A CLINICAL LECTURE ON A CASE OF SCURVY-RICKETS IN A BOY OF TWELVE YEARS.

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GENTLEMEN,—Many of you have already seen this interesting case, for I brought the boy before you on October 23rd, immediately on his admission to the hospital, but as I had not time on that occasion to go fully into his history, I had to content myself with calling your attention to the most salient features of his disease, for I was anxious that you should see them whilst they were in their most marked form. Briefly, as you will remember, his condition was this: He looked extremely ill; he was anæmic to the last degree, feeble, and listless; his ankles were swollen, his legs were motionless, and his gums were spongy and bleeding. His mother, who had just then brought him up to the Hospital for urgent admission, admitted, in answer to my questions, that he had been allowed to live on a diet which was entirely destitute of fresh fruit and vegetables. And you will remember that I told you in her presence that he was as surely suffering from scurvy as a sailor might be upon an Arctic expedition or in a prolonged sea voyage, in which fresh food was absolutely unobtainable.

I then called your attention to the fact that the boy was markedly rachitic; his ribs were swollen at the junction with the costal cartilages, as were also the carpal ends of his radial diaphyses. Of the lower end of his tibiae we could say nothing definite, as they were hidden in the midst of a large amount of soft effusor.

Whether the boy was rachitic during cradle life or not I was unable to say positively, though I thought it highly probable. At any rate he is extremely rachitic now. If, however, he was not rachitic in cradle life the interest of his case is increased, for then it would be that his diet in later childhood has produced rickets, and, as you know, rickets is peculiarly a disease of infancy, though the effects upon the skeleton may persist throughout life.

As I told you when we first saw the boy together, the diet of rickets is the diet of scurvy, and if a fairly healthy boy of, say, six or seven years is kept continuously upon a typical scorbutic diet, I do not see why the ends of his ribs and his radii should not begin to swell and keep on swelling just as in similar circumstances they would do in cradle life. But rickets is not confined to cradle life. It is specially a disease of infancy, merely, I suppose, because the dyspeptic child who has struggled through the perils of his cradle life is directly afterwards put upon a mixed diet, and it is this mixed diet that is the saving of him. If he were still kept on the scorbutic diet of his cradle life after he had reached the age of 3 or 4 years, he would either die of exhaustion or would be more than ever rachitic, or, perhaps like this boy, he would become the subject of unmistakable scurvy.

Thus, you see, I deem rickets to be the prescorbutic stage of scurvy; and when I am told that an infant or a child is the subject of scurvy, I do not need to be told that he is also rachitic. There is no boundary line between the rickets and scurvy of childhood; rickets gradually and imperceptibly passes into scurvy, and I cannot imagine any child to be the

subject of scurvy who has not beaded ribs and swollen ends of radii. Though Dr. Cheadle has given the name of scurvy-rickets to the disease, I should not be averse from entirely dropping that part of the word after the hyphen, and calling the disease simply scurvy, feeling well assured that the scurvy has been developed from pre-existing rickets.

Some physicians are apparently in favour of calling the disease simply hæmorrhagic rickets. But, surely, when hæmorrhage has occurred beneath the periosteum or into the planes of other connective tissues, the disease has become developed into something more than rickets. Hæmorrhage is not a symptom of rickets but is a most characteristic sign of scurvy. Thus, the disease is now scurvy, not rickets. But the word scurvy is so thickly surrounded with unpleasant associations, that he would be a rash practitioner who told a lady that her child was the subject of that disease. Indeed, the word rickets has in such circumstances to be mentioned with care, lest the mother and the nurse be grievously offended by it, though since these patent foods (so-called) have found an easy entrance into the nursery, parents have become somewhat more accustomed to the sound of the word, and are, in consequence, less shocked by its personal application.

The report of the case is by the dresser, Mr. Reginald Hughes, whose brother, Mr. Thomas Hughes, kindly took the photograph printed herewith.

The patient is a schoolboy, 12½ years of age, and he comes from the middle of Essex. He was apparently always healthy, though last February he was in hospital for division of each tendon of Achilles. From the time of that operation the child has been peevish and fretful, and the parents have given way to him on all points. He is very anæmic and sickly, and has a wrinkled face, like that of an old man. The knee-joints are swollen, and there is some fluid present. The ankles and feet are swollen, and œdematous. There is wasting everywhere. He is 3 feet 11 inches high, and his weight is 39½ lbs. The teeth are slightly loosened, the gums are spongy, and occasionally bleed, especially when he eats hard food. The abdomen is swollen; the liver and spleen are slightly enlarged. The urine was alkaline (specific gravity 1030), and it contained a considerable amount of blood. The heart and lungs were healthy.

As regards diet, his mother said that he persistently refused to eat vegetables and fruit, and that he took very little milk. She admitted that she let him have just what he wanted, and forced him with nothing. At break-

fast he had egg or bacon or some such thing, with bread and cocoa. At dinner he had meat or fish, with occasionally a little potato and more cocoa. Later in the day he had tea and bread and butter, with occasionally potted meat, and at supper he had bovril or cocoa.

This was certainly a highly improper diet for a boy, for it was conspicuously deficient in fresh, "living" food. I grant that in the daily menu there was a certain amount of the "living" food in the shape of milk, fresh meat, and occasional potatoes, but we gathered from the mother that he did not have much potato, and he would have had to eat a very large amount of fresh meat in order to take in the needful supply of fresh material—whatever that material may actually be—to make up the deficiency which was lacking by the absence of ripe fruit and green vegetables. Unfortunately, moreover, the boy seemed to prefer cream to milk. He took extremely small quantities of the latter.

What I would again ask you particularly to notice is the swelling about the lower end of each tibia and fibula. It is manifest in the photograph which I hand round. This swelling is due to extravasation of blood into or beneath the periosteum at the lower end of each tibial diaphysis. You will notice a similar condition about the lower end of the femur and also over each tarsus. Of course I cannot say for certain that the swelling is due to the presence of blood; but

n a somewhat similar case in an infant my colleague, Mr. Page, cut down upon a diaphysis and found a large collection of blood, and in several other instances a late colleague of mine at the Great Ormond Hospital, Dr. Barlow, found similar extravasations *post mortem*. These accounts, as some of you may be aware, are now classical. In this boy the legs were very tender; he never moved them, and he could not bear their being handled.

In his well-known lectures delivered at the Hospital for Sick Children in 1864, Sir William Jenner gave as the third characteristic symptoms of rickets a "general tenderness" of the child, so that it "cannot be moved without its uttering a cry." This certainly is not my experience. I have handled many rickety children, and in the course of a surgical examination, not always with absolute gentleness, but I confess that I have not noticed such "general tenderness." I make bold to think that when that illustrious physician was impressed with the importance of the symptom of general tenderness in rickety children he must have been examining cases in which the rickets from which they were suffering was extreme, and was gradually passing or had actually passed into a condition of scurvy, in which hæmorrhages were taking place into or beneath the vascular periosteum, and the bones in consequence were extremely tender. Strange to say, that acute observer and great clinical teacher told Dr. Barlow that he had never met with a case of rickets which was so advanced upon the road to scurvy as to be associated with subperiosteal extravasations of blood.¹

Our little patient has now been in the hospital ten days; and, though his condition has in that time greatly improved, he still presents some of the most characteristic signs of scurvy. Thus you will notice that he is excessively anæmic, and, on turning down his lower eyelid, I show you that the normal, bright-red capillaries are conspicuous by their absence. On removing his blanket you notice that his skin is not exactly pale; it is destitute of the natural hue, and is muddy-looking. This is probably due to a disintegration of some of his red corpuscles, and to certain constituents of their colouring matter leaking out of his ill-nourished capillaries, and becoming deposited in the surface. His gums, which at the time of his admission were greatly swollen, blotchy, and unwholesome, are now almost normal in appearance, and they no longer bleed.

There is no possibility of your getting away from the diagnosis which some of you have already made of this case, namely, rickets and scurvy. Last Monday (October 30th) we had under discussion, as you will remember, a girl with septic osteomyelitis. But I think that none of you would suggest that this is a case of that nature, with the septic inflammation attacking the lower end of each tibial and fibular diaphysis. Osteomyelitis is a common disease with children, but it is a terribly acute and lethal affection. A boy who was the subject of it would not be sitting contentedly as you see this boy. He would be looking desperately ill; his temperature would be high, the bone-ends would be exquisitely tender, and the neighbouring skin would be red, hot, and cedematous. I admit that these ankles are somewhat tender, but the skin over them is pale and cool. Moreover, as the House-Surgeon, Mr. Priddle, and the dressers will tell you, the boy has greatly improved since he has been under antiscorbutic treatment. No internal medicamentation, outward applications, or carefully-ordered diet would have had any influence whatever on the osteomyelitis of childhood; and if in such a case the surgeon refrained from active treatment the whole diaphyses would be quickly ruined, and perhaps the limb or the life lost. Lastly, I have never seen such a symmetrical distribution of the acute disease. This symmetry itself suggests some general constitutional affection. Certainly the case is not one of osteomyelitis.

Is anyone of you inclined to regard the case as one of a "rheumatic" nature? "Rheumatism" is a comprehensive term, and useful as a refuge when the practitioner is face to face with obscure affections. But the absence of constitutional excitement, the quiet condition of the ankles, and the absence of the redness and heat about the joint are all against that theory. Moreover, the disease is, as I have said, rapidly clearing up under an antiscorbutic diet, and his temperature is normal.

Having regard to the absolute uselessness of the boy's legs, and to the fact that he never moves them when in bed or on

a chair, it is likely that the thoughts of some of you have wandered, if only for a moment, to infantile paralysis. But in that condition, although there might be local tenderness in the limbs, there would not be marked, symmetrical swellings, as you see here. Still, when scurvy attacks an infant, it is quite easy to fall into the error of attributing the motionlessness of the leg and thigh to an attack of poliomyelitis. Indeed, only on Monday last I went a considerable distance into the country to see an infant of seven months, who, it was thought, might be the subject of infantile paralysis, though the actual explanation of the pseudo-paralysis was periosteal hæmorrhage, the result of acute rickets. This infant had been brought up on a well known patent food administered in one of those terrible tube-bottles which are in too general use.

Since this boy has been in the hospital, he has been fed upon fresh fruit and vegetables, beef jelly, sweetened lemon juice, milk, meat and bread and butter, and he has, as you see, greatly improved upon the diet. He looks happier because healthier; his ankles are much less swollen, and he moves his legs a little; his gums have lost their sponginess, and blood has entirely disappeared from the urine.

Altogether, the case is one of great interest and rarity. I certainly never saw scurvy in a child older than three years, though I know that it has been met with in children up to ten years by others. Still, I am sure that I might look for a long while before discovering many such records. A country boy of ten years is almost invariably a great lover of fresh fruit. Is it not somewhere about this age that he is apt to creep through a hole in the hedge to possess himself of a few of his neighbour's apples? But, strange to say, this boy has been subject to no such temptations.

Possibly you may imagine that scurvy-rickets is more apt to be found amongst the children of the poor than of the rich, but, as a matter of fact, the reverse is the case. If the poor woman sees upon the the hoardings or in the railway carriages the speciously illustrated advertisements of those patent foods (so-called), she pays no heed to them; their price has fortunately placed them beyond her reach. But the well-to-do mother takes a delight in buying them. I have seen many infants with scurvy-rickets, and my general impression is that the worst cases were those which had been fed upon the most extensively advertised patent foods. One of the last cases that I saw, however, was an infant in a large house in Kensington, who had developed the disease by being fed upon fresh cow's milk diluted with large quantities of barley water. This feeding had been going on for a long while. Some infants become scorbutic by being continuously fed upon pancreatised milk, and I very much fear that the widespread habit of scalding the nursery milk with the view of freeing it of certain germs of disease materially detracts from its value as a food for hand-reared infants. At any rate, I would urge you, as Dr. Cheadle has done in his valuable Lectures² upon the artificial feeding of infants not to keep on continuously with any kind of artificial food. But both doctor and mother are apt to be so pleased when they have at last discovered a food which apparently suits the baby, that they are afraid to give it up when the dietetic crisis has entirely passed away. And so it is that the intercurrent of rickets and of scurvy is invited.

There is another reason, as pointed out by Dr. Cheadle, why the child of the poor person is not so likely to develop scurvy as the child of the rich, and that is because the former is likely to have a much more liberal and varied diet. In the out-patient department of the hospital you ask a mother how her dyspeptic or rachitic child is fed, and her reply is generally of this sort, "We gives him something of what we have ourselves." Of course, physiologically, this may be very wrong, and something worse than wrong, when the infant is allowed to sip beer out of his father's mug, or even gin and water out of its mother's glass. But, on the whole, the system is not without its recommendation. For the potato and gravy, the chicken bone, or bit of crust which is given to be sucked, and the piece of banana or pulp of apple with which some of these small, rachitic infants are indulged, have probably the effect of preventing the rickets advancing a stage further, and becoming scurvy. For there is no hard and fast line between rickets and scurvy; the diet which begets rickets if only continued long enough will produce typical scurvy.

I ask my friend, Dr. Mahon, of Ballinrobe, who is sitting

beside me, if he ever saw a case of scurvy in a child in the West of Ireland, and he replies that he is glad to say that the people there are not yet sufficiently "civilised" for their children to be even rachitic. Yes, civilisation is certainly not an unmixed blessing. Not very many years ago rickets was unknown in America; now there is plenty of it in certain parts of New York and other large cities. So also with Victoria and New South Wales; with the introduction of civilisation rickets has made its unwelcome appearance in Melbourne and Sydney, and the fear is that it means to stay. Rickets must, therefore, no longer be considered "the English disease."

REFERENCES.

- ¹ The Bradshaw Lecture, BRITISH MEDICAL JOURNAL, November 10th, 1894, p. 1031. ² Smith, Elder, and Co., 1894.

THE HARVEIAN LECTURES ON THE SURGICAL TREATMENT OF TUBERCULOUS DISEASES.

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LECTURE II. TUBERCULOUS PERITONITIS.

[AFTER some remarks on acute tuberculosis of the peritoneum, the lecturer went on to describe the following chief types of lesions which may be found in cases of tuberculous peritonitis:]

VARIETIES.

1. The peritoneum may be studded with tubercles of varying size, some small, greyish and transparent (the ordinary miliary tubercles) others larger and tending to become cheesy. These tubercles are scattered over both the visceral and parietal peritoneum, and there are frequently patches of fibrinous exudation over them which readily peels off, and in the early stage there is not necessarily any matting together of the intestines or shrinking or adhesions of the omentum or mesentery. The peritoneum is usually markedly hyperæmic. In these cases there is almost always more or less fluid present in the abdominal cavity, usually of a lightish straw colour, but in more acute cases somewhat opalescent or stained with blood. The sensation on putting one's finger into such an abdomen is the same as if it were passed into a bag of sago.

The tubercles are not usually distributed over the whole cavity, but are most numerous in patches, and this is more especially the case where the origin of the tuberculous peritonitis is some tuberculous lesion inside the abdominal cavity. For example, in peritonitis originating from the tubes, the tubercles are most numerous in the pelvis, and become less numerous as one passes from it; or, again, in tuberculous peritonitis originating in connection with tuberculous ulcer of the intestines, the tubercles are in the first instance most numerous over the intestines in the neighbourhood and less numerous elsewhere.

Although, as has been said, in these cases the intestines are not usually matted together, still in some instances the fibrinous exudation produces sufficient adhesion to prevent the free spread of the fluid over the whole abdominal cavity, and thus we may have one or more collections of fluid simulating cystic tumours.

2. In a second set of cases the formation of adhesions and fibroid induration of the omentum and mesentery are the marked features. The intestines become bound together in masses by new fibrous tissue in which miliary tubercles may be present. These adhesions are often very firm, and may in parts constrict the intestines, or in other parts, from their shrinking, lead to kinking of the intestine, and in both ways they may cause partial or even complete obstruction. Further, the intestines are not only adherent to one another, but they are also very apt to contract adhesions to the abdominal wall, and this forms a great difficulty in performing laparotomy in these cases, and it is also the great objection to aspirating the abdomen where fluid is present. Where the whole abdomen is involved these adhesions are especially marked

between the liver and diaphragm. The omentum is also early involved in these fibroid changes, the two layers of the omentum become matted together, and the whole structure becomes much thickened and shrunken, and in an advanced stage forms a thick rolled-up mass like a sausage lying about or above the level of the umbilicus, and usually running more or less transversely across the abdominal cavity; it is generally adherent to the abdominal wall at this part. This thickened omentum contains numerous tubercles in its substance and scattered over its surface. The mesentery is also markedly affected with similar changes, and becomes thickened and shrunken and drags the small intestine up towards its root, and thus it comes about that when fluid is present it is most apt to collect towards the left side and lower part of the abdomen. The mesenteric glands are also enlarged, but not as a rule cheesy.

In these cases a certain amount of cirrhosis of the liver may be met with, or there may even be tubercles in the substance of the liver. The spleen also is not uncommonly enlarged, either as the result of passive congestion or hyperplasia, or in some cases from the presence of tubercles. This adhesive form of tuberculous peritonitis may or may not be accompanied by the presence of fluid, and where fluid is present it is most often encapsuled, and there may be more than one collection.

3. A third condition which may be present, and one which is extremely grave, is that the tubercles run together and form large masses which undergo caseation; the mesenteric glands are enlarged and caseous; the omentum is often converted into a caseous mass, and caseating masses form in the adhesions which bind the intestines together. The contractions and distortions of the intestine and the shrinking of the mesentery and the other changes mentioned under the second form are usually much exaggerated. This condition is especially associated with intestinal ulceration, and the intestine is usually thinned, and may even be perforated. Collections of fluid are frequently present, most usually encapsuled and multiple, and not uncommonly purulent. The pus may present all the characters of ordinary tuberculous pus, but in other cases it may be foul smelling, either as the result of infection through the intestinal walls, or as the result of actual perforation of the intestine. In this form, in infants especially, the condition may be most marked in the neighbourhood of the umbilicus, and the pus may make its way through the abdominal wall at this point. Where this occurs we find a sinus passing into the abdomen, and not uncommonly a fecal fistula is present as well. As this fecal fistula is usually in the small intestine, and often pretty high up, the child rapidly emaciates and goes down hill.

In both the second and the third forms the upper part of the abdominal cavity may be healthy, the disease being shut off above by the adhesion of the omentum to the abdominal wall at the level of the umbilicus. In other cases it may be still more circumscribed, and form a limited patch with fluid in the interior, this condition being most commonly met with in the form of an irregular fluctuating swelling in the left iliac fossa. Another local form is where it is limited to the pelvic region, which occurs especially in connection with infection from the Fallopian tubes; or, again, we may meet with a tuberculous perityphilitis forming a mass in the right iliac fossa, which has been mistaken for a malignant tumour. These masses are usually secondary to tubercle of the appendix or cæcum.

There are many intermediate forms and varieties of these pathological changes which it is impossible to enumerate here, but these are the three chief types, and they are of especial importance from the point of view of treatment.

ETIOLOGY.

[As regards the etiology of tuberculous peritonitis, the lecturer pointed out that it usually occurred in the course of tuberculous disease elsewhere, such as phthisis, tuberculous diseases of bones and joints, genito-urinary tuberculosis, etc., but in some few cases it has been the only tuberculous lesion discovered, and may therefore be looked upon as of primary origin. Judging from a number of published statistics, the frequency of the primary forms as compared with the secondary is something like 2 to 10 per cent. In these primary cases it has been supposed that the bacilli enter through the wall of