped the shaft on its inner aspect (fig. 1 m). The carpal and metacarpal bones showed nothing characteristic.

Ankles (fig. 2). (The x-ray pictures were taken in the lateral position.) The early manifestations of scurvy were more frequently noted at the ankles than at the wrists and were of the same general nature. By far the most characteristic and constant change was a defect at the anterior corner of the tibia. The defect took the form of a spot of rarefaction involving both cortex and cancellous tissue (fig. 2 a, b and c), a cleft (fig. 2 d, e, g, h, l, m and o), bagging (fig. 2 f and i), and bagging and cleft (fig. 2 j). As in the case of the radius the cleft varied from a degree at which it was just discernible on careful examination (fig. 2 e and d) to one in which it was most obvious, and extended one-third or one-half the way across the bone. In some instances the corner seemed drawn out to an abnormally sharp point (fig. 2 k). In one the anterior corner appeared cracked off (fig. 2 m). In many the posterior corner as well showed the scorbatic lesion: cleft (fig. 2 a, 1 and o); bagging (fig. 2 c) and over-extension and pointing of corner (fig. 2 d, f, i and k). It is significant that cleft formation occurred far more commonly at the anterior than at the posterior corner. Because of the super-imposition of the shadow of the fibula the changes at the posterior corner of the tibia were often obscured. In fig. 2 o, epiphyseal separation seemed imminent because of the proximity of the anterior and posterior clefts. The lattice was curved and appeared to rest against the end of the shaft as the rocker of a rocking chair rests against the floor. In fig. 2 b, a cleft must have been present but became obliterated as the result of the compression. In contrast to this, in fig. 2 e, the lattice has not been pressed back, hence the cleft is visible.
The fibula was much less affected than the tibia, but in occasional cases showed clefts at the anterior corner (fig. 2 d), usually occurring in association with similar clefts at the anterior corner of the tibia. In a number of cases the fibula was cupped and either the anterior or posterior angle was drawn to an unusually fine point. The lower end of the fibula in many films was obscured by the presence of the nucleus of ossification of the tibia, or, as already stated, by the tibia itself. The tarsal and metatarsal bones show nothing characteristic.

Shoulders, elbows and knees (fig. 3). (The shoulders were studied only in the antero-posterior position.) The characteristic early lesion was a cleft immediately underlying the lattice at the outer corner of the end of the bone. It varied from a minute spot of rarefaction to a crevice which extended half through the breadth of the bone (fig. 3 a and b). In cases in which scurvy had become more advanced other changes developed, such as cupping of the outer half of the end of the bone and over-extension and pointing of the corners. However, the cleft just mentioned was the only pathognomonic early sign noted.

(The elbows were examined in a position of partial flexion with the forearms supinated. The angle was about 135 degrees. The arm was slightly abducted at the shoulder. The upper end of the radius lay in almost complete supination.) The only positive sign found was a nick at the upper end of the radius in three cases. In two the nick was situated at the inner corner and in the other at the outer corner (fig. 3 c and d). In some cases the lower end of the humerus showed a thin lattice band which was not characteristic of scurvy. It was noteworthy that, with the exception stated, the elbow appeared free from involvement, even though lesions were well marked at wrists and ankles.

(The knee was examined in the lateral view only.) The study was productive of much less of value than in the case of either wrist or ankle. The lower end of the shaft of the femur presents so uneven a surface in the lateral position, that super-imposition blurs the outline. Only in advanced cases were characteristic lesions delineated. The upper end of the tibia was more satisfactory than the lower end of the femur, but not as valuable as the lower end of the tibia or the radius and ulna at the wrists. An early sign at the upper end of the tibia was a cleft between the lattice and body of the bone at the posterior corner. As usual with scurbitic clefts, its size varied from being just perceptible (fig. 3 e) to a crevice extending one-third or more toward the front of the bone (fig. 3 f and g). In some cases a cleft was found anteriorly as well as posteriorly (fig. 3 g). The upper epiphysis of the tibia is shaped like a yachting cap, with the vizor overlapping the tibial tuberosity. (Fig. 3 i, shows complete epiphyseal separation and is included here in order to show what the shape of the epiphysis actually is.) In a number of cases the characteristic change was a spot of rarefaction under the vizor, which made the latter conspicuous and appear to stick out from the front of the bone (fig. 3 e, h and g). It is not certain how specific of scurvy the changes enumerated at the upper end of the tibia actually are, with the exception of the clefts. The upper end of
the fibula was not of much aid, as this bone is too protected by the tibia. In an occasional case it showed slight cleft formation at the posterior corner. The anterior corner was obscured in most of our films by the overlying shadow of the tibia. Because of lack of good x-ray films observations have not been made on the upper end of the femur.

**Ribs.** These bones are affected in scurvy as in rickets earlier than the bones of the extremities, but the costochondral junctions, which are the sites of predeliction for the disease, cannot be photographed clearly enough in the living child to render them satisfactory for study.

**Distribution of the lesions** (fig. 4). An x-ray of the arm and leg from one patient has been reproduced in order to illustrate the grouping of the lesions in the different bones and their relation to each other. The signs of scurvy are so characteristic at both wrists and ankles as to be pathognomonic. Those at the knees also seem definite. An unusual lesion at the knee is the minute cleft at the posterior corner of the upper end of the fibula. The absence of evidences of scurvy at the elbow is usual. The impression has been gained that the lower end of the tibia, as seen in lateral view, most commonly shows evidence of the disease. On the other hand, in some children the lower ends of the radius and ulna have exhibited the more striking and characteristic lesions. Characteristic signs have been found in the upper ends of the humeri in x-ray pictures taken to show the chest and, as a result, scurvy has been recognized. Probably antero-posterior views of the knee and ankle would have furnished comparatively early and definite evidences of the disease, had they been available. But it must be remembered that good films of the legs in the extended position are exceedingly difficult to obtain in scurvy on account of the induced pain.

**The explanation of the early signs.**

Certain growth processes in scurvy which are fundamental to its comprehension must first be discussed. Some are normal, some pathological.

1. In scurvy calcification takes place in the normal manner. It is inhibited only if rickets is present.

2. The epiphyseal cartilage continues to produce new cells, that is, it continues to grow, even after the scurvy has developed. Growth in length of the long bones, therefore, keeps on, unless the disease become so severe that even proliferation in the epiphyseal cartilage stops. The practical significance of this for us is that in scurvy the cartilage keeps giving off its framework of matrix substance for the shaft to take over and form into bone.

3. Osteoblastic activity in the shaft and growth activity in the proliferative cartilage are distinct processes, carried on by different kinds of cells, apparently under different control. Although, then, the cartilage cells divide and growth in length goes forward, osteoblastic bone formation stops* and growth in thickness ceases. This means specifically that new

* Osteoblastic activity may not stop completely in scurvy; it virtually stops, however. New bone may form at scattered points where, presumably, the growth stimulus happens to be great enough to affect the cells even in spite of the inhibiting influence of the vitamin C deficiency.
bone not only no longer forms on the surfaces of old bone, but does not develop on the framework of calcified matrix substance which the cartilage keeps furnishing.

(4) The resorption of bone which was already formed before the scurvy began goes on at an increased rate throughout the skeleton. The histologist finds evidence of this by means of the microscope; the best evidence, however, is derived by inference from the x-ray film, which shows progressive thinning of the shadow cast by the bone, the longer the disease lasts. Destruction of bone, though universal in scurvy film, occurs in certain regions much more rapidly than in others, and the regions at which it takes place most rapidly of all are those which in health were the seats of most active growth. Under normal conditions bone is formed far more rapidly at the end of the shaft just under the cartilage than elsewhere, and it is in this region in the bone which suffers from the rarefying process cut of all proportion to the rest.

(5) It is most interesting that, though the lamellar and trabecular systems of the bone undergo disintegration in scurvy, as just stated, the framework of calcified matrix substance which the cartilage keeps furnishing to the shaft, relatively speaking, escapes. Normally this is in greater part destroyed almost as soon as formed. The practical meaning is that the framework of calcified matrix material, as such, keeps accumulating and increasing in size at the end of the shaft.

(6) The capillaries of the shaft leak plasma and also here and there permit the passage of red blood cells in small or, sometimes, in very large numbers. The result is that capillary hemorrhages occur at various points in the substance of the bone, and sometimes large haemorrhages develop either in the marrow cavity or under the periosteum. It is known that the walls of the blood vessels become permeable in scurvy, and unable to hold their contents. It is suspected that the circulation of nutrient fluids is also impaired, and the osteoblasts and bone corpuscles no longer receive adequate nourishment. But of this possibility no direct knowledge has been obtained.

(7) The marrow cells migrate away from the ends of the bones where growth is occurring rapidly, leaving the supporting connective tissue framework exposed to view. This connective tissue framework is termed in German, ‘Gerüstmark,’ or marrow framework, as translated into English.

These are the pathological processes which are at work in scurvy. The first six are the ones responsible for the characteristic phenomena in the x-rays; the last is one of the cardinal histological signs of scurvy, but does not influence the x-ray picture. It is now possible to attempt to give understandable explanations for the x-ray changes. The scorbuitic lattice will first be described.

Perhaps the most characteristic phenomenon in the x-ray film in cases of scurvy and the one earliest to appear is the dense shadow lying across the end of the shaft. This shadow is cast by the scorbuitic lattice (fig. 5, 6, 7 and 8). In brief outline, the lattice is the framework of calcified matrix substance of the cartilage. It is bare, since it is devoid of any covering of bone. Present in embryonic form in normal growing bone in the provisional zone of calcification of the cartilage, it reaches pathological proportions in scurvy because, while its formation continues, its destruction ceases. It casts a dense shadow because of its great content of lime.
In scurvy, as under normal conditions, the capillaries keep invading and destroying the columns of cartilage cells as fast as the latter mature, but leave untouched the supporting calcified framework of matrix substance. Accordingly, the cartilage keeps leaving behind, as it grows, its framework of calcified matrix material. The continuous formation and giving off by the cartilage of this framework of calcified matrix substance is a normal growth process, which is preserved in scurvy unless the disease becomes most severe. Under normal conditions the framework of calcified matrix substance is destroyed in its greater part as soon as, stripped of its cartilage cells, it is reached by the invading capillaries of the shaft. This destruction takes place close to the junction of cartilage and shaft. In the healthy infant perhaps four-fifths or nine-tenths of it is destroyed almost immediately after its formation. Only here and there in the lines of strain constituent parts of the framework escape destruction, become covered with encasements of bone as the result of the activity of the osteoblasts, and made over into the trabecular system. Under the influence of scurvy the framework of calcified matrix substance of the cartilage is either not destroyed at all, or else is most imperfectly destroyed.* This cessation in the orderly destructive process is a pathological condition which occurs in scurvy and, also, congenital syphilis. Bone is a living tissue, as already pointed out, and like other living tissues, is dependent on the steady flow of nutrient material, and cannot withstand the conditions imposed by scurvy. Undoubtedly the reason why the framework of calcified matrix substance escapes destruction in scurvy is that it is a dead tissue.

Normally the osteoblasts rapidly encase the parts of the framework of calcified matrix substance which escape destruction with layers of bone. Because of their inability in scurvy to cover over any part of this framework, the framework remains bare of bone. This breakdown in bone building in this particular place is a most characteristic abnormal phenomenon not only in scurvy but also congenital syphilis. It has great practical importance. Bone is a fibrillar tissue; without reinforcement with covering layers of bone the framework of calcified matrix substance has little strength and fractures easily. The framework of calcified matrix substance is exceedingly dense. Its density is readily understandable as soon as it is recalled that in the original cartilage in which it was formed its walls were separated from each other only by the columns of cartilage cells. In other words, in the aggregate there is a great mass of it. Moreover, because it is pure matrix material without cells, it probably takes up lime salts in greater concentration than bone. The bone corpuscles themselves do not take up lime salts. Its content of calcium per unit of volume must be much greater than bone whose trabeculae are, relatively speaking, few in number and widely separated. Undoubtedly a subjective factor is concerned in the dense appearance of the lattice shadow in scurvy. The faintness of the shadow cast by the rest of the bone, and in particular

* The even regular physiological destruction stops. Irregular focal destructions take place, in particular where fractures have occurred.
by the zone of rarefaction lying next door, makes the lattice shadow seem denser than it actually is*

The lattice stains a deep blue with haematoxylin and shows no cellular structure, except where cartilage cells have been entrapped and preserved from contact with the capillaries. Bone, in contrast, stains pink with eosin and shows its contained bone corpuscles. The two can be easily distinguished from each other by histological methods, and the identity of the lattice with the calcified matrix substance of the cartilage established beyond a doubt. In figures 5 and 9, the lattice is beautifully shown, and with its black appearance and dense ‘thickety’ structure forms a marked contrast to the sparse thin trabeculae of the cancellous tissue.

The idea that in scurvy, and also in congenital syphilis, the lattice of calcified matrix substance is responsible for the heavy band of shadow at the end of the shaft and for the peculiar liability of the end to fracture is new. Years ago Fraenkel\(^1\), in his monograph on the x-ray manifestations of scurvy, ascribed the bright band of shadow to the ‘Trümmerfeldzone.’ The ‘Trümmerfeldzone,’ according to him, was the region of fracture. The shadow was produced by the fragments of the matrix material and adjacent trabeculae of bone, which were packed together with blood clot lying between. This idea has been accepted without question by the various writers on the subject since that time. In reality, blood clots cast no more shadow than soft tissues in general. It is not the compressed fragments of the fractured lattice which cast the shadow, but the lattice itself. In some cases the fragments are compressed; in others they are scattered or few in number. The compression factor tends to be offset by the rarefaction factor, because rarefaction seems to set in rapidly where fractures have occurred.

The zone of rarefaction (fig. 9 and 10) lies at the end of the shaft next to the lattice on the shaftward side of the latter. It is one of the classical x-ray signs of scurvy, and is always sought for, whenever scurvy is suspected. As described by Wimberger and other writers, it extends completely across the bone, but, if the disease has not progressed far in the bones, it may be only partially developed, or not present at all. The zone of rarefaction is not, therefore, a constant sign of scurvy, and cannot be regarded as being essential for the x-ray diagnosis. When fully developed, the zone of rarefaction is as broad as, and usually much

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* In the cases of advanced scurvy which have been studied the proliferative cartilage has been abnormal. The cells have been small, their formation into columns irregular and the quantity of matrix substance excessive. The scorbutic lattice is formed in the cartilage and its pattern and the thickness of its structure are determined in the cartilage. The lattice was most irregular in the cases of advanced scurvy reported here and its structure in places was much thicker than normal. The abnormal thickness in the lattice structure was also a factor in making the shadow cast an especially heavy one. We have not dared to say that scurvy affects the growth of cartilage, making it abnormal, as it does bone, because of the difficulty in eliminating the possibility that rickets, also, was present in these advanced cases and affected the cartilage. We are, however, inclined to think that scurvy does affect the cartilage, making the matrix framework more irregular and heavier than normal. The affection of the cartilage in this particular way is another reason why the scorbutic lattice casts so heavy a shadow.
broader than, the lattice. It is sharply bounded on the lattice side by the dense band of the latter; on the shaft side, however, it does not have a well-defined margin, but merges with the shadow of the shaft. On the shaft side, also, its development is often irregular, since it may extend into the shaft substance further in some places than in others. The rarefaction zone may be as conspicuous in its way as is the lattice or, on the other hand, be so poorly defined as to escape observation. In most advanced cases the zone of rarefaction may be so made in the skigram as to make the epiphysis with the lattice seem entirely separate from the shaft, without even cortex left to join the two together. Like the lattice, it is broader at the fast-growing ends of the long bones than at the slowly-growing ends, and may be visible in the former and not in the latter. In many of the films used for this study, the zone of rarefaction was entirely absent, or only partially developed. The spots of rarefaction (fig. 1 g and i; fig. 2 c) and the clefts (fig. 1 d; fig. 2 h and l) in the first cases actually represent local, probably early developments of the rarefaction zones.

When the zones of rarefaction are studied under the microscope, it is found that the trabeculae of bone are thin, and many of them show signs of surface disintegration. They are not only thin, but are widely separated from each other, obviously because intervening trabeculae have been removed. Around many of the trabeculae are swarms of osteoblasts and similar groups of cells mark places where the trabeculae formerly were. Often in the centre of the groups of cells trabecular fragments are found. The marrow cells have largely or entirely gone from the spaces between the trabeculae. Scattered sprinklings remain. The connective tissue framework of the marrow which is revealed to view as the result of the departure of the marrow cells resembles loose embryonic tissue. Capillary haemorrhages are almost always present (fig. 11) and large haemorrhages may be present (fig. 6). The cortices bounding the rarefaction zones usually are reduced to shell-thickness and in places may be lacking altogether. On the outer and inner surfaces of the cortices are usually found many osteoclasts, and osteoclasts also are scattered in the regions of the trabeculae.

The fractures of the lattice (fig. 6, 8, 12, 18) are conspicuous in the histological preparations, but are invisible in the x-ray films, though their presence can often be inferred in the latter. The zone of rarefaction, on the contrary is much more conspicuous in the x-ray film than in the histological preparations; indeed, on examination of histological preparations with the microscope, it is often a cause for surprise that the region should have appeared so rarefied in the x-ray film. As in the case of the rarefaction zone, fracture may extend all across the bone, giving rise to true epiphyseal separation. On the other hand, it may extend only part way across or may occur at points widely separated from each other. The fractures take place almost always through the lattice and are situated most commonly close to its junction with the bone. On examining sections under the microscope it seems astonishing that the lattice should have given way instead of the greatly rarefied trabeculae of the shaft immediately adjacent, because the latter are so thin and
widely separated, whereas in contrast the lattice is thick and abundant. Though, as already stated, the fractures most commonly take place in the lattice close to its meeting points with the bony trabeculae, they may occur through the middle portion and sometimes they involve the bony trabeculae as well. Fractures do not occur through the lattice immediately beneath the cartilage. Thus, when the epiphysis separates from the diaphysis it always carries some and usually the greater part of the lattice with it.

When the lattice is the seat of extensive fracture the microscope shows its fragments lying in all directions. It evidently shatters, as might be expected of an extremely brittle substance, like glass or china. The fragments may appear as rounded or quadrilateral masses or as splinters. In some cases they are pressed together; again one fragment is found impaling another or several fragments are impacted into one large mass. Around and between the fragments it is usual to find masses of fibrin and in some cases recent haemorrhage. In the regions where fractures have occurred great quantities of cells, evidently osteoblasts, are found which surround the broken fragments like swarms of bees (fig. 14). In many cases these cells seem to have settled thickly on the fragments and the fragments seem in process of disintegration. Where fragments have been jammed together, the blue-staining homogeneous material has changed to a granular detritus which stains pink with eosin. Many giant cells are found in the regions of the fractures, but do not seem to be the chief agents which are bringing to pass the destruction. When the fractures are of less extent the same picture obtains, but is more circumscribed.

Before leaving the subject of fractures, attention must be called to the frequency with which they occur. They are present in the fast-growing ends of the long bones in every case in which the x-ray picture shows a well-marked lattice and must be numerous long before epiphyseal separation occurs. Experience would indicate that in scurvy breaks occur at this point and then at that point and keep multiplying until the end of the bone becomes so weakened that it gives way entirely. The idea that the disintegration of the lattice began so early, advanced so insidiously and was so universal was a new one. Of course, it is understood that as the result of sudden strain the lattice may shatter completely across in a moment.

The spots of rarefaction at the corners of the bone (fig. 15, 16, 17, 18, 19, 20, 21, 22, 28) present microscopic pictures such as might be expected from the descriptions already given. Where 'corner' spots of rarefaction appear in the x-ray films, the cortex is found to be much thinned and in some places it has disappeared altogether. The cortex always shows fracture, and about the points of fracture rarefaction processes seem to be in full operation. The trabeculae of the adjacent cancellous tissue show signs of disintegration. Large areas are entirely devoid of trabeculae. In these one finds clumps of osteoblasts marking the spots where the trabeculae formerly were and perhaps in the middle of them trabecular remnants in the form of a pink-staining granular detritus. Between the trabeculae connective tissue is found. In some
places this connective tissue has a loose structure and resembles embryonic tissue (the supporting connective tissue framework of the marrow); in other places, particularly along the cortex, the cells are quite closely packed together, as in the fibrous marrow of rickets. Haemorrhages may or may not be present. The reason for the appearance of rarefaction in the x-ray picture is the reduction in size, or even partial absence, of the cortex and in the number and size of the trabeculae. The rarefaction may extend into the lattice.

The typical clefts, cracks or crevices of rarefaction at the 'corners' of the ends of the shafts have not been studied histologically, for the reason that none of the infants autopsied showed well-marked examples. The impression is gained from the x-ray pictures that the bone substance has been pulled apart at the site of the cleft, and it is expected that evidence of trauma in the soft tissues as well as in the trabeculae will be found. From the examination of the spots of rarefaction at the corners just described, it is practically certain that any evidence of trauma would be limited to the osseous elements and that the soft tissues would have stretched and would show no evidence of injury. It would be anticipated that in the areas embraced by the clefts the cortex would have disappeared or been reduced to extremely thin shell-like fragments of bone and that the trabeculae would have entirely disappeared or been represented by residual fragments or detritus surrounded by masses of osteoblasts. The marrow cells would have disappeared and connective tissue would fill in the area. Strands of fibrin would be found running through it which was in process of invasion and organization by the blood vessels. Haemorrhages might be found or pigment from previous haemorrhages.

'Bagging' of the corners in the x-ray film is best understood through examination of the accompanying illustrations (fig. 24, 25, 26, 27, 28 and 29). In 'bagging' the essential condition is the atrophy or fragmentation of the cortex. The loss of cortical support permits the soft tissues to be squeezed outwards when they are compressed as the result of impaction of the shaft against the epiphysis with fragmentation of the lattice. The 'bag' cover is the periosteum.

Over-extension and pointing of the corners is related to 'bagging'; this can readily be understood from the illustrations (fig. 30, 31). When this phenomenon is present in the x-ray film, the lattice will be found to have developed around the side of the proliferative cartilage. The absence or great reduction in the cortical shadow just under the lattice and the rarefaction of the adjacent cancellous tissue throws the lattice into a sharp relief in the x-ray film and makes the latter appear as a long drawn-out corner.

The rarefaction of the shaft, upon which so much emphasis is placed in the x-ray studies of scurvy, is not an early sign of the disease and is not pathognomonic, since it occurs in other illnesses in which the
bones undergo rarefaction. In some of the present cases it was extremely marked (fig. 5). The shafts of the long bones are merely cylinders of bone filled with soft tissue. The soft tissue does not cast shadows of any consequence. The 'cortical shadows' in the x-ray film are cast by the sides of the cylinder, which lie parallel to the rays and hence offer thick obstacles for the x-ray to penetrate. The shadows of the 'bone proper' between are nothing more than the superimposed shadows of the front and back walls of the cylinder, which lie at right angles to the rays; in other words, they represent merely a double thickness of cortex. In scurvy the current teaching is that the cortices appear thin and the bone between presents a ground-glass appearance. Some students have even regarded this appearance as specific. The cortex in scurvy does become thin and the thinness comes out in the projections of the sides. The intervening bone does not present a ground-glass appearance, if the rarefaction has reached an advanced development; it is not peculiar to scurvy. The ground-glass quality is caused by an increased porosity of the cortex, and the increased porosity is the result of absorption of the walls of the tunnels in the cortex which transmit the nutrient blood vessels. These tunnels or apertures in the cortex become larger and hence the cortex actually does become more porous. The thinness of the cortex allows a small degree of porosity to show in the x-ray film.

The subperiosteal haemorrhages which constitute such a well-known sign of scurvy in the x-ray film do not, strictly speaking, come into the scope of this article. Subperiosteal haemorrhage develops only when the involvement of the bone has reached an advanced stage. The subperiosteal haemorrhages originate at the ends of the bones and extend towards the middle. They are the result of the fractures of the cortex and lattice at the end of the shaft. Presumably, when the end of the bone gives way, periosteal vessels are torn and the blood escapes under sufficient pressure to lift up the periosteum. McLean and McIntosh have shown that in scurvy the periosteum becomes loosened, so that it can be separated from the underlying cortex with a minimum of force. Their observations suggest that extravasated blood can burrow beneath the periosteum much more easily in scurvy than in health. From the x-ray point of view it is interesting that the subperiosteal haemorrhages do not become outlined and hence visible until about ten days after treatment is instituted. Ossification in the periosteal membrane covering the clot does not begin until the vitamin C deficiency is corrected. An interval is required before it progresses far enough to produce visible changes in the x-ray film.

Discussion.

Rickets is a disease of the entire bone; scurvy, practically speaking, is a disease of the growing ends, and the disturbances to which it gives rise are essentially limited to the growing ends. The weakness imparted to the end of the bone by rickets causes the end to bend; the weakness
THE RECOGNITION OF SCURVY

from scurvy causes it to break, and the finer changes in the x-ray film, such as have been described, are all due to weakening and breaking. Spots of rarefaction and clefts in certain characteristic regions have been described, for example, in the outer rim of the lower end of the radius and in the anterior rim of the lower end of the tibia. Unfortunately the variety of views in these studies was not complete; in particular, views of the ankle and knee in the antero-posterior position were lacking. If x-ray examinations had allowed the study of the shaft ends from all sides, doubtless other favourite sites for lesions would have come to light. The examiner, in search of early signs of scurvy, does not need to know the favourite sites, however, if he will make use of his knowledge of the modus operandi of the disease. He will then look for the scorbutic lattice and evidence that the lesions in question are in the lattice or adjacent portions of the shaft. In particular he will investigate the peripheral region of the end of the shaft, since this is where strains seem first to produce their effect and early lesions develop in consequence. Lesions such as have been described at the periphery of the lattice, especially if they are multiple, are characteristic of scurvy. The only other disease which causes a lattice to form is congenital syphilis. The characteristic lesions which have been found in scurvy have never been seen in congenital syphilis, although the lattice often fractures completely across in syphilis.

The sites of election for lesions in the lattice are the points where it is especially apt to be subjected to stress and strain. It has been assumed that the anterior rim at the lower end of the shaft of the tibia is a point of especial selection, because of the pull of the weight of the foot in the recumbent position. Similarly, the outer rim of the lower end of the shaft of the radius tends to give way because of the strain imposed by the weight of the hand when the arm is in mid-pronation, a hypothesis which is further borne out by the frequent occurrence together of cleft formation (pulling apart) at the outer side of the radius and evident compression of the end of the ulna and adjacent part of the end of the radius (fig. 1, m and n). A cleft forms in the outer part of the upper end of the humerus because of the pull of the muscles in abducting the arm and supporting it in the abducted position. The fibula is little affected because it is splinted by the tibia. Fractures of the lattice are exceedingly common in the ribs because of the strain of the respiratory movements. The impression has been gained that the fracturing force required to injure the scorbutic lattice, when the latter is well developed, is extremely slight. It is believed that the lattice, unsupported by covering layers of bone, has not much more strength than so much chalk.

There is another factor which determines the vulnerability of the end of a bone in scurvy, and that is the rate of growth. Scurvy, like rickets, affects the end of a bone in direct proportion to its rate of growth. The more rapid the growth, the greater the lattice production, and the broader the lattice becomes, the greater the liability to break. The exemption from x-ray signs of scurvy of the ends of the bones meeting at the elbow
joint is explained by their slowness of growth, as also in the case of the small bones of the hands and feet. There has, however, been found with the microscope crumbling of the shell of lattice at the posterior corner of the head of the olecranon process of the ulna, though it grows most slowly (fig. 32 and 33). It is attributed to pressure from the weight of the arm resting on the elbow. The middle ribs become especially fragile at their anterior ends because of their exceedingly rapid growth. It is suggested that the rationale in the development of the early lesions is as follows:—As the result of the deficiency in vitamin C, healthy growth of bone ceases, the scorbutic lattice forms, the adjacent trabeculae of bone and the enveloping cortex become thin and the end of the bone correspondingly weak. The factor of strain is supplied. The part of the weakened end which bears most of the strain gives way. Bone cannot withstand strain in scurvy; first, all power of repair through the production of new bone is lost as the result of the specific action of the disease; second, strain stimuli, which, under normal conditions, would cause new bone to form, seem to have a reverse action and hasten the destructive process. Rarefaction proceeds rapidly. Broken lattice or bone fragments are absorbed entirely. The weakness advances and extends. Other trabeculae further within give way. A vicious circle is established; the process advances with increasing rapidity until it reaches through the entire breadth of the bone.

The scorbutic deformity of the rib.

In the normal infant the enlargement of the costo-chondral junction is greater on the inside of the thorax than on the outside. In rickets and scurvy the disproportion becomes even more pronounced. Inasmuch as only the external surface of the costo-chondral junction is accessible to clinical examination, it alone will be considered. The palpatory phenomena in cases of moderate severity will first be described, and afterwards in cases in which the disease has reached its most advanced stage of development.

In some moderately developed cases the examining finger passes along the rib in a straight line until it reaches the chondral junction, when, without encountering any ridge, it slips into a little hollow not more than 2 cm. wide, formed by the cartilage. The hollow lies just beyond the end of the rib, as if, as is actually the case, the cartilage at its beginning were bowed slightly inwards (fig. 34 b and c). If the examiner palpates in the reverse direction, that is, from cartilage to rib, the hollow is better felt. As soon as the finger crosses the hollow, it encounters a barrier formed by the end of the rib, which may be so marked as to make it seem as if the cartilage had fallen back slightly, leaving the end partially uncovered. In other cases a slight variation is encountered in that the actual line of junction of rib and cartilage is elevated into a ridge, which the palpating finger is conscious of surmounting just before slipping into the depression (fig. 34 d). In still other cases a further variation is found in the form of a hollow on the rib as well as on the cartilage side of the junction (fig. 34 a and e). In these circumstances the line of junction
forms quite a sharp ridge, from the summit of which the adjacent sides of the depression slope away like the sides of a roof. If the infant is not fat the hollows in the costal cartilages, as well as the scorbutic rosary, are visible and appear as grooves or gulleys, bounding the elevations on their inner side. The scorbutic costo-chondral junction feels hard and whatever deformity is present seems sharply limited to it. The rib

![Diagram of rib deformities](image)

**Fig. 34.**—Drawings from sections of sample ribs from five infants proved to have scurvy at autopsy. The sections were taken in the transverse direction. Consequently, the upper contour of each costo-chondral junction represents the outer surface and the lower the internal surface. The hatched portion represents the rib, the white, the resting cartilage and the stippled the proliferative cartilage. Descriptions of the different types of deformity which these drawings illustrate have been given in the text.

maintains its normal arch in scurvy. The costal cartilages of the middle ribs normally do not continue in the exact lines of the ribs, but are deflected slightly inwards. In scurvy the slight normal angles formed either remain unaltered or else become actually increased. The result is that the scorbutic deformity, then, is situated at the apex of an angle, feels angular, and acquires an additional prominence in that way. The deformity is most
marked in the fifth, sixth and seventh ribs. In the fourth it is not sufficiently well developed to be characteristic, and in the eighth, ninth and tenth is not so typical.

The opportunity of carefully examining the costo-chondral junctions has occurred in only two infants, in whom the disease had reached its most extreme development. In one, a living infant under the care of Dr. Thomas Cooley in Detroit, the thorax was sunk anteriorly. With each inspiration the sternum and cartilages appeared sucked back towards the vertebral column, forming a hollow several centimetres deep; with expiration, the hollow suddenly flattened. In this case the junction of cartilage and rib was marked externally by a sharp angle, which in expiration was about 45 degrees and in inspiration became a right angle. In the other infant, seen only after autopsy, the middle group of costo-chondral junctions presented contours such as have just been described. The eighth and ninth ribs, however, showed a true 'bayonet' type of deformity (fig. 35). The plateau of the rib surface suddenly stopped; the palpating finger passed over a ledge and dropped, as it were, to the costal cartilage. This is the only example encountered meriting the term 'bayonet deformity,' and in this case it was limited to the lower true ribs.

In autopsy cases four different pathological conditions have been observed which influence or determine the contour of the costo-chondral junction in scurvy.

(1) In all cases of scurvy in which involvement of the costo-chondral junctions is clinically apparent, the cartilage-rib junction is actually broadened. The disturbance of growth caused by scurvy renders the junction weak. Nature responds by broadening the opposing surfaces of the junction. As the result of the resolution of forces at the cartilage-rib junction, the proliferative cartilage develops new columns of cartilage cells along its periphery and these are thrown out obliquely or even at right angles to the main axis of the shaft. As seen in the single plane of the histological preparation, they radiate out from the sides of the proliferative cartilage fanwise. Inasmuch as the growth of shaft always follows the growth of the cartilage in an obligatory fashion and is really determined by the latter, the shaft keeps developing up along the sides of the proliferative cartilage until it encircles the latter. Ultimately the proliferative cartilage becomes shaped like the head of a mushroom or a knob which the end of the shaft embraces in a cup. In this way the cartilage-shaft junction becomes broadened and the broadening shows itself internally and externally through a ridge or elevation. In some cases of scurvy further broadening of the cartilage-rib junction is due to the weakness of the cortices and lattice which give way with the result that the end of the shaft becomes impacted against the cartilage centrally and the peripheral portions are forced outwards.

(2) Normally, as stated, the costal cartilages of the middle and lower ribs do not continue in the line of the rib, but are bent slightly inwards and also upwards. This change in direction occurs abruptly at the costo-chondral junction. In scurvy this internal bend of the cartilage may become increased, in some cases enormously. As is well known, in cases of severe scurvy the thorax is flattened. The flattening is due to the fact that the sternum and adjacent cartilages occupy a more posterior posi-
tion than in the normal thorax, and this backward position has been caused by a sharper bend at the cartilage-shaft junction. The sharper bend is the result of fracture of the lattice. The increased angle formed by rib and costal cartilage causes the enlargement of the costo-chondral junction to be sharp and angular and to protrude. Incidentally, in this connection attention must be called to the fact that the fracture of the ribs through the lattice, which is apparently so common a phenomenon even in moderately severe scurvy, breaks the continuity of the costal arch and permits false motion with the respiratory movements. In the severe case seen in Dr. Cooley’s clinic the cartilage moved on the rib at the seat of the break as if hinged. Even in moderately developed examples of the disease the cartilage must rock slightly to and fro with inspiration and expiration on the broken end of the rib, even though the movement is not apparent. The fact that a rocking movement occurred at the site of fractured lattice during life probably accounts for some of the peculiar features of the histological picture of the fracture region in the rib.

(8) As the result of the fracture of the lattice there may be an internal shift of the costal cartilage on the shaft. This is illustrated in fig. 36. From the study of the direction of the strands of fibrin and blood vessels and the relation of the fragments of the broken lattice to each other it can be seen that the proliferative cartilage has slipped inwards on the shaft. This internal shift would tend to produce a hollow on the cartilage side of the junction and would bring the end of the rib into prominence externally. At the beginning of these studies, it was believed that this shaft accounted for the rare phenomenon of the ‘bayonet deformity.’ It was found, however, that the shift inwards was either not present at all or was very slight and in the single instance in which the deformity merited the term ‘bayonet,’ the underlying pathological condition was entirely different.

(4) In the single instance of ‘bayonet deformity’ it was the cartilage which was deformed and responsible for the musket-barrel-bayonet relationship. The resting cartilage had been pulled inwards on the proliferative cartilage where the nutrient vessels of the proliferative cartilage, the cartilage canals, enter and the overlapping proliferative cartilage on the outer side had been forced against the resting cartilage. This same bend has been seen in the cartilage in one other case, which was not sufficient, however, to produce a ‘bayonet’ deformity. It is believed that this peculiar deformity of the cartilage was the result of the pull of the diaphragm which is attached to the cartilages of the lower ribs. It is possible that the resting cartilage itself is weakened in scurvy so that it bends more easily than normally.

Thus the cartilage-shaft junctions of the ribs in scurvy are deformed in different ways and the fact of differences in contour in different cases or at different levels in the same infant does not seem peculiar.

The explanation for the peculiarities of the deformity of the costo-chondral junction in scurvy is that the rib remains rigid and the weakness is sharply limited to the costo-chondral junction. In rickets (fig. 37) the deformity at the costo-chondral junction owes its peculiarities in large part to the fact that the rib itself becomes weak and bends inwards. In scurvy the grooves at the sites of the costo-chondral junctions or Harrison’s grooves so characteristic of advanced rickets, are never
seen. The bony thorax remains firm and retains its normal contour. If any part yields, it is the cartilages and sternum. In advanced scurvy, as in the case cited, the thorax gives in the same way as does the normal thorax when severe inspiratory obstruction exists; it is the lower part of the sternum and the cartilages which is sucked inwards. The maintenance of the rigidity of the ribs, while the cartilage bends is a basic fact in the understanding of the characteristic signs of scurvy at the costo-chondral junctions.

The value of the scorbutic deformity at the costo-chondral junction in the diagnosis of scurvy.

The enlargement of the costo-chondral junction in scurvy cannot be classified as an early sign, though it is probably the earliest clinical sign produced by the skeleton. Of the cardinal signs of scurvy the enlargement of the costo-chondral junction is certainly one of the earliest. But it cannot appear until the bone changes are so advanced that the lattice has time to form and to give way. A blindfolded person palpating the chest of normal, rachitic and scorbutic infants would make many mistakes. The costo-chondral junction of the healthy infant may be so ridged as to suggest scurvy and the hollow in the proximal portion of the costal cartilage may be present, also. Particularly when the thorax is 'square shaped,' the cartilage and shaft form an angle which is quite marked, and the line of junction is thrown up into a ridge similar to that seen in scurvy. In rickets the costo-chondral junction may show characteristics of scurvy. The deformity of the costo-chondral junction constitutes one of the cardinal signs of scurvy. Its great usefulness, however, is to suggest scurvy. Time and again it leads to the discovery of the disease. In rare instances it may be so marked that it establishes the diagnosis. An interne was on a visit of inspection in a distant paediatric clinic. A baby on a stretcher was waiting to be wheeled to the operating room. Mechanically the interne felt the costo-chondral junctions and suddenly realized that a scorbutic deformity was present. He hastened to report. The director of the clinic and he arrived in the operating room to find the surgeon bewildered by the discovery of a subperiosteal hematoma.

The nineteen cases studied at autopsy.

As stated at the beginning of this paper, studies have been made of the bones of 582 children between the ages of two months and two years, dying from a variety of diseases in the wards of the Harriet Lane Home. Among this group of children nineteen have been found, through histological examination of the bones, to be suffering from
THE RECOGNITION OF SCURVY

scurvy (table 1). In only two of these nineteen children had the diagnosis been made clinically. In seventeen the disease was never thought of as a possibility during life. In the pathological department scurvy was recognized in five of the nineteen cases, and in two additional cases it was suspected. The pathologists, then, in the course of their routine autopsies did not recognize the disease in twelve cases. In most instances death occurred so soon after admission (in one case before the examination was complete) that adequate opportunity for clinical examination was not given. In the majority of the undiagnosed cases the obvious illness (for example, pachymeningitis, pneumococcal meningitis, pneumonia, dysentery) was so severe as to absorb the entire attention and make consideration of additional disease seem superfluous. In some of the cases the scurvy was only slightly developed and not recognizable clinically, though in others it was well developed and in one flagrant. The scurbutic deformity of the rib, which might have given the clue, was passed over as rachitic in several instances. In one instance the physician had suggested scurvy but abandoned the thought when the x-ray department reported that the films showed rickets. The reason that the disease was overlooked on routine autopsy examination was undoubtedly that not one of the pathologists happened to have a special interest in diseases of the bone in children. These experiences have had a special importance because they have awakened the consciousness that scurvy had been occurring more frequently than had been realized and had been escaping diagnosis. The discovery of these cases had an unsettling effect. Previously scurvy had been considered easy to recognize. Now it is believed that even among these 532 autopsy cases there must be still other cases of scurvy which have not been recognized. Earlier examinations were performed before the examiners were trained in the recognition of the disease and it should be noted that the histological signs do not become unmistakable until the disease has been at work for some time in the skeleton. In this respect scurvy is so different from rickets which usually clearly declares itself plainly from the beginning. The incidence of rickets in the same series has been approximately 27 per cent.

The other data brought out in the study of this group of autopsy cases are not important. It is perhaps interesting that in five cases the age was five months or less and that in one it was three. Hess, in his book on scurvy, states that the youngest age at which he has found the disease was four-and-a-half months. Perhaps a noteworthy fact is the association of scurvy with the major illnesses. It has been looked for in connection with digestive disturbances, failures in nutritional anaemia, dysentery, etc., but, quite illogically, not as an accompaniment of lobar pneumonia, meningitis, etc. Great difficulty was experienced in deciding in regard to the presence or absence of an associated rickets. In nine out of the nineteen cases histological examination made it certain that rickets was present, in four left the matter in doubt and in six indicated
### TABLE 1.

**Infants dying in the Harriet Lane Home in whom scurvy was proven by special histological study of bones.**

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Colour</th>
<th>Age in months</th>
<th>Clinical Diagnosis</th>
<th>Pathological Diagnosis</th>
<th>Rickets</th>
<th>Scurvy diagnosed clinically</th>
<th>Scurvy diagnosed at autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. S. 559</td>
<td>M.</td>
<td>B.</td>
<td>10</td>
<td>Scurvy; Rickets; Sickle cell anaemia; Diarrhoea with acidosis and dehydration.</td>
<td>Scurvy; Rickets; Sickle cell anaemia; Lobular pneumonia.</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>R. W. 587</td>
<td>F.</td>
<td>W.</td>
<td>3</td>
<td>Pachymeningitis</td>
<td>Pachymeningitis</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>M. S. 601</td>
<td>F.</td>
<td>B.</td>
<td>5</td>
<td>Prematurity; Prematurity; Miliary tuberculosis.</td>
<td>Tuberculosis</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>J. W. 654</td>
<td>F.</td>
<td>B.</td>
<td>6</td>
<td>Diarrhoea with dehydration; Rickets.</td>
<td>Ulcerative enteritis; Scurvy.</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>R. S. 988</td>
<td>F.</td>
<td>B.</td>
<td>11</td>
<td>Prematurity; Lobular pneumonia; Dehydration, acute.</td>
<td>Pneumonia; Scurvy (?).</td>
<td>Doubtful</td>
<td>-</td>
<td>(?)</td>
</tr>
<tr>
<td>J. W. L. 1027</td>
<td>F.</td>
<td>W.</td>
<td>6½</td>
<td>Pneumonia; Dehydration, acute; Acidosis.</td>
<td>Pneumonia; Scurvy.</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>A. P. 1135</td>
<td>F.</td>
<td>B.</td>
<td>8</td>
<td>Scurvy; Rickets; Dehydration. (Twin)</td>
<td>Scurvy</td>
<td>Doubtful</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>J. B. 1161</td>
<td>M.</td>
<td>B.</td>
<td>5</td>
<td>Dysentery.</td>
<td>Dysentery</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>M. B. 24</td>
<td>M.</td>
<td>W.</td>
<td>5</td>
<td>Pneumonia</td>
<td>Pneumonia</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Professor S. B. Wolbach, of the Department of Pathology of Harvard University, very kindly examined the histological preparations in this series of cases and either confirmed or established the diagnosis of scurvy.*
<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Colour</th>
<th>Age in months</th>
<th>Clinical Diagnosis</th>
<th>Pathological Diagnosis</th>
<th>Rickets</th>
<th>Scurvy diagnosed clinically</th>
<th>Scurvy diagnosed at autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>F. B. 198</td>
<td>F.</td>
<td>W.</td>
<td>6</td>
<td>Diarrhoea with extreme dehydration and acidosis.</td>
<td>'History of diarrhoea; Oedema of lungs; Peculiar form of osteochondritis resembling a previous case of scurvy.'</td>
<td>+</td>
<td>-</td>
<td>(?)</td>
</tr>
<tr>
<td>E. A. 440</td>
<td>F.</td>
<td>W.</td>
<td>9</td>
<td>Dysentery</td>
<td>Dysentery</td>
<td>Doubtful</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>G. H. 449</td>
<td>M.</td>
<td>B.</td>
<td>6</td>
<td>Diarrhoea with dehydration and acidosis.</td>
<td>Pneumonia (early); 'Healed rickets' (?)</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>L. T. 468</td>
<td>F.</td>
<td>B.</td>
<td>6</td>
<td>Pneumococcal meningitis.</td>
<td>Pneumococcal meningitis; Rickets.</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>A. W. 504</td>
<td>F.</td>
<td>W.</td>
<td>7</td>
<td>Otitis media; Pyelitis; Dehydration.</td>
<td>Otitis media; Pneumonia.</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>D. M. 50</td>
<td>M.</td>
<td>W.</td>
<td>6</td>
<td>Septicaemia; Pneumococcal pneumonia.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. W. 578</td>
<td>F.</td>
<td>B.</td>
<td>10</td>
<td>Meningitis, pneumococcus; Rickets.</td>
<td>Meningitis, pneumococcus; Pneumonia; Rickets.</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>F. R. 581</td>
<td>F.</td>
<td>B.</td>
<td>23</td>
<td>Pneumonia</td>
<td>Pneumonia</td>
<td>Doubtful</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>A. B.**</td>
<td>M.</td>
<td>B.</td>
<td>7</td>
<td>Sinusitis</td>
<td>Scurvy</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>L. T. 869</td>
<td>F.</td>
<td>B.</td>
<td>4½</td>
<td>Meningitis; Meningococcus.</td>
<td>Meningitis</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

**This child had most extreme scurvy which was not recognized in the out-patient departments of the Harriet Lane Home, the clinic of the Wilmer Ophthalmological Institute, and of the Nose and Throat Department. The diagnosis of sinusitis was made in the Wilmer Clinic. In reality a subperiosteal haemorrhage in the roof of the orbit was present.
that rickets was not there. The difficulty encountered was that scurvy, if at all advanced, deprives the trabeculae of their osteoid envelopments and so takes away one of the chief means of recognizing rickets. It became necessary, therefore, to rely on the appearance of the cartilage for evidence, and in a number of cases there was doubt whether the changes there were due to rickets or to scurvy. The discussion of the relationship of rickets and scurvy in the present cases and in general is deferred until further studies have been made.

One hundred and twenty-five cases studied clinically*.

As indicated at the beginning of this article, scattered clinical observations were made on 125 cases of scurvy during the course of the x-ray studies. The results of these observations will be considered briefly.

Age at which scurvy became manifest. In the group of 125 cases, the age at which scurvy became manifest ranged from four months to five years. The disease was rare before the age of six months (see chart I)

**CHART I.**

Age incidence in 125 cases of scurvy. The solid blocks represent the number of full term babies in whom the disease became manifest at the ages indicated; the cross-hatched blocks represent the number of premature babies with scurvy at the same age period.

* This represents the total number of cases of scurvy among 35,000 consecutive admissions to the dispensary and wards of the Harriet Lane Home over a period of eight years.
and the majority of cases (59 per cent.) occurred between seven and nine months of age. This is in accord with the clinical observations of other authors. It is of passing interest that a twenty-seven months old child, who was mentally defective, had had a previous attack, of equal severity, at the age of six months. This was the only child in the series who suffered from two attacks, and both of these are incorporated in the chart. The five-year-old child, however, was, also, mentally deficient.

**Diet and other predisposing factors.** All of the infants in this group of cases were artificially fed. Most of them were receiving pasteurized milk which was usually boiled in addition. Four were fed on evaporated or condensed milk. Few had begun to take solid food. No case of scurvy in a breast-fed infant has been encountered although a few apparently authentic cases have been reported; in these the scurvy became clinically manifest so soon after birth that they may better be regarded as instances of congenital scurvy. In this connection it is perhaps of interest that one instance of undoubted congenital scurvy has been discovered in the autopsy material examined here. The case is to be reported.

Other factors of minor importance include (1) race, (2) prematurity, (3) twin births, and (4) infection. Of the 125 cases, 75 per cent. occurred in white infants. This is in contrast to the racial incidence of rickets and tetany, which in Baltimore predominates in the coloured child. Inasmuch as the ratio of blacks to whites in the dispensary population at large is approximately half and half, this percentage may be significant. It is possible that more of the coloured infants are at least partially breast fed, or that greater carelessness in feeding results in the accidental introduction of larger amounts of the anti-scorbutic factor. It is also possible that there is a real difference in racial susceptibility. Adequate data for the determination of these points were not available.

There were seventeen premature babies in this group, representing an incidence of 13.6 per cent. Without available statistics as to the exact incidence of prematurity among the total admissions to the Harriet Lane Home, it is impossible to estimate the significance of this figure. It seems, however, as though it represented a fairly high percentage. The premature babies did not develop the disease at a younger age than the full-term babies (see chart 1).

Twin births in this series numbered seven. In three instances, only one of the pair was affected. This suggests a difference in individual susceptibility. In one instance, however, the other twin had died six weeks previously of pneumonia and was reported to have had some tenderness of the legs at the time; in another, the more vigorous twin had occasionally received orange juice and vegetables while the scorbutic twin had con-
sistently refused them; in the third instance, one twin had been receiving orange juice for a week, when scurvy developed in the other from whom the orange juice had been withheld on account of diarrhoea. It is possible that the twin who escaped scurvy did so only because the anti-scorbutic regime was started just in time to stave off manifest symptoms of the disease. With regard to the other two pairs, both twins were affected simultaneously in one instance; in the other instance, one twin developed the disease two months earlier than the other. It was stated that both of these were receiving the breast in addition to artificial feedings until two-and-a-half months before scurvy developed in the first twin. This again suggests a difference in individual susceptibility, but it seems more probable that most of the breast milk, during the period of mixed feeding, was taken by the twin who was temporarily spared. In the case of twins, an economic factor may contribute to the development of scurvy, since the cost of anti-scorbutic substances for two is naturally twice that for one.

The importance of infection as an etiological factor in the production of scurvy cannot be estimated from this study, although in several instances, it seemed to bear some relationship to the onset of symptoms, at least as a precipitating factor.

**Time required for the development of scurvy.** It was not possible to determine this in all cases, because of the inaccuracy of the records, since mothers, having previously been advised to give orange juice, often conveyed the impression that they were doing so, quite irrespective of the truth, while the examiners just as frequently failed to check their veracity in spite of obvious discrepancies. In 37 of the cases, however, the time required for the development of manifest scurvy could be estimated with a fair degree of accuracy as there was exact information regarding the period of breast feeding and the use of anti-scorbutic substances. Only one of these patients had received any orange juice at all, and with him it had been discontinued for a known length of time. In these cases, then, the time required for the development of scurvy was computed by subtracting from the age at which the scurvy was diagnosed the duration of symptoms and the period of breast feeding. On the basis of these calculations, the time required varied from two to nine months. There was only one case, however, in which it developed in as short a time as two months. In the majority of instances (55\% per cent.) it required from six to seven months (chart IIb). The age incidence of these cases is indicated in chart IIa and they are tabulated in detail in table 2.

In the older literature on adult scurvy, it is found that in sailors the disease frequently developed in from four to six weeks and rarely required
more than two months. Lind', in his 'Treatise on Scurvy,' described two cruises in which 'the scurvy began to rage after being a month or six weeks at sea' and another in which 'after leaving the coast of Mexico, in less than seven weeks at sea, the scurvy became highly epidemic.' One is immediately confronted with the problem as to why so much more time is required in the infant than in the adult. Although some authors believe that the infant under five months of age is able to synthesize the anti-scrobutic vitamin it seems more logical to explain the difference on variations in the degree of deprivation. The diet on which the sailor developed scurvy in the eighteenth century was often completely lacking in vitamin C, whereas, in the milk that is fed the infants of to-day, traces probably remain, even though most of it is destroyed by pasteurization.

**CHART II.**

![Chart](image)

a. Age incidence of 37 cases of scurvy in which the time required for the development of the disease was known. (There was one premature baby in the seven months' group.)

b. Time required for the development of scurvy in 37 cases. (One that developed in two-and-three-quarter months is charted as three months; one in four-and-three-quarter months is charted as five.)

**Seasonal incidence.** The question of seasonal incidence was investigated on the chance that it might throw further light on the etiological factors concerned in the development of scurvy. Analysis of the entire group of 125 cases showed that the greatest number for any single month occurred in September (21 per cent.), while more than a third of the total number occurred in September and October together. A more detailed analysis was made of the above 37 cases, in which the duration of conditions conducive to scurvy was known, since in them it could be shown over what period the scurvy was developing as well as the time at which it became manifest. The results of this analysis are presented graphically in chart III. Here again the greatest number of cases occurred in September (22 per cent.), while 40 per cent. occurred in September and October together. It is clear from the chart, that in Baltimore, it is during the summer months that scurvy is generally 'brewing' with a sudden peak in the incidence of manifest cases in the early fall. This is in direct contrast to the seasonal incidence of rickets, and suggests that season as such is not directly responsible. Indirectly it may affect the
## Table 2.

**Thirty-seven cases in which the time required for the development of the disease was known.**

<table>
<thead>
<tr>
<th>Sex &amp; Age</th>
<th>Weight</th>
<th>Ht.</th>
<th>Time of breast feeding</th>
<th>Symptoms and Duration</th>
<th>H/T***</th>
<th>Month of onset ***</th>
<th>Time required for scurvy to develop</th>
<th>X-ray changes</th>
<th>Ca</th>
<th>P mgm. per cent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>M—W 7</td>
<td>27</td>
<td>14</td>
<td>0</td>
<td>Pain knees flexed 4 wk.</td>
<td>+/4</td>
<td>Sept.</td>
<td>6 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—C 7½</td>
<td>14</td>
<td>14½</td>
<td>0</td>
<td>Cried when handled 3 wk.</td>
<td>+/4</td>
<td>July</td>
<td>7 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—C 7</td>
<td>19</td>
<td>1</td>
<td>0</td>
<td>Pain, rt. leg 3 wk.; left leg 1 wk.</td>
<td>O/O</td>
<td>Nov.</td>
<td>6 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W 9</td>
<td>1 mth.</td>
<td>One foot tender 1 mth.</td>
<td>Cried when knees moved 1 wk.</td>
<td>O/O</td>
<td>Feb.</td>
<td>7½ mth.</td>
<td>Slight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W 8</td>
<td>13</td>
<td>20</td>
<td>0</td>
<td>Cried when legs moved 3 mth.</td>
<td>O/O</td>
<td>May</td>
<td>5 mth.</td>
<td>Long standing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W 8½</td>
<td>19½</td>
<td>4 d.</td>
<td>3 wk.</td>
<td>Gums bruised 5 wk. Fretful 3 wk. Arms and legs tender 10 d.</td>
<td>+/4</td>
<td>Sept.</td>
<td>8 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W 8</td>
<td>18½</td>
<td>3 wk.</td>
<td>Cried when touched 4 d.</td>
<td>+/5</td>
<td>Sept.</td>
<td>6 mth.</td>
<td>'Typical'</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W 10½</td>
<td>18½</td>
<td>24</td>
<td>1 mth.</td>
<td>Disliked movement left leg 2 d.</td>
<td>+/5</td>
<td>Mar.</td>
<td>9 mth.</td>
<td>Marked (four plates)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—C 7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Disliked movement left leg 2 d.</td>
<td>--</td>
<td>Oct.</td>
<td>7 mth.</td>
<td>Definite</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W 9½</td>
<td>13½</td>
<td>28</td>
<td>2 wk.</td>
<td>Diarrhoea; pain in legs 3 wk.</td>
<td>+/2</td>
<td>Aug.</td>
<td>8 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W 7½</td>
<td>13½</td>
<td>27½</td>
<td>0</td>
<td>Legs drawn up 6 d.</td>
<td>+/8</td>
<td>Sept.</td>
<td>7½ mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W 8½</td>
<td>17½</td>
<td>0</td>
<td>0</td>
<td>Pain, right leg 3 d.</td>
<td>+/½</td>
<td>Aug.</td>
<td>8½ mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W 5½</td>
<td>10½</td>
<td>3 mth.</td>
<td>Cried when handled 10 d.</td>
<td>Retrobulbar haemorrhage. Occult blood in stools.</td>
<td>O/O</td>
<td>July</td>
<td>2 mth.</td>
<td>Marked 'at least 2 mth.'</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W 8½</td>
<td>17½</td>
<td>2 wk.</td>
<td>Screamed if legs touched 3 d.</td>
<td>O/2</td>
<td>June</td>
<td>8 mth.</td>
<td>Long standing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—C 9½</td>
<td>0</td>
<td>Vomiting, irritable, legs flexed 3 wk. Bl. nose and stool. Petechiae on abd.</td>
<td>+/2</td>
<td>Apr.</td>
<td>9 mth.</td>
<td>Definite</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = premature

---

**Notes:**

- **Symptoms followed trauma**
- **H = Haemorrhages into gums**
- **T = Teeth**
- **Ca = Calcium**
- **P = Phosphorus**
- **mgm. per cent.**

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TABLE 2.—Continued.

<table>
<thead>
<tr>
<th>Sex &amp; Race</th>
<th>Age in months</th>
<th>Weight lb.</th>
<th>Ht. in.</th>
<th>Time of breast feeding</th>
<th>Symptoms and Duration</th>
<th>H/T**</th>
<th>Month of onset ***</th>
<th>Time required for scurvy to develop</th>
<th>X-ray changes</th>
<th>Ca</th>
<th>P mgm. per cent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>F—C</td>
<td>7</td>
<td>13½</td>
<td>—</td>
<td>0</td>
<td>Pain in legs 3 d.</td>
<td>O/O</td>
<td>May 7 mth.</td>
<td>Marked. + Rickets</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W</td>
<td>7½</td>
<td>15½</td>
<td>—</td>
<td>0</td>
<td>Swelling rt. knee &amp; ankle 2 d.</td>
<td>+/+</td>
<td>Jan. 7½ mth.</td>
<td>Definite</td>
<td></td>
<td>9.5</td>
<td>4.8</td>
</tr>
<tr>
<td>F—C</td>
<td>13</td>
<td>12</td>
<td>25</td>
<td>0</td>
<td>Screamed when touched 2 mths.</td>
<td>O/O</td>
<td>July 4 mth.</td>
<td>Marked</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W</td>
<td>8</td>
<td>13</td>
<td>—</td>
<td>2 wk.</td>
<td>Sickly 1 mo. (Early changes in X-ray then) Bl. gums later</td>
<td>—</td>
<td>Sept. 6½ mth.</td>
<td>Well marked</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>9½</td>
<td>18</td>
<td>—</td>
<td>2 wk.</td>
<td>Pain 2 mth.</td>
<td>+/2</td>
<td>Nov. 7 mth.</td>
<td>Advanced</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—C</td>
<td>7½</td>
<td>13½</td>
<td>—</td>
<td>3 mth.</td>
<td>Cried when picked up &amp; arms raised 2 wk. Pain in legs later</td>
<td>+/1</td>
<td>May 4½ mth.</td>
<td>Definite. + Marked Rickets</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>8</td>
<td>15½</td>
<td>—</td>
<td>0</td>
<td>Screamed when legs touched 1 wk.</td>
<td>+/+</td>
<td>Nov. 7½ mth.</td>
<td>Early</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—C</td>
<td>6</td>
<td>13½</td>
<td>—</td>
<td>0</td>
<td>Pain rt. leg 5 d.</td>
<td>—</td>
<td>July 6 mth.</td>
<td>Relatively early</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>7½</td>
<td>9</td>
<td>22</td>
<td>0</td>
<td>Cried when touched or moved 3 wk.</td>
<td>O/1</td>
<td>Oct. 6½ mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>10</td>
<td>13½</td>
<td>—</td>
<td>7 wk.</td>
<td>Rt. leg sore 3 wk.; then left</td>
<td>O/O</td>
<td>Oct. 7½ mth.</td>
<td>Definite</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>10½</td>
<td>12½</td>
<td>25½</td>
<td>few d.</td>
<td>Pain in legs 1 mth.; later in shoulders</td>
<td>O/O</td>
<td>Feb. 9½ mth.</td>
<td>Marked</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>7</td>
<td>19½</td>
<td>—</td>
<td>0</td>
<td>Cried when moved 10 d.</td>
<td>—</td>
<td>Sept. 6½ mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>8</td>
<td>11½</td>
<td>—</td>
<td>15 d.</td>
<td>Did not want to be touched 3 d.</td>
<td>+/+</td>
<td>Feb. 7½ mth.</td>
<td>Moderately developed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F—W</td>
<td>12</td>
<td>18½</td>
<td>29</td>
<td>5 mth.</td>
<td>Cried when legs moved 1 mth.</td>
<td>+/6</td>
<td>Oct. 6 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M—W</td>
<td>7½</td>
<td>14½</td>
<td>—</td>
<td>1 mth.</td>
<td>Cried when legs moved 1 wk.</td>
<td>O/O</td>
<td>Sept. 6 mth.</td>
<td>Well developed</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Symptoms followed trauma  
*** Onset of manifest symptoms  
** H = Haemorrhages into gums  
T = Teeth
situation in two ways, through (1) the cost of oranges, which is the source of vitamin C commonly employed in Baltimore, and (2) the frequency of diarrhoeal diseases in hot weather. Because the cost of oranges is higher during the summer months, which is not the season for citrus fruits, it proves inhibitory to some and oranges are consequently omitted from the diet while no substitution is made for them (as by tomato juice). In other instances, the occurrence of diarrhoea may lead to the postponement or discontinuation of the use of orange or tomato.

**CHART III.**

Seasonal incidence in 37 cases of scurvy. The black dots indicate cases of manifest scurvy occurring during the different months of the year. The arc leading to each dot indicates the period during which conditions conducive to scurvy existed, in other words, the months during which no antiscorbutic was received.
THE RECOGNITION OF SCURVY

juice, which, through oversight, it is not started or resumed when the intestinal disturbance is over. It is possible, also, that diarrhoea interferes with the absorption of vitamin C when it is supplied.

First symptoms and signs. 'Any way you lay him is the way he lays. If you lay him on his side, he makes no effort to move or nothing. It looks like most of his trouble is from the hips down.' That, in the words of a mother, typifies the clinical picture of scurvy! As a general rule, the symptoms of scurvy appear abruptly. Pain and tenderness of the extremities, or symptoms referable to pain, such as disinclination to move, crying when handled, drawing up of the legs, 'rheumatism,' gave the first evidence of the presence of the disease in 115 (92 per cent.) of the cases, and was ultimately present in all but four. Over and over again the uniformity and prominence of this complaint made a strong impression. The pain was occasionally (twelve cases) initiated by trauma and was usually accompanied by irritability and fretfulness. In ten cases, there was also enough swelling to attract the mother's attention and to figure in the complaint offered by her. In one instance fretfulness was the only symptom mentioned. Haemorrhages into the gums furnished the initial complaint in only five instances, although they were present in forty-six. They were not present in any instance in which there were no teeth, and in 20 per cent. of the infants with teeth they were also lacking, even though scorbutic lesions were well marked in the skeleton. It can be stated with certainty on the basis of this study that when haemorrhages are found in the gums, the signs of scurvy in the skeleton will be well developed. In one case, the appearance of blood in the stools antedated the onset of pain by a week. Its presence was noted in two other instances. Haematuria, usually microscopic, was occasionally found. Epistaxis occurred in three instances, but not as the initial complaint. Petechial haemorrhages were extremely infrequent and retrobulbar haemorrhage occurred only once.

There was a complete lack of parallelism between the duration of symptoms and the apparent duration of the disease process as evidenced by the x-ray pictures. Well-developed scurvy was frequently present in the skeleton when there had been symptoms for only a few days. In one case, in which there were as yet no symptoms, but in which well-marked scurvy was discovered by x-ray, reference to a set of x-rays taken four-and-a-half months previously indicated that it was already present at that time!

Since, then, there is this long latent period during which scurvy may be manifest in the skeleton without recognizable clinical symptoms, attention is attracted to the possibility that less specific prodromal symptoms may precede the others. Many authors describe, as premonitory signs of scurvy, such symptoms as anaemia, disinclination to eat, failure to gain weight or to grow in length. Anaemia was not consistently present in this group of cases, even when there was evidence that the scurvy must have existed for some time. Loss of appetite was mentioned rarely, and then only as an accompaniment of the other manifest symptoms,
Undernutrition was also an inconstant finding. A third of the infants, irrespective of the apparent duration of the scurvy, were of normal weight or above. Slight undernutrition characterized another third, while the remaining third showed marked degrees of undernutrition. There was no real basis for attributing this directly to the scurvy. The length was recorded in only twenty-four instances. Although half of these infants were undersized, this number included seven premature babies, so that this data cannot be regarded as significant. In a few instances it was noted that the baby had 'looked badly' or 'seemed sickly' for a few weeks before the pain or disinclination to move was apparent. Definite fretfulness and irritability, though frequently mentioned among the early symptoms, rarely preceded the notice of pain, and were undoubtedly attributable rather than antecedent to it. One child, who was being seen regularly in the out-patient department during the four-month period when scurvy (in retrospect) is known to have been present, was gaining weight steadily and at each visit it was noted that 'feedings are well taken.' It would appear from this study that scurvy, like latent tetany, may, and usually does, from the symptomatic standpoint become suddenly manifest. No explanation can be offered for this sudden appearance of symptoms, excepting in those few cases in which trauma seems to have produced them, probably by precipitating subperiosteal haemorrhage or epiphysial separation at sites already prepared for them. The suddenness of the disappearance of symptoms under treatment has always been mysterious. Marked improvement, in every instance, was evident within a day or two after the institution of treatment.

In conclusion, it may be remarked that there are no early signs of scurvy. The slight x-ray changes described in this paper enable the diagnosis to be made by x-ray examination of the skeleton somewhat earlier than previously. The scorbutic deformity of the rib is useful as a means of recognition of the disease when well developed. The chief reliance, however, must still be on the symptoms and of these, pain and tenderness in the legs are the most important. It is possible that, in the future, more careful questioning of the more observant mothers will give some indication of more definite premonitory signs. Every infant, known not to have received anti-scorbutic substances over a period of two months, should be held under suspicion. It must be hoped that the numerous studies now in progress in various parts of the world on the metabolism of ascorbic acid will reveal some simple way for the early recognition of the disease.

REFERENCES.
Fig. 1.—For explanation see text.
Fig. 2.—For explanation see text.
Fig. 4.—For explanation see text.

Fig. 5.—A.S., 559. X-ray picture of a series of ribs. The ribs show extreme rarification. The dense shadows at the ends are caused by the scorbutic lattice.
Fig. 6.—A. S., 559. Microphotograph of one of the ribs in fig. 5. The cartilage can be seen above stained deep blue. At the bottom of the cartilage the scorbutic lattice shows beautifully as a hedge of deep blue-staining trabeculae of calcified matrix substance without any addition of bone. The trabeculae of bone are greatly rarefied and are greatly reduced in number and the cortices appear as thin shells. At the lower left hand corner a round haemorrhage can be seen. The cortex and the trabeculae of bone stain pink in contrast to the lattice which stands deep blue. The preparation gives a beautiful demonstration of the differentiation of calcified matrix substance from bone by staining methods. All across the lower part of the lattice one sees fractures. Some of the trabeculae of bone are fractured also.
Fig. 7.—A.S., 559. Microphotograph (higher power view) of scorbutic lattice of fig. 6. Above is the proliferative cartilage. The lattice stains a deep blue and is quite devoid of any covering of bone. Bone, if present, would appear pink (grey in photograph) and would have a cellular structure. The interstices of the lattice are occupied by connective tissues.
Fig. 8.—A. S. 559. Microphotograph (higher power view) of the scorbutic lattice of fig. 6. The fractures of the lattice are beautifully shown. One can see that the line of fracture has occurred at the junction of the lattice with the trabecular system of the shaft, because below the fracture one can identify trabeculae of bone by their cellular structure whereas above the line of fracture the dark staining lattice shows no cells. Along the line of fracture are bands of fibrin. Below is the typical connective tissue framework of the marrow.

Fig. 9.—G. H., 449. X-ray picture of a series of ribs, which show the great cupping so characteristic in the rib in advanced scurvy. The dark shadows at the sides of the cup are produced by the scorbutic lattice. Below the lattice shadows in the three ribs on the right the zones of rarefaction can be beautifully seen. The microscopic preparation, shown in fig. 10, was taken from the third rib from the right. Rickets as well as scurvy was present in this case.
Fig. 10.—G.H. 449. Microphotograph taken from fig. 9, third rib from right. Above is the cartilage. Next below is the scorbutic lattice which stains black and has a dense structure. Below the lattice is the zone of rarefaction. In this zone the trabeculae are sparse and extremely thin. The marrow cells have gone, leaving the connective tissue framework, which appears white, exposed to view. Still lower down the trabeculae of bone, which appear pink (grey in the photograph) look fairly normal. In the lower corner of the preparation the marrow cells are present and their situation can be identified by their dark blue stain. The cortices are extremely thin and at the upper boundaries of the rarefaction zone are shell-like and fragmentary. Fractures have occurred through the lattice at many points as is indicated by fragments which lie cross-wise or at queer angles.
Fig. 11.—A.P., 1135. Microphotograph from the narrow cavity to show fresh haemorrhages and, also, the characteristic scorbatic changes in the marrow. The haemorrhages obviously occurred around the branches of the small vessels, which can be indistinctly seen in the microphotograph. The red blood cells were ensheathed around the branches. The marrow cells have largely gone but not entirely, as sprinklings can be seen scattered about in the connective tissue. The presence of the loose embryonic-like connective tissue between the trabeculae instead of the ordinary obscuring masses of marrow cells is characteristic of scurvy histologically.
Fig. 12.—A.P., 1135. Microphotograph from the upper end of the humerus showing comminution of the lattice. At the top of the microphotograph is the cartilage, immediately below is the lattice which has been reduced to fragments. At the bottom one sees bone, the cells of which are not visible, because out of focus. Everywhere between the trabeculae is the typical scorbutic connective tissue.
Fig. 13.—M.B. 24.—Microphotograph (high power view) of rib shown in fig. 38, in order to illustrate fracture of the lattice. The fractures and the masses of fibrin which run between the fragments can be beautifully seen. The two large black fragments in the centre of the picture have been impacted and are disintegrating in their interiors. Around the fragments one sees many connective tissue-like cells which are osteoblasts. Large osteoclasts can also be seen. The marrow cells are gone.
FIG. 14.—M.B., 24. Microphotograph from rib of fig. 36 showing the large numbers of osteoblasts which surround the fractured fragments of lattice. In the text it was stated that the osteoblasts surrounded them, like 'swarms of bees'. The fragments lie in typical scorbutic connective tissue, the 'Gerüstmark' of the Germans.
Fig. 15.—M.B., 24. Microphotograph from the lower end of the tibia, showing a beautiful 'corner' lesion of rarefaction. The cortex has been absorbed in large part; only fragments remain. The adjacent trabecular framework has also been largely absorbed. The scorbutic lesions seem to be limited to the corner. In reality, however, minute fractures of the finer structure were found with the microscope in the underparts of the lattice at a number of points.
Fig. 16.—M.B., 24. Microphotograph (higher power view) of corner shown in fig. 15. The trabeculae have disappeared from the central area but their debris can still be identified by the groups of osteoblasts which surround them. The cortical shell on the left is undergoing destruction. The soft parts show no sign of injury. It is obvious, however, that the trabeculae and cortex are disintegrating and disappearing.
Fig. 17.—M. B., 24. X-ray picture of the lower ends of the radius and ulna. The histological preparations in the microphotographs in figs. 18 and 19 were taken from the radius. There is a minute spot of rarefaction at the outer corner of the radius which is too small to show clearly in the x-ray film. The outer corner in the film has merely an indistinct outline.

Fig. 18.—M. B., 24. Microphotograph from radius in fig. 17. The low power microphotograph shows quite beautifully the spot of rarefaction at the outer corner of the radius on the left-hand side. The cortex has been partially absorbed as have, also, some of the adjacent trabeculae. A similar area of scurvy rarefaction can be seen in the opposite corner. At scattered points in the under surface of the lattice fractures were found which were too small to show in this view.
FIG. 19.—M. B., 24. A high power microphotograph showing the outer corner of the radius exhibited in fig. 17 and 18. The cortex is disintegrating and has disappeared entirely in the lower part of this view. The fragment which is shown is filled with osteoclasts and shows a cystic formation below. Crowds of osteoblasts surround one of the adjacent trabeculae, which is undergoing destruction.
**Fig. 20.**—M. B., 24. X-ray picture of the upper end of the humerus. A glance shows the spot of rarefaction in the characteristic situation at the outer angle.

**Fig. 21.**—M. B., 24. Microphotograph from the outer angle of the upper end of the humerus shown in fig. 20. It can at once be seen that the corner is most abnormal. The cortex is largely gone and that which remains has been fractured and is being destroyed. The adjacent parts of the lattice have also been fractured and are being removed. Swarms of osteoblasts surround the fragments. It is interesting to see that bone tissue has formed considerably to the outer side and above the proliferative cartilage.
Fig. 22.—M. B., 24. X-ray picture of lower end of femur. The inner corner, left in the figure, shows a characteristic spot of rarefaction.

Fig. 23.—M. B., 24. Microphotograph of inner corner of lower end of femur from fig. 22, showing extensive destruction of the cortex and adjacent lattice. A lattice has started to form at the side of the proliferative cartilage, separated from the rest by a tongue of the latter which extends downwards almost to the periosteum. Rarefaction is rampant in this island of lattice separated by the tongue. If this island of lattice were only larger, it might give rise in the x-ray picture to the appearance of 'over-extension and pointing' of the corner of the end of the shaft.
Fig. 24.—A. P., 1135. X-ray picture of the lower ends of the tibia and fibula, showing lattice formation, cupping and also 'bagging' of the outer corner (right in figure).

Fig. 25.—A. P., 1135. Microphotograph of the outer corner of the lower end of the tibia exhibited in fig 24. The microphotograph gives a good example of 'bagging' and also a most typical scorbutic affection of the bone. The cortex has disappeared except for scattered fragments. Extensive fractures of the lattice have taken place. As the result of the loss of cortical support the 'corner' soft tissues have been squeezed outwards. The periosteum remains and forms the outer boundary of the 'bag.' This microphotograph makes clear why the bagged area should appear so rarefied in the x-ray film and devoid of cortical shadow, and why it should extend out beyond and envelope the end of the lattice.
Fig. 26.—A. P., 1135. X-ray picture of the lower end of the femur, showing a scorbutic lattice, zone of rarefaction, "bagging," over-extension, and pointing at both corners.

Fig. 27.—A. P., 1135. Microphotograph of the outer corner (right in fig.) of the lower end of femur (fig. 25). This microphotograph really needs no explanation. The lattice has been completely shattered, the cortex has gone. Fragments of the lattice extend to the extreme limit of the proliferative cartilage and considerably to the outer side of the latter.
Fig. 28.—A. P., 1135. X-ray picture of the upper end of the humerus. The outer corner shows a minute spot of rarefaction.

Fig. 29.—A. P., 1135. Microphotograph of outer corner of upper end of humerus as shown in fig. 28, showing a characteristic scorbutic lesion. The heavy lattice has been shattered and masses of osteoblasts surround the fragments. The cortex has been partially destroyed and areas of rarefaction are developing in the interior.
FIG. 30.—A. P., 1135. X-ray picture of upper end of tibia and fibula. The inner corner of the tibia has been injured at autopsy. The outer corner shows a minute spot of rarefaction.

FIG. 31.—A. P., 1135. Microphotograph of outer corner of upper end of tibia, as shown in fig. 30. The microphotograph illustrates beautifully the fractures of the lattice with a splinter lying at right angles, also loss of cortex beneath the lattice just at the corner. The loss of the cortex just under the outer end of the lattice might give rise to the appearance in the x-ray film of 'over-extension' of the corner and 'pointing.' Extensive rarefaction has taken place in the framework of bone.
FIG. 32.—A. P., 1135. X-ray picture of the upper end of the ulna. This fails to give any indication of scurvy. Nevertheless, the histological preparation from the posterior angle of the upper end of the olecranon shows a scorbutic lesion.

FIG. 33.—A. P., 1135. Microphotograph of posterior corner of upper end of the olecranon as shown in fig. 32. Fractures of the lattice have occurred, presumably from pressure of the elbow against the bed. The fragments are in process of absorption. The contrast between the bone with its cells and the lattice substance without cells and coloured deep blue is striking.
Fig. 35.—A. B., 1183. This is the only example of true 'bayonet deformity' encountered. The drawing has been taken from the ninth rib. This rib and the tenth alone showed the bayonet deformity. The ribs above showed the ordinary scorbutic deformity. The rib has been cut transversely so that the upper contour corresponds to the outer surface of the costo-chondral junction. Examination of the drawing shows that the resting cartilage has been pulled inwards on the proliferative cartilage, so that the outer part of the latter overlaps the former and sticks out above it forming a ledge. The examining finger passing along the outer surface of such a rib, would encounter a ledge and drop down on the cartilage. But the ledge is formed not by the end of the bone but by the proliferative cartilage. The bayonet deformity is most uncommon in rickets and is almost a misnomer. In this case advanced rickets was present. It is suspected that the bayonet deformity is always a combination of scurvy and rickets and occurs only in the lower true ribs and is caused by the pull of the diaphragm which is attached to the resting cartilage.
Fig. 36.—M. B., 24. Microphotograph of a rib, cut in a transverse direction to show the shift inwards of the cartilage on the shaft. The outer border of the costo-chondral junction is to the right. It is seen at once that a fracture has taken place through the under surface of the lattice and extends entirely across the bone. The lattice fragments have been impacted in some places and in others have been entirely absorbed. Through this study of the finer structure, in particular the blood vessels and strands of fibrin and the fragments of trabeculae one can be certain that the cartilage with its attached lattice has shifted slightly in an inwards direction on the broken end of the shaft. One can also be certain that the cartilage has been bent inwards slightly on the shaft so that the angle formed by the two is slightly greater than normal. On the left-hand side in the line of fracture is seen an area of rarefaction. Above this area is unbroken lattice. From this sample of unbroken lattice it could be imagined what would be the appearance of the lattice if fracture had never occurred. The development of the shaft around the sides of the proliferative cartilage is beautifully shown.
Fig. 37.—Camera lucida drawing of transverse sections of a typical scorbatic and a rachitic rib. The upper borders correspond to the outer surfaces of the costo-chondral junctions. The drawings speak for themselves. In rickets the sharp angle of junction between cartilage and shaft is not apt to be present because of the deep rachitic intermediate zone interposed between the proliferative cartilage and the shaft.