TUBERCULOUS DISEASE OF BONE.

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II.

I HAVE already referred to the existence of small hard masses in the cancellous tissue of scrofulous bones. On examining sections of these bones, we often see accumulation of osteoblasts resting against the trabeculae of the bone, and projecting into the medulla as dark vitreous bodies. These overgrown and calcified osteoblasts may gradually extend themselves into the area occupied by tubercle in the medulla, and so form in the cancellous tissue of the bone a small mass almost as hard and dense as a little bit of flint. Such a dense mass excites inflammation and necrosis of the bone surrounding it; and, until the sequestrum is removed, suppuration continues, and hence the persistent nature of not a few cases of scrofulous diseases of bone.

It is a matter of some importance to determine if tubercle of the medulla commence in a hyperplasia of the cells over a diffused area of the adenoid tissue, such as we might imagine would occur from the result of a contusion; or, if it begins at a definite point in the epithelioid lining of the capillaries, it may be from the action of a specific bacillus, and extends to the surrounding structures. It is certain that, in many sections of diseased bone, we find active proliferation of the brood-cells progressing over considerable portions of the adenoid tissue, without there being evidence of the formation of tubercle in the part. But, it must be remembered, we can only examine very thin sections of bone under the microscope; and it is impossible, therefore, to determine what may have been the condition of the medulla which has been removed. That is, tubercle may have existed in the medulla contiguous to the areas in which the cells are undergoing proliferation, but may not have been included in the section. The evidence, however, so far as it goes, is to the effect that the increased cell-growth in the adenoid tissue is the primary change, and leads to the formation of tubercle through the crowding together of round cells, which are unable to escape from the part, because the channels through which they ordinarily pass into the circulation are choked up. Supposing this to be the true pathology of the disease, it would account for the beneficial results which, experience teaches us, follow the efficient drainage of the diseased bones.

Turning from the examination of the medulla to the cartilage of bones affected by tubercular disease, we find that, at the angle between the epiphysal and articular cartilages, there is almost always evidence of abnormal action, commencing in the substance of the cartilage. We not unfrequently observe patches in the matrix of the cartilage, which appears granular, and much more transparent than the surrounding parts. As the disease advances, the cartilage-matrix in the affected part yields to the pressure of the increased cell-growth; for we find, in this transparent area, that the nuclei of the cartilage-cells proliferate rapidly. In a more advanced stage of the disease, these cells may be seen crowded together; the patch becomes opaque; the cells undergo fatty degeneration, and become disintegrated; compound cells and a fine reticulum of fibres may be seen in the part. These changes occur independently of capillaries or any other vessels, and the cartilage-cells immediately surrounding the affected area appear to be absolutely unaffected. It is not uncommon to see the cells throughout the thickness of a portion of the epiphysal cartilage broken down in this way, and a passage thus formed through the cartilage, which unites small tuberculous cavities in the medulla, contained in the diaphysis and epiphysis of the bone; so that, in the cartilage, as well as in the medulla, there seem to be well-defined areas of tissue, in which an overgrowth of cells occurs, leading to degenerative changes in the part; but, in the early stages of the disease, these areas are surrounded by perfectly healthy tissue. It is worthy of remark, that these changes most frequently take place in those parts of the growing bone which would be most liable to suffer from contusion.

At an early stage of the disease, we find the articular cartilage separated from the epiphysis, and this condition is more marked round the ligamentum teres. The separation of the articular cartilage from the bone is clearly the result of changes such as I have described, commencing in the medulla, upon which the cartilage rests. In fact, in its early stages, the so-called scrofulous disease of the hip-joint is a disease of the medulla, and of the deep or

attached surface of the cartilage forming the head of the femur; the same remark applies to the extremities of the other long bones.

In approaching the subject of the cause of tubercle in the medulla, it would have been satisfactory to have discovered evidence of the presence of a specific bacillus in the early stages of the disease; but, so far, no traces of such a definite cause of tuberculosis in bone can be discovered in the human subject. There can be no question, however, that, in the cancellous structures of the lower extremities and of the bodies of the vertebrae, but especially in the head of the femur, tubercular disease is of common occurrence, and, as in its early stages, it is most marked in the line of resistance to force transmitted along the bones; it seems reasonable to suppose that contusion of the affected part is in some way not unfrequently connected with the disease. Clinical experience lends support to this idea, for we often have a history of a fall or injury as being the assigned commencement of the disease. Healthy children, however, repeatedly meet with such accidents, but seldom suffer from scrofulous disease of the bones. On the other hand, among the children of the poor reared in our large towns it is common; these children have but few inducements to take exercise, one of the most important and essential factors in promoting sound health among the young. Moreover, both within their houses and in the courts and alleys in which they amuse themselves, they breathe a dirty atmosphere, and exist to a large extent without the necessary and salutary influence of sunlight. These children are not only pale and sickly-looking, but are characterised by weighing considerably less than children of the same age and height residing in rural districts. Light weight is synonymous with irritability of tissue, or with tissue incapable of healthy reparative changes when it has been contused or otherwise injured. In such children, the cell-hyperplasia, which is the natural consequence of shock or the contusion of tissues, in place of taking on a reparative action, has a tendency to become excessive and revert to embryonic conditions, and so the agglomeration of cells in the medulla, such as I have described, occurs. It is quite consistent with this idea to suppose that inherited tendencies may increase, and, in some cases, be the sole cause of an unstable condition of the tissues.

The treatment of the early stages of scrofulous diseases of the bones would seem to rest most securely on a correct knowledge of the nature of its causes and pathology. Nevertheless, authorities differ on this point, for Mr. Howard Marsh observes, in his lecture on Hip-Disease in Children, that "although the pathology of many cases is thus obscure, the doubt is practically of slight importance; for, in respect of treatment, whether they are scrofulous or whether they are not, one law must be rigidly observed, that is the law of absolute rest." But if, in the majority of cases of so-called disease of the hip-joint, the abnormal action commences in the medulla contained within the head of the bone, and is a local affection, surely it is opposed to reason to lay a little child affected in this way upon his back for months together in the wards of a London, or any other, hospital. It seems to me hardly possible to conceive any position in which a little patient could be placed more likely to produce unhealthy tissues. Granting that rest is necessary, it can be well applied to the part by means of a Thomas's or Sawyer's splint, and with a thick-soled boot to the sound limb, and crutches; the child can then move about with comfort. In fact, if the pathology above inculcated be correct, it stands to reason that, so far from absolute rest in bed being the main object of treatment, it is exercise in the open air, sunlight, wholesome food, proper clothing, that the patient requires. At the same time, a flexed limb may be extended, and the diseased bone kept from pressure or injury in the manner above described. A little London patient, protected in this way, and sent to one of the many convalescent homes that now exist all over the country, will, from my experience, best secure the relief or cure of the disease from which he is suffering. For the well-to-do, sea-air, and, if possible, a mountain-home, are highly to be recommended.

In most cases, cod-liver oil and iodide of iron should be administered, and continued counter-irritation, in the form of iodine paint, applied over the skin of the groin on the diseased side. The same line of treatment may be adopted to affections of the extremities of the other long bones in the early stage of the disease. If such treatment, after a prolonged trial, fails, we may have to resort to draining the bone, under antiseptic precautions. It is not, however, the object of this paper to discuss the treatment of cases in which the disease has advanced to that stage where the joint and surrounding structures have become extremely compromised. The affection we have been considering is a chronic one. Its early stages

are well marked, and it is then we may, by judicious treatment, confidently hope to avert the serious consequences which threaten those suffering from this disease.

BRITISH MEDICAL ASSOCIATION.

FIFTY-FIRST ANNUAL MEETING.

PROCEEDINGS OF SECTIONS.

AN ADDRESS ON CHRONIC INFLAMMATION OF BONE.

Read in the Section of Pathology.

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(Concluded from page 1116.)

TURNING, now, to the cranial bones, several new causes of enlargement present themselves for consideration; and these I now propose to investigate.

Localised thickenings of the cranial bones may, as in others, be due to syphilis and struma, but neither of these produces an uniform enlargement. These uniformly thickened skulls, examples of which occur in almost every museum, are still ill-understood.

Several distinct appearances are presented by these specimens.

1. In some, the outer and inner tables are distinct, compact, and of no great thickness; the diploë is well-defined, with a regular open texture. It is, indeed, normal in all respects, except for its great thickness. Here is an example of this form, which, I think you will see differs from all the others. It is part of a skull of a man aged 30, who committed suicide, many years ago, at the London Hospital. A portion of the same is in the museum of the College of Surgeons.

Writing on hypertrophy of bone, Sir J. Paget attributes to concentric hypertrophy, from shrinking of the brain, many thick skulls.¹ He does not describe the appearances in such cases, but gives an illustration—which I pass round—taken from a similar section of

Fig. 7.

this same skull, so that we may take it as representing his idea of the change. He describes it thus: "The thickening of the skull is effected by the gradual remodelling of the inner table and diploë of the bones of the vault; so that, although the exterior of the skull may retain its natural form and size, the inner table grows more and more inwards, as if shrinking towards the retiring and shrinking brain—not thickening, but simply removing from the outer table, and leaving a wider space filled with healthy diploë."

He gives another case of a young lady, intellectually feeble, whose cranial cavity was singularly contracted, the skull having adapted itself to an imperfectly grown brain by a hypertrophy of its diploë, while externally the skull was not below the average female size. It seems to me that this case offers a different explanation of itself, and one that may be applied to the specimen I have handed round, and of all thick skulls presenting similar characters.

So far as I can gather, there is no appearance which suggests the present existence or progress of this "concentric hypertrophy." I am rather inclined, on the contrary, to hold that the shrinking brain is replaced by cerebro-spinal fluid, an appearance frequently observed, and that the thickening in such skulls as these takes place during

¹ On this subject of shrinking brain, with alteration in the bone, I may quote a remark from Billroth, who says "that the filling up of the diploë with bony substance is so common in advancing age as to be scarcely pathological." This seems to me to need confirmation. He does not state if, at the same time, the bone is thickened.

the period of development only, and that it is not a true example of accommodating hypertrophy. The capacity of the cranial cavity is, I imagine, determined—as indeed Paget has suggested—by the growth of the brain, or in some perhaps by an early ossification of the bones with closure of sutures. Admitting this, two explanations suggest themselves.

First, after the lateral increase, or increase in area, has been checked, the process of growth continues and leads to a thickening of the bone; the new material being added to the surface instead of to the margin. In this way, the size of the head may not be diminished.

Secondly, it is not unlikely that rickets may produce this appearance. In this disease, as is well known, the cranium becomes enlarged, often with remarkable elevations round the centres of development, producing the so-called natiform skull. A section through such a skull of an adult, in the College of Surgeons, shows exactly the appearances presented by this piece of bone and by Paget's drawing. I have not met with any description of an adult rachitic skull where enlargement had persisted. Sir J. Paget's description applies, without doubt, to the early stages, specimens of which exist in the College museum, and are described as "very light, almost friable, not porous, but like fine cloth or felt."

I must not omit to mention here that M. Parrot has attributed these bossy or natiform skulls to congenital syphilis; but many others hold the opposite view, and my own observations amongst living children lead me to decide in favour of rickets.

It is worthy of more than passing remark, that in the skull alone do we find a permanent increase in size in rickets, though, of course, not in all cases. It suggests some difference between the subsequent modelling process in the two parts of the skeleton.

2. In another form, the bone is porous throughout the inner and outer tables, as much so as the intervening substance. Irregular gaps occur in the section, indicating possibly the situation of the diploë. Many have the mortar-like appearance described by Mr. Durham, while others are more dense. Probably, as has been suggested, these two are but stages of one process. Mr. Durham, however, regards the two as distinct, the porous form—at least some of them—he considers examples of cured osteomalacia; the denser ones as rachitic. The majority of these skulls are no doubt examples of osteitis deformans, in which disease, as is well known, the bones are uniformly thickened. No trace of the compact tissue is left at all. Several of these specimens illustrate this appearance.

Another disease said to lead to the production of this appearance is osteomalacia. This has generally been stated since the publication of Mr. Durham's paper in the *Guy's Hospital Reports* for 1864, and since Mr. Solly's communication to volume xxvii of the *Medico-Chirurgical Transactions*. Paget alludes to it upon the authority of the above writers. I have examined, with regard to this point, the specimens upon which the statement of enlargement has been based, and I cannot but think that a different and truer explanation can be given, and indeed, that in this malady there is no increase in the thickness of the skull. Here is Mr. Durham's specimen. The calvaria is seen to be much thicker than normal, and to show all the appearances described in osteomalacia. It is by no means so thick as in the dry specimens just exhibited, and it is far more porous.

Mr. Solly's specimen may be seen in the museum at St. Thomas's Hospital. It is a dry preparation of about the same thickness as this one, and shows the same degree of porosity.

In the same bottle with Mr. Durham's specimen are portions of several other bones from the same patient, none of which show any thickening, and it will be found to be true that in no other bone does an increase in size take place. Surely, therefore, unless we admit that the effects of disease are entirely different in the cranial bones from what they are in the long bones, this is almost sufficient to negative the conclusion of enlargement.

Again, in some well-marked examples of the disease, the cranial vault is, if anything, thinner than usual; at least it is not increased in thickness, though showing well the characteristic changes. Mr. Solly records one such case, and explains the absence of thickening by the short duration, which was four and a half years, while that in which the enlargement occurred had existed eight years; and he says he has no doubt that, had his second patient lived, an increase would have taken place. In opposition to this opinion, it must be said that, though the skull was not thick, the other bones exhibited the softening effects of the malady in an extreme degree, the cranial bones themselves being deeply affected.

Mr. Durham's patient in whom the skull was thick had been ill but three years; so that, upon the ground of duration of the disease,