THE BRADSHAW LECTURE
ON
INFANTILE SCURVY AND ITS RELATION TO RICKETS.

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Mr. President,—Eleven years ago I submitted to the consideration of members of our profession the results of an analysis of thirty-one cases of what I believed to be infantile scurvy. Such cases, though scantily reported in England, were well known in German medical literature, and described under the title of "Acute Rickets." So far back as 1873 Dr Ingerslev, a Swedish physician, had suggested the scorbutic nature of one case under his care. But scurvy had been dismissed by the German school, amongst other reasons because no sponginess of gums was to be found in many of the cases. In 1881 Dr. Gee had given a brief but graphic description of five cases, under the title of osteal or periosteal cachexia. He insisted that the obscure symptoms referable to the bones and the cachexia were not explicable by either rickets or congenital syphilis. But in 1878 three cases were fully described by Dr. Cheadle, in which definite sponginess of gums occurred, and likewise obscure symptoms referred to the lower limbs. These cases he claimed on clinical grounds to be true scurvy. In 1879 and in 1882 Dr. Cheadle published further examples, and he suggested that the disease could be grafted on a rickety stock. Dr. Cheadle's papers laid down the original lines of the true interpretation of the disease which we have to consider, and I do not think that anything has invalidated the soundness of his conclusions. There had been a singular paucity of pathological evidence on this subject, and I was able, as the result of three *post-mortem* examinations, to formulate the anatomical substratum, which appeared to explain many of the symptoms found. I propose to review the main clinical and anatomical features of the group, to give the conclusions deduced eleven years ago, and then to state briefly how far these have been modified by subsequent investigation and criticism.

The group of symptoms to which attention is called has generally a rather sudden onset beginning at any period after the age of 4 months, but predominantly in what may be called the second period of infancy, that is to say, from the age of 9 months to that of 18 months. The child is generally rickety, but fairly clothed so far as subcutaneous fat is concerned. There may have been antecedent digestive troubles, or, on the other hand, in respect to obvious symptoms little to show. But a careful observer will have noticed a certain amount of pallor. Suddenly, without obvious cause, the child becomes fretful in a special way. So long as it is left alone it is tolerably quiet. The lower limbs are kept drawn up and still. But when placed in its bath or otherwise moved there is continuous crying, and it soon becomes clear that the pain is connected with the lower limbs. At this period the upper limbs may be touched with impunity, but any attempt to move the legs or thighs gives rise to screams. Next, some obscure swelling may be detected; first on one lower limb, then on the other, though it is not absolutely symmetrical. The skin still remains pale, and there is no local heat, and no pitting. The swelling is ill-defined, but is suggestive of thickening round the shafts of the bones, beginning above the epiphysial junctions. Gradually the bulk of the limbs affected becomes visibly increased. No fluctuation can be obtained, but a little œdema may now appear on the feet. The position of the limbs becomes somewhat different from what it was at the outset. Instead of being flexed they lie everted and immobile, in a state of pseudo-paralysis. The knee-jerks may be obtained, and likewise the plantar reflexes. About this time, if not before, great weakness of the back becomes manifest. A little swelling of one or both scapulæ may appear, and the upper limbs may show changes. These are rarely so considerable as the alterations in the lower limbs. There may be swelling above the wrists, extending for a short distance up the forearm, and some swelling in the neighbourhood of the epiphyses of the humerus. There is symmetry of lesions, but it is not absolute; and the limb affection is generally consecutive, though the involvement of one limb follows very close upon another. The joints are free. In severe cases another symptom may now be found—namely, crepitus in the regions adjacent to the junctions of the shafts with the epiphyses. The upper and lower extremities of the femur, and the upper extremity of the tibia, are the common sites of such fractures; but the upper end of the humerus may also be so affected. Rarely a fracture occurs in the femur, at some distance from the epiphyses. A very startling appearance may be observed at this period in the front of the chest. The sternum, with the adjacent costal cartilages and a small portion of the contiguous ribs, seems to have sunk bodily back, *en bloc*, as though it had been subjected to some violence which had fractured several ribs in front and driven them back. Occasionally thickenings of varying extent may be found on the exterior of the vault of the skull, or even on some of the bones of the face. There is great tenderness, but, as with other lesions, there is no local heat, and there is seldom change of colour. Here also must be mentioned a remarkable eye phenomenon. There develops a rather sudden proptosis of one eyeball, with puffiness and very slight staining of the upper lid. Within a day or two the other eye presents similar appearances, though they may be of less severity. The ocular conjunctiva may show a little ecchymosis, or may be quite free. With respect to the constitutional symptoms accompanying the above series of events the most important feature is the profound anæmia which is developed. Whatever there may have been at the onset, when once the limb affection has become pronounced the pallor becomes intensified. The anæmia is proportional to the amount of limb involvement. As the case proceeds, there is a certain earthy-coloured or sallow tint, which is noteworthy in severe cases, and when once this is established bruiselike ecchymoses may appear, and more rarely small purpuræ. Emaciation is not a marked feature, but asthenia is extreme and suggestive of muscular failure. The temperature is very erratic; it is often raised for a day or two, when successive limbs are involved, especially during the tense stage, but is rarely above 101° or 102°. At other times it may be normal or subnormal.

Let us now consider the digestive tract. The condition of the gums is very important. Defective observation on this point has led to frequent misconceptions as to the nature of the disease. Stated generally, the gum condition may be said to bear a definite relation to the number of teeth which have appeared. If several teeth have appeared, considerable sponginess of the gums may be manifest. Fleshy swellings form, which even project from the mouth, and give rise to bleeding and fœtor. But if only a few teeth have appeared, the sponginess may be slight though definite, forming a narrow, fleshy ridge round each tooth.

If no teeth have appeared the gums may be normal or may present small bluish extravasations over the sites of the oncoming teeth, The condition of the gums sometimes gives rise to trouble in feeding. Occasionally there is nose bleeding. As a rule the appetite is fair, sometimes it is ravenous. There is seldom vomiting or diarrhœa, but a few cases have been recorded of blood in small quantities being passed *per anum*. The urine often contains a trace of albumen and a little blood. It is sometimes scanty in amount, and coarse concretions of uric acid may be passed. In rare cases the spleen is a little enlarged. Except in the fatal cases, the heart and lungs give rise to no symptoms.

What is the course of this disease? If the cachexia is very profound, the supervention of bronchitis, pleuro-pneumonia, severe diarrhœa, or an intercurrent exanthem, may bring about a fatal issue. It is sometimes a relapsing disease, and may run a chronic course over many months; but the average duration, when uninfluenced by treatment, is from two to four months. The chief interest of the disease turns on the limb affection, and its progress I will follow with a little more detail. First with regard to the cases in which no frac-

tures are detected. By slow degrees the swelling subsides, and with it the tenderness lessens. There is considerable wasting of the muscles, and it becomes evident now that there is a sheath of firm resisting tissue round part of the bone shaft which has been chiefly affected. This is most commonly found round part of the femur, but may be traced round part of the tibia also. Such sheaths, although often found in both lower limbs, are not generally of equal extent on the two sides. It may take months before these osseous sheaths are absorbed. By degrees voluntary mobility is restored, and the child also begins to raise itself from a horizontal posture. In the cases where fractures occur near the epiphysial junction, there is satisfactory repair, with little or no deformity. The same may be stated with respect to the ribs. In the rarer event, when the fracture occurs in the middle of the shaft, there is often an extensive callus and some deformity. The anæmia, the hæmorrhages, the asthenia, the pain, subside *pari passu* with the clearing up of the limb affection. Thus far, on the clinical side; let us now consider what *post-mortem* investigation teaches with respect to this disease. In two of these children, aged respectively 23 months and 19 months at death, the appearances were practically identical with what was found in another case, aged 10 months, and what has been since found in other cases by subsequent observers.

The specimens [exhibited] show that the essential lesion is the subperiosteal blood extravasation, with its secondary consequences. Thus, around the shaft of the femur, there is an extensive sheath of blood clot, which intervenes between the periosteum and the shaft except for a short distance at the upper extremity. A fracture had occurred through the junction area, between the lower epiphysis and the shaft. The surfaces were rough, but not splintered. There was evidence of rickets in the loose spongioid ossification of this region, and also in the diffuse ossifying centre of the lower epiphysis. The periosteum was intensely vascular, but there was no lymph or pus. In the muscles of the thigh the superficial layers contained a certain amount of pale yellow blood serum, which to some extent macerated the muscular tissue. In the deeper layers there was extensive blood clot, pretty evenly disseminated. The periosteum of the right tibia was also thickened and vascular. There was blood extravasation between it and the shaft along the whole length. Fracture was present in the loose, imperfectly ossified region, connecting the shaft with the epiphysis. The trabecular structure in the interior of the bone broke down easily, and there was much hæmorrhage in the medulla of the shaft The ossifying centre of the epiphysis and the loose ossification in the junction area resembled what was found in the femur. There was extensive blood extravasation in the deeper layers of the muscles of the leg. The periosteum of the fibula was only slightly detached, but it was very vascular, and there was slight extravasation near the extremities. The bones of the other side were similarly affected, though not quite to an equal amount. The scapulæ were very interesting. They showed extensive blood extravasations on both dorsal and ventral surfaces. The dorsal extravasations associated with muscular extravasations had given rise to swellings, which had been obvious during life. On the ventral surface the upraised periosteum had deposited a thin, new layer of osseous material; in this respect resembling the bony sheaths which had been felt during life, in the middle stage of the recovering cases, on the shafts of the lower limb bones. The humerus in this case showed no subperiosteal effusion, but in addition to the evidences of spongioid ossification, showed extensive hæmorrhage into the medulla.

I may refer to the condition of the ribs in another case. The periosteum was found extensively detached, with much chocolate-coloured detritus of broken down blood clot between the periosteum and the rib. The ribs themselves were quite bare, and were broken away close to the junction region with the costal cartilages. On section of the ribs it was found that the bony material was a mere shell, and there was an extensive endosteal extravasation. The cranium showed thin areas of subperiosteal hæmorrhage in the neighbourhood of porous bone on both parietals. Extensive blood and serum extravasations were found in the thigh muscles, the muscles were wasted, and subperiosteal extravasations were found in connection with the innominate bone and the femora.

The other case showed changes almost identical with those which I have described. I need not further particularise them, but will only mention two points of interest. Although there was no fluid effusion into any joint, there was a minute extravasation into the synovial membrane of one hip-joint.

The specimens from these cases are on the table, and besides the subperiosteal extravasation the hæmorrhage into muscles and the fractures I ask your attention to the remarkable absorption of bony material in the centre of this detached shaft of a femur. A vertical section has been made and the blood clot removed, both from outside and inside the bone. It will be seen that the compact tissue is reduced to a mere shell, thus resembling the state of the ribs which I have described. Microscopical examination of these bones confirmed the naked-eye appearances. The periosteum was vascular and thickened, but without cellular infiltration. The bone presented extensive absorption of trabecular structure, showing large spaces with eroded margins, and at the junction regions characteristic rickety ossification.

The visceral changes were not very important. In one case there was blood-stained serous effusion in one pleura, with extensive petechiæ on the parietal pleura; in another there was a hæmorrhagic focus in one lung with a little lymph over it; in a third there was some splenic enlargement, with blood extravasation under the capsule, and with some also into the substance. In the light of these anatomical substrata, we are now able to explain many of the symptoms which I have described. The special characters of the lower limb swelling—namely, the deep thickening referred to the neighbourhood of the bone shaft, the solid effusion into the muscular masses, the exquisite tenderness, the pseudo-paralysis, the absence of local heat or redness, are all in harmony with the conditions found *post mortem*. The absence of any true inflammatory sign is in harmony with the general absence of persistent pyrexia. The occasional moderate elevation of temperature may well be explained by the tension set up by the blood effused beneath a rather resisting membrane. The anæmia, which becomes so profound after the onset, and which is increased after each fresh involvement of a limb or portion of a limb, is explained by the successive internal hæmorrhages, which are just as much an impoverishment as if the blood had been discharged from the body. The fractures are not difficult to understand when we consider not only the lack of support outside the shaft in consequence of the stripping up of periosteum, but also, in the advanced cases, the hæmorrhage into the centre of the shaft with the extensive absorption of trabecular structure. In no bones is this internal absorption, combined with extravasation outside and inside, carried to a greater degree than in the ribs, and this explains the extraordinary deformity of the chest to which I have referred, which deformity is almost pathognomonic of the disease in question. The irregular bony sheaths round the long bones, felt during the middle stage of recovery of the severe cases, is due to the osseous material which has been thrown out by the upraised periosteum, and is analogous to what is found round the base of a recovering cephalhæmatoma. I may refer once more to the figure showing the same process over the ventral surface of the scapula.

The extremely interesting eye changes which I have already described are explained thus: the periosteum which lines the under surface of the roof of the orbit is even in the normal condition very loosely attached to the bone. The proptosis which occurs in this disease is caused by a blood extravasation into this easily distended space between the roof of the orbit and its periosteum. The eyeball is pushed downwards and forwards, and the upper lid becomes tense and bulged, and sometimes a little discoloured. The swellings on the cranium are due to blood extravasations, especially in connection with portions of bone previously more vascular than normal, owing to pre-existing rickety deposit.

Thus far we are on safe ground, and there can be little difference of opinion as to the anatomical conditions found and their relation to the symptoms observed; but when we go a step further and inquire as to the true nature of this disease and its etiology, there is room for great divergence of opinion. First, as to the question of rickets. The doctrine held by many German authorities that these cases were

examples of acute rickets was formulated without adequate *post-mortem* evidence, and it was obviously unsatisfactory. Clinically it is true that, in the great majority, antecedent rickets is shown by beaded ribs, large epiphyses, and delayed teething. Two manifestations of rickets, namely, marked head sweating and laryngismus stridulus, are often present throughout the course of the disease now under consideration. Moreover, after the subsidence of the special group of symptoms which I have described, there are in a large number of cases persistent signs of rickets, which run the ordinary, typical, somewhat protracted stages of that malady. In the specimens pourtrayed there is evidence of a definite amount of rickety change in the spongioid ossification in the epiphysial junction region. But Sir William Jenner, to whom I showed these specimens, told me that in the very large number of cases of rickets investigated by him in years past, he had never seen any such condition as the hæmorrhages here present. Nor have I been able to find in any of the pathological accounts of rickets any record of such a condition. But although in these specimens there is a tangible amount of rickets present, yet in some milder cases which have recovered, the amount of rickets clinically evident has gone to the vanishing point, and in some cases it has been impossible to say clinically that rickets was present at all. Let us further note that cases of aggravated and progressive rickets, which are occasionally met with in which there is excessive bony deformity, with multiple fractures, or with extreme bone softening, do not present in spite of cachexia, during life, the symptoms which I have described, or *post mortem*, the hæmorrhages here shown. This was a difficulty quite recognised by the German writers, who wrote with inadequate knowledge of the *post-mortem* conditions, and they drew the distinction between acute rickets and severe rickets. It is clear that the phenomena of our disease and those of rickets, as commonly understood, though often coexistent, do not present what logical writers describe as concomitant variations.

As to congenital syphilis: In a very few cases an early history, compatible with that disease, was obtained, but no conclusive sign of active syphilis was noted during the course of the malady in question; nor was any syphilitic lesion found *post mortem*. The course of the limb affection, even with the pseudo-paralysis and the deep thickening, is distinct, clinically and pathologically, from that of any syphilitic bone disease with which we are acquainted. Though in a vague, indeterminate way some of these cases have been reported as infantile rheumatism, study of the descriptions given has shown that the joints proper were not invaded. This negative feature, with the freedom from heart affection, are adequate practical reasons for dismissing rheumatism as a factor in our disease.

None of the cases, so far as I know, belong to families of "bleeders." Moreover, the general absence of joint involvement shuts out the special limb attacks of hæmophilia. Although it is true that ecchymoses of various kinds occurred, and certain internal hæmorrhages likewise, yet small extravasations were rare, and the protracted course, and the special type of limb affection were unlike any case recorded of purpura hæmorrhagica.

With what known disease, then, does the group under consideration present most affinities? I submit that it presents most affinities with scurvy. The bone lesions which I have described have been found in scurvy, especially in young adults and adolescents. The special forms of fracture in the neighbourhood of the epiphyses have been described as occurring in young scorbutic subjects as far back as the time of Poupart. The special fractures of ribs, in which the rib proper is separated away from the cartilage, have been repeatedly observed in adult scurvy. The subperiosteal hæmorrhages again have been found. They are graphically described by our countryman Lind, whose work on scurvy was the pioneer of modern knowledge of that disease. They have also been described by Budd. In the cases of scurvy which occurred during the siege of Paris several necropsies were made in which blood was found effused under the periosteum of the tibia. Endosteal blood extravasations have also been described, both in the long bones and in the ribs, and a rarefying osteitis, like what I have mentioned, which doubtless greatly facilitated the proneness to fracture. The predominance of lower-limb affection, however explained, is the same in our malady and in adult scurvy.

Passing to other structures, the hæmorrhages and serous exudations of the muscular masses, and the consecutive wasting of the muscles, are characteristic. Moreover, the scanty internal hæmorrhagic lesions which I have described, as far as they go, agree with what has been found in scurvy. The great pallor and the muddy tint, probably due to reabsorption of altered hæmoglobin, are also common to our disease and to typical scurvy. Finally, with respect to the gums. We may classify the infantile cases into those who have limb symptoms with spongy gums, and those who have limb symptoms without spongy gums; and we have found that the state of spongy gums is practically conditioned by the presence of teeth. If there are several teeth, the sponginess is efflorescent, and in the severe cases the fœtor, the bleeding, and the protruding tumours are indistinguishable from what we find in typical scurvy. If there are no teeth, there is no sponginess, though there may be found on careful inspection minute ecchymoses. Now the greatest stress has been laid upon the gum condition in adult scurvy; but there is abundant evidence that a toothless man may present the limb weakness, the cachexia, and anæmia, and yet show no sponginess of gums whatever. In mild cases of land scurvy, also, it is constantly observed that the sponginess is limited strictly to the neighbourhood of those teeth which remain; and, if there be large intervals where the teeth have been lost, no sponginess appears in these intervals. I may here refer to a series of five cases which have been under my observation at different periods, but which in age were beyond the limit of infancy. In these five cases—children from 2 years to 10 years of age—there was undoubted scurvy, produced, I believe, by a curious hysterical antipathy to vegetables and meat. The gum affection was generally more severe than that of the infantile cases above described. The limb affection resembled that of the infant cases, but was less severe. The study of these cases occurring in childhood shows, I believe, a middle term (so far as the symptoms are concerned) between the infantile group and adult scurvy. The inverse relation to which I have referred is reasonable when we consider the physiological differences which exist. Before the eruption of the teeth there is a much smaller aggregate of blood vessels and blood supply, and much less liability to attrition than at a later period, when every tooth means the presence of a large leash of vessels. The physiological activity of every part of the growing bones of an infant is one of the most striking features of its physical life. This activity is modified apparently in the most rapid and subtle way by changes of general nutrition, and if there has already been induced a condition of rickets, with the alterations in the epiphysial line and in the periosteum which are characteristic of that disease, there is manifestly a prepared soil on which hæmorrhagic lesions can develop if an adequate blood alteration is forthcoming. Let me state it in another way. Rickets *per se*, as I have already said, does not (so far as experience goes) lead to these hæmorrhages. If it were so, considering how common a disease rickets is, surely these hæmorrhagic cases would be more frequently met with. Scurvy, we know from experiences in adults, is adequate to the production of such hæmorrhagic lesions. If, then, in our infantile cases the blood change due to scurvy comes into operation, the rickety change already present may act as a physiological determinant of the sites in which scurvy becomes manifest.

Now let us inquire whether there is anything in the antecedent conditions of these children which brings them into line with those under which adult scurvy arises. What are the conditions under which adult scurvy arises? There are many predisposing circumstances of faulty hygiene; but surely there is a sufficiently conclusive experience that prolonged deprivation of fresh vegetables or their equivalents is the most constant fact amongst the antecedents of the disease. I have said prolonged, because it is clear that the organism has the power of drawing on its reserves for lengthened periods to meet the deprivation of a complete aliment; and I have used the phrase "fresh vegetables or their equivalents" because we now know that fresh uncooked meat and fresh milk are antiscorbutic as well as, though perhaps not in so rapid a way, as fresh vegetables and fresh

fruit juices. Looked at as antiscorbutics, probably much larger quantities of fresh uncooked meat and fresh milk are needed than fresh vegetables and fresh fruit juices.

The chemistry of scurvy is still an unsolved problem—or perhaps I should say an incompletely-solved problem—for, thanks to the labours of Garrod, Ralfe, and others, we know that there is a diminished alkalinity of blood, and probably some fault in the presentment of the saline constituents of the food and in the facility with which they part with their bases. But the problem is possibly biological as well as chemical. It seems fair to say that the further we get from a living food the less is the antiscorbutic power. Fresh vegetables are more powerful antiscorbutics than preserved or cooked vegetables. Raw meat is more antiscorbutic than cooked meat, and raw meat juice than beef-tea. I suspect it will ultimately be found that raw uncooked milk is more antiscorbutic than cooked milk. What is there, then, in common between the antecedents of our infantile patients and those of recognised scurvy? So far as faulty hygiene is concerned, there is little in common. The majority of the infantile cases have been found in healthy homes and amongst good surroundings.

Let us turn to the question of food. In the group which I have described, in no single case at the time of onset of the malady has the child been breastfed. In the great majority, where complete details have been obtained, these infants are found to have been nourished on what may be called "preserved foods." In the front rank come the various proprietary infant foods, prepared by the addition of water to certain powders. Then come the different forms of condensed milk, and the proprietary foods made with condensed milk. Then come cases in which, either accompanied by proprietary food or not, fresh milk has been given, but with extreme dilution, during the later stages of infancy. Other categories I will speak of subsequently. Now let it be assumed, for the sake of argument, that such a diet as I have described is a scorbutic diet; and that an infant presenting the symptoms described is at the time being fed on this diet. The obvious test of the reasonableness of the scorbutic hypothesis is to alter the food in the antiscorbutic direction, and note the results. For the condensed milk let fresh cow's milk be substituted. For extremely diluted cow's milk let undiluted fresh cow's milk be substituted, as, for example, a full pint for a child of six months old. Instead of the proprietary food let some sieved potato be mixed with the milk every day, and a tablespoonful of meat juice or gravy likewise. Finally, let a tablespoonful of orange juice or grape juice be administered every day in divided doses, mixed with water as required. And what is the result of these very simple alterations? The result in two or three days is startling. As a rule, the food is taken greedily and without digestive disturbance. The child becomes much more contented; the tenderness of the limbs rapidly diminishes; the sponginess of the gums almost immediately recedes; the pallor becomes notably less; if there has been any renal hæmorrhage it ceases; fresh ecchymoses rarely appear.

It is clear that the progress of the disease is definitely arrested. So far as the local treatment of the limbs is concerned nothing is essential but the maintenance of rest in the horizontal position; and this meets another indication, namely, to prevent possible heart failure, consequent upon the extreme anæmia and fatty degeneration. Experience has abundantly shown that the blood tonics, such as iron, arsenic, and phosphorus, are useless if the proper change is not made in the diet; and if the proper change is made they are scarcely necessary; although fresh air and sunshine will not prevent the oncoming of the disease, they probably aid recovery when the food change has been effected. It is occasionally found that after the scorbutic need has been satisfied, and the cachexia has subsided, the child is no longer able to assimilate the large quantities of vegetable material and fresh undiluted cow's milk, which at the beginning of the treatment were taken with avidity and digestion. This also is parallel with what has been found in the treatment of adult scurvy.

This completes the recapitulation of the results submitted to the profession in 1883. I now pass to the second part of my lecture, and shall state as concisely as I can how far the former conclusions have been modified by subsequent experience and criticism. First let me mention the experience of others. A considerable number of examples have been recorded both at home and abroad. Dr. Cheadle has reaffirmed his original propositions with further illustrations and the account of one important necropsy, to which I will subsequently refer. Dr. Gee, Dr. Eustace Smith, Dr. Goodhart, Dr. Railton, Dr. Sutherland, Dr. Green, and Dr. Charpentier have published cases, or series of cases. In Germany series of cases have been recorded with considerable detail by Dr. Rehn, of Frankfurt, and Dr. Heubner, of Leipzig. The general conclusions previously stated have been strongly supported by these two eminent physicians. The most striking confirmation has come from America—a country which rivals our own in the favour with which proprietary infant foods have been received, and in the reluctance of well-to-do mothers to suckle their offspring.

The first case which appears to have been observed was in 1889, and was reported by Dr. Northrup, physician to the New York Foundling Hospital. In 1891 11 cases were collected by him; and in February of this year no fewer than 106 cases were reported to the New York Academy of Medicine by Dr. Louis Starr, Dr. Rotch, Dr. Holt, and others.

Detailed histories of 36 of these have been analysed by Dr. Northrup. It is obvious from his paper that the clinical and anatomical features described are identical with those of the group under consideration, and the conclusions arrived at are almost word for word those which were stated in 1883. My own experience of this disease since 1883 comprises 33 typical examples, besides a number of what may be called "borderland" cases, to which I will presently refer. But after this general enumeration of available material, let me refer to some of the results of the collective experience. First as to anatomical results. A very striking case was communicated to me by my friend Dr. Stephen Mackenzie. On *post-mortem* examination extensive subperiosteal and endosteal extravasations and fractures of ribs and lower limb bones were found. But in addition there were innumerable small hæmorrhages in the intestines, mesenteric glands, lungs, and the pyramidal structure of the kidneys.

Dr. Colcott Fox has published drawings of a remarkable case with extremely widespread subperiosteal hæmorrhages. In the American series, collected by Dr. Northrup, there are four *post-mortem* examinations recorded. The most important one is Dr. Northrup's case. This was a child 18 months old with extensive subperiosteal hæmorrhages, but no rickety changes. The necropsy recorded by Dr. Cheadle presented only slight rickets, and the bones and muscles were free from hæmorrhage. Besides the characteristic hæmorrhages into the gums there were extensive extravasations into the lungs, and minute extravasations into the intestinal mucous membrane and lymphatic glands. One of the most interesting recent communications is that of Dr. Sutherland, who, whilst laying great stress on the frequency of the limb extravasations, reports a fatal case like Dr. Cheadle's in which the bones were free. Dr. Sutherland has also reported two examples of extensive hæmatoma of the dura mater, which he believes to have been due to infantile scurvy. A similar condition is recorded as occurring in adult scurvy. Since Dr. Sutherland's paper was published I have also had a case of a boy, aged 15 months, who under my observation presented slight scurvy of the gums. From the history I believe it probable that he had had an earlier attack of this disease. At the *post-mortem* examination an old extensive hæmatoma of the arachnoid cavity was found. This was possibly due to a long-past scorbutic hæmorrhage. At the Children's Hospital, Great Ormond Street, since 1883 three *post-mortem* examinations on cases of scurvy have been made: one under Dr. Cheadle, to which I have already referred; one under Dr. Abercrombie, and one under Dr. Lees presented characters identical with those described in the first part of the lecture. Besides the actual necropsies there is a certain amount of anatomical experience which has been obtained by surgical exploration during the life of the patients. The most striking example of this kind was recorded by Mr. Page, in which prolonged incisions were made down to the bone in both thigh and leg, and great masses of blood clot removed. There was recovery, but in addition to the operation an entire change was made by Mr. Page in the diet, and the proprietary food with which the child had been fed since three weeks old up to nine months was replaced by fresh food. In some of Rehn's

cases and in some of the American cases simple exploratory incisions have been made, and the existence of blood extravasation under the periosteum established. Thus far as to anatomical results. And now as to the clinical addenda to our knowledge. Mr. Holmes Spicer has drawn the attention of ophthalmic surgeons to three examples of the curious eye affection which I have already referred to in the first part of this lecture. After pointing out the association of this special form of orbital hæmorrhage with other scorbutic signs, he suggests, in the case of a handfed child of seven months, that a similar orbital hæmorrhage was the only scorbutic sign. This is a difficult doctrine to accept, but I believe it to be sound. Out of seven of my cases in which orbital hæmorrhage occurred, four presented the orbital symptoms before the limb symptoms, and in some of these the limb symptoms were slight, there being great local tenderness, much irritability, but little swelling.

I have spoken of hæmaturia as one amongst the symptoms of our typical group. Dr. Gee, and subsequently Dr. John Thomson, have pointed out that occasionally hæmaturia may be almost the solitary manifestation of scurvy in infants, and that it may immediately vanish after the employment of fresh food. Sir William Roberts has told me that he has observed examples of this scorbutic hæmaturia, without other symptoms, but responding to antiscorbutic diet. I have notes of two cases of hæmaturia in which, though no bone lesions were to be felt other than rickets, there was an excessive irritability, resentment to the slightest touch of the lower limbs, and some amount of anæmia. They had been fed on preserved food, and one of them immediately responded to antiscorbutics; the other I have been unable to trace. These cases lead me to mention another example of what may be called a borderland condition. There are cases of rickety infants in whom, though no lesions pointing to scurvy can be detected, the irritability and tenderness are out of all proportion to the signs found. The substitution or increase of what may be called "living food" in the diet, will often entirely eliminate this irritability and tenderness, just as in the typical cases which I have already described. I find that Dr. Cheadle and Dr. Eustace Smith have noted a similar experience. Just as we are familiar with a vanishing point of rickets, we may possibly have to recognise a vanishing point of scurvy. The collective experience shows that some of the cases have had an early infancy with much stress and storm in the way of digestive disturbance. The artificial food upon which ultimately they have been nurtured is a sort of survival of the fittest, as being the one attended with least diarrhæa and vomiting; and it is whilst assimilating this artificial food that the scorbutic symptoms have developed. But I wish to emphasise the statement that in chronological order the symptoms of our disease cannot justly be regarded as the last term of a condition of marasmus. It is not in the least comparable to the appearance of purpura at the close of a chronic wasting disease. Further, it is recorded that, in a great many cases, at the onset there is no obvious digestive disturbances, or one only of very moderate degree. Many of the later reports state that the children were well clothed with fat, and regarded by the friends as well nourished. The immediate determining cause of the catastrophe is difficult to ascertain. In some of the cases, as in adult scurvy, a slight accident—bruise or fracture—seems to have started the complaint.

One of the most suggestive results of the collective experience relates to the social state of the children amongst whom the disease predominantly occurs. The clinical *tout ensemble* of a typical example is as striking in its own way as that of myxœdema or acromegaly. If this were a common disease in our large out-patient departments of the general hospitals and the children's hospitals it would be recognised; but amongst the very poor it is not a common disease. Of the 33 typical examples which have come under my care since 1883, only 6 were hospital cases. The others were the children of well-to-do people with healthy surroundings and good homes. There was nothing to suggest either wilful neglect or faulty hygiene in any gross sense. We may speak with confidence of what we ourselves observe as to the relative frequency of a disease in different groups of people; but it is somewhat rash to generalise as to the change of type of any disease in different times. Nevertheless, I will hazard the statement that this disease is probably more frequent now than, say, twenty-five years ago. It is, I think, inconceivable that men of the authority of Jenner and West and Hillier should not have insisted upon it if this group of symptoms had often occurred within the common range of their experience. I observe that Dr. Northrup, in his valuable summary of the American experience, states that it seems probable that the disease is increasing in frequency in America. Is there anything noteworthy of late years about the bringing up of the infants of the well-to-do classes? I think it may be safely stated that, besides the increasing difficulty of getting the mothers to suckle their infants, there has been an enormous increase in England and America in the employment of proprietary infant foods. The proprietary foods are much more extensively used among the well-to-do than among the poor. But different forms of condensed milk have also come into extensive use, and to a considerable amount among the poor.

Is there any other reason why the poor, though by no means exempt from the disease in question, should suffer from it in a remarkably less degree than the rich? I believe that an important suggestion made by Dr. Cheadle gives a second clue to this remarkable difference. The children of the poor at a much earlier period than those of the well-to-do receive small portions of the same food as that of which their parents partake. No doubt in consequence of such indulgence there are occasional primary digestive disturbances. Nevertheless, some breaks are made in the monotony of the diet, and probably some antiscorbutic article of food is taken. Among the children of the poor, potatoes especially are given at a much earlier period than to the children of the well-to-do. Thus, although the children of the poor are rickety, they are much less frequently scorbutic than the children of the rich.

Thus, to sum up, the children of the poor suffer less from scurvy than the children of the rich, because poor parents cannot afford to buy the proprietary food which the rich parents buy, and because the poor parents, even when they use condensed milk, give their children a mixed diet at a much earlier period than rich people give it.

Are there any addenda to be made to the former results obtained in respect to the diet of the children who were attacked with this disease? I think there are. Permit me to speak first of the results of my own later experience on this point. It still stands out as a striking fact that the proprietary foods are the great offenders, especially those which are prepared with water and with condensed milk, or with a very small amount of cow's milk. Condensed milk is responsible for a fair number. The disease also occurs when very diluted cow's milk is used, and especially when for some reason, after a long employment of considerably diluted milk, the dilution is suddenly carried to a further stage, even with or without the substitution of some artificial food. The disease also occurs when peptonised milk has been given over long periods. Several definite examples have been observed in infants to whom for several months as much as a pint and a-half and one quart of humanised sterilised milk have been given in the twenty-four hours. By this preparation I mean milk which has been deprived of half its quantity of casein, and which has been subsequently sterilised, and in several cases stored for some weeks. I think there is reason to suspect that the boiling of cow's milk and prolonged sterilisation (especially at high temperatures) lessens in some degree its antiscorbutic quality. In Germany and in America the sterilisation of milk has become much more systematised and extensively adopted than with us. Professor Heubner, of Leipzig, in his summary of conclusions in regard to scurvy in infants, speaks with the greatest caution on this point. He is very emphatic against prolonged sterilisation, or sterilisation at high temperatures. The same suggestion is embodied in the summary of the American experience to which I have referred. Any extensive employment of the storing of sterilised milk for long periods by the dairy companies is, I believe, attended with risk; but when we consider the risks of the dissemination of various diseases by unscalded milk, and the serious primary digestive disturbances that arise in connection with it, it would be a retrograde step to say a word in discouragement of the routine practice of scalding milk. There is, I think, a way

of meeting the difficulty. If I have made my meaning clear, it will be abundantly manifest that as a rule it is the prolonged use of a defective diet which induces the symptoms which we have considered. The early cases of infantile scurvy are very exceptional. The age at which we ought to be on the *qui vive* for the initial symptoms of scurvy is about the eighth month of infantile life. If by that period a moderate quantity of scalded milk have been given (say, 1½ pint in the twenty-four hours), then, instead of adding one of the proprietary foods as is the common practice, we may ward off the occurrence of scurvy by the addition of gravy or meat juice to the milk, but still better by the addition of some carefully sieved potato. The fear of non-assimilation of starch has prejudiced us unduly against the use of living food. Should it be found that potato, cooked and sieved and given with milk and gravy, is not tolerated (which is a very rare event), we have in small quantities of the juice of fresh fruits a ready method of anticipating scorbutic symptoms. There is still one other point to which I must refer as one of the alleged results of the later experience. I refer to the possibility of scurvy appearing in infants at the breast.

At the discussion on infantile scurvy at the Berlin International Medical Congress, Dr. Pott stated that he had seen one example of twin children suffer from the disease although breastfed. No details are given as to whether the mother was scorbutic or as to the total amount of milk taken by the infants: but it is stated that their suckling was impossible on account of the soreness of the mouth. No information is given as to other scorbutic symptoms, and it seems an open question whether the stomatitis was really scorbutic. The case scarcely invalidates my original contention. In the *Deutsches Archiv für klinische Medicin* for 1880 there is the record of an epidemic of scurvy by Dr. Kuhn, of Moringen. There are 13 cases of infants recorded, several of them quite young, and being suckled by mothers who were scorbutic at the time. These infants suffered from severe catarrh of the mouth, from bronchitis, and from some skin affections, for example, pemphigus, erythema, and in a few cases petechiæ. Some of them died, but I fail to find any account of *post-mortem* examination. The details are too meagre to help us in our inquiries. The time at my disposal will not permit me to refer at length to the criticisms which have been offered to the view held by Dr. Cheadle and myself as to the pathology of this disease. They may be summarised under these three headings: (1) That the disease is nothing more than acute rickets; (2) that the food conditions under which the disease arises are different from those under which undoubted scurvy arises; (3) that although the group of symptoms and the pathological appearances present a close resemblance to those of scurvy, these infantile cases cannot be considered true scurvy because they do not occur in an epidemic or endemic form.

With the first view—that the disease is nothing more than rickets—I have dealt at length in the course of this lecture, and I need not further discuss it. The German physicians, so far as my reading has gone, have relinquished it entirely. With respect to the second criticism—that the food conditions under which the disease arises are not truly scurvy-producing—I have given reasons for the opinion that the diet employed does not come under the category of living food, adequate in quality and amount. I have also shown that inadequacy of living food, *plus* a basis of rickets, gives the simplest explanation of the typical case. The immediate result of antiscorbutic treatment seems the most conclusive answer to this criticism, and in a crude way would seem to be a measure of the proportion of scurvy present in any given case. The third objection—namely, that the scorbutic nature of these cases cannot be accepted because the cases do not occur in epidemics—has been urged recently by Dr. Hoffmann in his work on constitutional diseases. In common with many of the German school, Dr. Hoffmann is dominated by the epidemic doctrine of scurvy, and by the hypothetical view that the efficient cause of it is some micro-organism. On that hypothetical view I will express no opinion, but English physicians since Lind's time are not dominated by the epidemic doctrine. The many undoubted sporadic cases are to them just as significant, and in some respects more instructive, than those which occur in epidemics. With these examples our cases come into the closest relationship, and a perfect series may be traced from infancy, through childhood, to adult life, in which the differences of symptoms are explainable by the different physiological activity of the tissues specially involved.

SIXTY-SECOND ANNUAL MEETING

OF THE

BRITISH MEDICAL ASSOCIATION.

Held in BRISTOL July 31st, and August 1st, 2nd, 3rd.

PROCEEDINGS OF SECTIONS.

SECTION OF MEDICINE.

FREDERICK T. ROBERTS, M.D.Lond., F.R.C.P., President.

A DISCUSSION ON FUNCTIONAL DISEASES OF THE HEART.

I.—R. DOUGLAS POWELL, M.D., Lond., F.R.C.P.,
Physician Extraordinary to the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Brompton Hospital.

THE subject which I have the honour to introduce for discussion to-day is one of such wide and ill-defined limits, that I fear I shall have great difficulty in keeping within the time at my disposal, and in bringing out those points which are for special discussion; but I shall do my best, and, by the aid of the diagrams and tables which I have had placed on the wall, I hope to be able somewhat to shorten what I have to say.

The term "functional diseases" (which is not of my choosing) has in recent times excited some controversy, but I am bound to say that I think it is a very excellent term to cover that large class of disorders of the heart which cause a vast amount of discomfort, and sometimes an acute degree of suffering, which lead up in many instances to distinct cardio-vascular changes of a morbid kind, and which are yet, in the early periods, not associated with any actual structural change of the heart. An old friend of mine was once consulted by an American lady on account of her health, and, after going through the whole of her symptoms with his usual thoroughness and care, he informed her that she had no disease of any organ whatever. The remark of the lady was, "Well then, doctor, I guess any way I don't function well." Functional disease of the heart may be described as a state in which, without any organic change that we can discover, it yet does not "function well." "A perverted action and sensibility of the heart, not dependent upon structural change."

I venture to think, however, that we must somewhat enlarge the scope of this definition. It is impossible in clinical medicine adequately to consider the heart as a working organ without taking also into consideration the conditions of the vascular system generally. We cannot but perceive that the heart and arteries constitute an interdependent mechanism, in whose grasp the blood is held—a mechanism regulated by an involuntary nervous system, in its turn watched and in part controlled by our consciousness; and this wider scope of our subject necessitates the substitution of the term "cardio-vascular system" for "heart" in our definition.

UNDUE PERCEPTION OF THE HEART'S ACTION.

Now, the first group of cardiac disturbances of which I shall have to speak brings into strong relief the close association between the small vessels and the heart of which I have been speaking. It is characterised by an undue consciousness on the part of the patient of his heart's function. This is a very common disorder, and amongst people of nervous temperament it sometimes amounts to positive suffering and disease. I have under my occasional observation at the present time a brother and sister, aged respectively 46 and 42, who have a very strong nervous inheritance. Their father died insane; their mother died of ascending paralysis