THE ETIOLOGY OF CERTAIN LESIONS AT THE HIP-JOINT.

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I PROPOSE to correlate some recent researches in the anatomy of the parts about the hip-joint, with special reference to their embryology, and to indicate the practical application of this by illustrative pathological conditions. In a paper such as this it is impossible to deal with all the various possible lesions, but I will mention a few of the more important from the standpoint of how they occur, giving an outline of the necessary treatment; this is better than taking a list of diseases and discussing them seriatim.

The first sign of the limbs in the developing foetus is seen at the third week, when the four limb buds appear; in the lower extremities the bud consists of ten lateral processes of the corresponding somites of mesoblast. As the processes grow out they as it were drag with them corresponding portions of the primitive neuroblastic neural tube, which ultimately form the spinal nerves, and in this way the twelfth dorsal to the fourth sacral nerves are formed. Recent work has shown that the lateral processes of the primitive segments consist of two separate parts—a central core of mesoblast, with a covering of mesenchyme. This mesenchyme is probably the more primitive body tissue in which stiffening takes place to form the skeleton; the original idea of mesoblastic uniformity is being gradually abandoned. As the limb buds grow out they lie at first parallel to the trunk, but by the sixth week of intrauterine life the lower limbs have assumed a position at right angles to the trunk, and some differentiation into their three mature portions has occurred. While this is taking place condensation of mesoblastic cells occurs in the centre of the bud, starting first in the proximal part of the free limb and in the tissues carrying it; these ultimately form the buried portions of the limb and also the limb girdle. At quite an early stage of condensation in the case of the lower limb the femur assumes a shape approaching in some measure its adult form. This shape is determined apparently (Hume) by the deposition on the femur of a limiting layer of cells, derived from the surrounding mesenchyme, this layer ultimately forming the periosteum of the bone. At the ends of the bars of mesoblast the covering layer is usually very thin, to allow, apparently, for the changes which occur later on in and around the epiphyseal line. When the layer is actually deficient, leakage of cells may ensue, which suggests the real origin of exostoses—comparable with the leakage of cells from beneath the periosteum of an adult bone at muscle or tendon insertions, where spurs and other outgrowths sometimes occur.

While condensation of the buried and free limb is taking place rotation has commenced in an inward and backward direction, so that the future flexor surface from being ventral becomes dorsal. When it is remembered how very fragile the foetal cells must be at this stage, in spite of the term "condensation" being applied to them, the cause of congenital malformation of the limbs or spine becomes more comprehensible. It is fairly easy to surmise the cause of congenital dislocation of the hip, the "central splint" of cells being severed where the free limb is attached to the trunk, when the limb buds rotate.

To carry explanations too far leads to difficulty, but it is interesting to remember in the case in point that an acetabulum of a kind is formed, in spite of the fact that the head of the femur has never been in its proper position. That circumstances will affect morphological change is evidenced by the more rational form the acetabulum assumes after successful treatment for congenital dislocation of the hip. All this evidence tends to support a theory of developmental anomaly as the cause of this condition, rather than of obstetrical accidents, etc.; but I suppose the explanation is of academic rather than of practical importance.

Anatomical landmarks are the most useful guides in classifying congenital dislocation of the hip, which may

be of any degree, ranging from a slight subluxation of the joint to a complete derangement, with the head of the femur lying anywhere from the dorsum ilii to the obturator foramen. The most common place is situated on the dorsum ilii above the acetabulum, where it is probably driven by the weight of the body falling on an already dislocated joint; that the capsule stretches with the excursion of the bone is what would be expected from the developmental evidence already considered, the capsule being mesenchymal in origin and really part of the limb bud.

It is unnecessary to enumerate the cardinal symptoms and signs of this condition, the most satisfactory diagnostic information being that obtained by x-ray photographs. Before passing to the rationale of hip-joint symptomatology in general, however, a note about treatment may be permitted. The earlier the condition is diagnosed the more successful is the treatment. This is now being practically standardized as reduction of the dislocation by manipulation and fixation of the limb, fully flexed and everted in a plaster case; from this position the limb is gradually released during a period of nine months or so. The form of treatment by clamps and splints, advocated on the Continent, has been abandoned.

The hip-joint, from its very nature, is fairly stable; in its resting position it is locked by the action of the ilio-femoral ligament, or Y-shaped ligament of Bigelow, which is rendered tense by full extension and slight internal rotation of the femur, muscular strain thereby being reduced to a minimum. When the joint has to be moved again it is unlocked by slight internal rotation of the femur, and the muscles take the strain. Consequently, when any condition in the hip-joint itself, or closely adjacent to it, causes the slightest effusion, the joint capsule and the ligaments, especially the ligamentum teres, must be relaxed, and the classical flexion, abduction, and external rotation occur. External rotation beyond that which occurs with an extended locked joint is made possible by the fact that flexion has occurred first. Muscle ache on standing or walking, apart from joint pain, is thus explained, while the degree of limp is determined by the extent of the deformity and any change in the upper end of the femur which may be present. The principal nerve supply to the hip-joint is by the obturator, the motor nerve of the adductor group of muscles, and, should it be irritated for more than a short time, the adductors pass into reflex spasm, causing the secondary deformities of hip-joint disease—namely, flexion and adduction. Internal rotation follows to a greater or less degree, in spite of the normal action of the adductors, owing to the fact that the patient lies on the sound side and gravity comes into operation.

In addition to the foregoing, which may be termed the gross signs of hip-joint disease, there is another equally important group of manifestations, not so obvious, perhaps, which depend on some anatomical change in the head or neck of the femur, the consequent deformities being best elicited by means of a tape-measure and a subsequent x-ray examination. The normal angle of the neck of the femur is 125 degrees, and the neck has a slight forward inclination of 12 degrees; it follows, therefore, that any alteration in the angle of the neck, or any bony changes in the head or neck, will be reflected in the total length of the limb, and in the relation of the tip of the great trochanter to Nélaton's line.

The question then arises how best to distinguish between the various possible conditions which may be present when one is faced with a case suggesting trouble in the region of the hip-joint. The age of the patient is of value, especially in such conditions as congenital dislocation of the hip, which is noticed first in young children, and adolescent coxa vara in children at or just after puberty; for the rest it is not of diagnostic value, at any rate at first. Following this a history is elicited which may bring out the story of an injury, even some time previously, which broadly suggests the possibility of one of the epiphyseal group of diseases, such as pseudo-coxalgia or traumatic coxa vara. A simple epiphyseal strain, with its consequent pain and limping, usually settles down after about ten days' rest in bed, but, failing this, an x-ray examination will often help to exclude more serious lesions. Careful physical examination will, of course, elicit difference in the length of the

limbs, as well as bony displacements and deep-seated tenderness, the possible conditions always being borne in mind. This physical examination should always be supplemented by x-ray photography, which it may be necessary to repeat at intervals; it is only since the advent of radiography that any really exact knowledge of the state of affairs has been possible.

Reference to the head of the femur prompts me to say a few words about its blood supply. The head receives a variable branch of the internal circumflex artery through the ligamentum teres, while the neck is supplied by numerous tiny twigs via the retinaculae in the capsule of the hip-joint from the digital anastomosis, which also sends a branch direct to the base of the neck. Recently some German anatomists have traced out the course of these rather feeble vessels in the bone itself, demonstrating that they run remarkably straight courses to form a network at the epiphyseal line. From this fact can be explained, perhaps, the changes which occur so frequently in the young when the epiphysis is moved relatively to the shaft of the bone, as by a fall. As the result of this injury there is thrombosis of the arterial whorl at the epiphysis; this is a possible explanation of the pseudo-coxalgia which crops up from time to time in hip-joints previously the seat of congenital dislocation which have been successfully reduced but too violently.

Epiphyseal strain, when it develops in the hip-joints of children following an injury, constitutes a definite threat of future trouble if not properly treated by rest and so forth, since cases occurring presumably without infection may end in a traumatic coxa vara; should a haematogenous infection be superadded pseudo-coxalgia will most certainly occur. Fortunately or unfortunately for this infective theory of the cause of pseudo-coxalgia, most careful cultures have been made in definite cases of the condition with only negative results, but this does not prove that the condition was always such. When cultures have been successful a *Staphylococcus aureus* of low virulence has been obtained, and, supposedly, a suppurative epiphysitis would have supervened had the micro-organism been more virile. When it is considered how frequently healthy children knock themselves about with no ill result the question arises why only some should suffer, especially as the upper epiphysis of the femur is always subjected to a sheering strain. Of late the importance of calcium metabolism and its relation to infections generally has attracted much attention; this is of special importance when dealing with bony conditions. Phosphorus and calcium taken by the mouth as food can only be metabolized in the presence of accessory food substances—the vitamins A and D—balance being preserved between these two elements and the excess being excreted. This is not the whole story, in the light of Collip's researches into the functions of the parathyroid bodies, since he found that, quite apart from vitamins, the normal level of blood calcium depended on the presence of the hormone of these bodies in the circulation.

"Late rickets" is a convenient label to apply to those epiphyseal changes in patients who are too old for this disease in its generally accepted form. Is it not more desirable to recast the whole story of calcium metabolism, and to presume, at any rate for the moment, that rickets is one indication of a general metabolic dysfunction which may be manifest at any time, at any rate in the early years of life? We may go further and consider a condition such as urticaria, which is by no means infrequently present in cases of rickety and adolescent coxa vara. Does not this suggest the importance of general measures as well as local splinting, etc., in the treatment of these bone joint lesions?

In days gone by cod-liver oil was the standby in all these nutritional diseases, but of late the results have not been uniformly successful, the reason being shown to rest in its variable vitamin content. When cases of bone disease are treated by sunlight, the ultra-violet light of which synthesizes the vitamin in the skin, remarkable results have been obtained, especially in conditions such as pseudo-coxalgia, which is often most difficult to distinguish from tubercle and has been treated similarly.

Pseudo-coxalgia is essentially an epiphyseal disease, appearing first as a destructive process, quickly continuing

until a natural attempt at arrest brings about hypertrophic growth of bone, frequently very soft, with the result that the top of the femur is mushroomed and coxa plana results. The Americans describe coxa plana, due to this cause, as resembling the buffer of a railroad car rather than a mushroom, the top of which is really too big in proportion to its stem to be an exact simile. Treatment in the early stages must be expectant, with rest and massage at definite times; later deformities may require adjustment.

Mention of the femoral epiphysis leads on to another condition in which it is affected—namely, certain types of coxa vara. It is impossible in a short paper to enter into a full discussion of this disease, but as a rule it is bilateral, and angulation occurs at one of three points—epiphyseal, cervical, or basal. As regards the types, these are fairly well defined, the most interesting being the infantile, or cervical, which x-ray evidence definitely indicates as being of developmental origin. So far as can be ascertained, it appears that a diaphyseal spur develops which has a separate centre of ossification between the epiphysis and diaphysis proper, forming a triangular wedge of bone (Elmslie). The cementing cells between the portions of bone are weakened and give way, there being two layers instead of the usual one, as a result of the strain of the body weight. In the adolescent type there is no diaphyseal spur, and the slipping of the epiphysis is due to the weakening of the cementing cells from injury to the blood supply, and presumably to faulty calcium metabolism at the place, the changes not being comparable with those of true rickets. The onset is marked by pain and limping, the limb being flexed, adducted, and everted. Treatment aims at correcting the deformity by suitable extension; it should be continued until skiagrams show that buttressing by new bone has taken place.

In a purposely short paper it is impossible to go fully into all the conditions about the hip-joint; those mentioned have been discussed very broadly to avoid dogmatism.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SCLEREMA NEONATORUM.

THE case of an infant who undoubtedly suffered from this unusual complaint, and is now alive and in good health, seems sufficiently interesting to be recorded.

The mother, aged 36, who had a history of scarlet and rheumatic fevers, indigestion apparently of hepatic origin, and several attacks of urticaria, was in poor health up to the time of her confinement on December 16th, 1929. She had been married eighteen months before she became pregnant. She does not appear to have had sufficient exercise. Her appetite was small, but she enjoyed fats in the form of butter and cream. She had mitral disease, which at first caused little inconvenience, and she complained of various "rheumatic" pains, induced by climatic changes. The father, a meagre, C 3-looking individual, has suffered for many years from chronic nasal catarrh.

The mother commenced to vomit as soon as she became pregnant, and continued, on and off, during pregnancy. She took "milk of magnesia" continuously, and had some difficulty with constipation. She lost weight, and had several attacks of bronchitis and night sweats. By the seventh month she complained of palpitation, swelling of the ankles, morning and evening, and of attacks of fainting. Her urine, which was tested frequently, never contained any albumin, but phosphates and urates were present in excess of normal. For the last two weeks prior to her confinement her abdomen felt "senseless." After being nine hours in labour she was given chloroform and delivered with forceps of a female child, which weighed 5½ lb. She recovered from the confinement fairly well, but had several attacks of vomiting, and the involution of the uterus was delayed. She had no milk till the ninth day.

Delivery was hastened in view of the mother's cardiac condition. On the cord, which was unusually thick, there was, about one and a quarter inches from the umbilicus, what proved to be a haematoma, two and a quarter inches long, dipping into Wharton's jelly. The placenta appeared to be normal. The child seemed to be heavier than it actually was, and its skin was hard and stiff, preventing it from moving its limbs, but not interfering with sucking. The hardness, which apparently involved the whole skin, seemed most pronounced and remained longest over the buttocks, which were dark blue in colour. At no time did the skin pit on