NORMAL AND ABNORMAL GROWTH AT THE UPPER END OF THE FEMUR

J. D. MORGAN and E. W. SOMERVILLE, OXFORD, ENGLAND

From the Nuffield Orthopaedic Centre, Oxford

It is the purpose of this paper to describe and illustrate how alterations in the fertility and position of the primary and secondary epiphyses may affect the shape and length of the upper end of the femur.

It is suggested that under normal conditions of growth two processes are involved: firstly, ossification of parts of the cartilaginous anlage; and secondly, growth of the whole. Ossification takes place in each area in which a blood supply is present: namely, 1) in the shaft—periosteal arteries and nutrient artery; 2) in the metaphysis—nutrient arteries; and 3) in the epiphysis—epiphysial arteries. The shaft with its double blood supply is ossified long before the epiphysis.

Growth of the whole—Growth in length takes place at both ends of the bone in the area known as the epiphysial plate. Growth in width follows the increase in diameter of the epiphysis.

DEVELOPMENT OF THE FEMUR

At birth the shaft is a calcified tube containing the actively proliferating nutrient vessels and closed at its ends by the cartilaginous epiphyses. Each epiphysis has one or more vascular islands—the ossific nucleus—but the epiphysial "plate" is not yet fully formed.

As age advances the mature epiphysial plates are formed—each in its own time and place.

A mature plate consists of a circumscribed area in which the cartilage cells are arranged in columns, the whole being sandwiched between the bony end plates of epiphysis and metaphysis. In this situation the cells are subjected to the growth pressure and by their
interposition they separate the vascular territories of the metaphysis and epiphysis. Multiplication of these cartilage cells leads to growth in length of the bone and this growth takes place in the direction of the columns. At the lower end of the femur this direction is roughly in the line of the shaft of the bone.

Growth from the lower plate commences at an early age by pushing before it the almost flat side of the B-shaped lower epiphysis, adding length to the end of the bone as from an indented disc.

At the upper end of the femur the epiphysial plate is more complex. At first it appears to stretch across the upper end of the bone as a crescentic line (Fig. 1), but this effect is soon modified by the appearance of a mature portion of the plate on the medial side opposite to the newly formed and vascular capital epiphysis. Vigorous growth by this mature part of the crescent leads to an elongation of the femoral neck. Because of this early and unopposed spur of growth the femoral neck at this age is more vertical than in the adult, a normal condition known as infantile coxa valga.

Such a state of affairs does not last long, probably because of the wakening activity of the hip abductors in walking, for the lateral portion of the crescentic pre-plate now becomes organised, after the appearance of a new vascular "island" in contact with it. This is the epiphysis of the greater trochanter.

Thus there are two separate areas at the upper end of the bone where rapid growth is taking place. Increase in length is therefore, as it were, a resultant of these two forces interacting to determine the angle of the neck and the length of the shaft.

The first effect of the increasing growth activity of the lateral part of the plate is a marked lessening of the valgus angle of the neck. This is accomplished by increasing growth of the lateral part of the shaft. It can readily be imagined, therefore, that anything which interferes with this sequence of events will lead to deformity of the upper end of the bone and almost always to some permanent shortening of the thigh.

It is said that only 30 per cent of the length of the femur is produced by the upper epiphysial plate. But, as most of this growth takes place in the first few years of life, any interference with it then will cause the greatest deformity. It is interesting to review the progress of such abnormalities of the upper end of the femur as congenital coxa vara in the light of this concept of its longitudinal growth. Following the progress of such a case by serial radiographs may provide ideas regarding its etiology and may suggest some effective mode of treatment.

**PATHOLOGICAL CONSIDERATIONS**

**Congenital coxa vara**—Figure 2 shows such a case. The radiograph was taken on the day of birth. Eight months later the radiograph (Fig. 3) showed that the ossific nucleus of the femoral head was just visible, placed centrally in an acetabulum which was quite well developed. It also showed a dark shadow appearing as the "ghost" of the missing upper end of the femur. The relationship of the "ghost" to the ossified shaft suggested fracture of the shaft of the femur. In fact the mother did volunteer the information that in the eighth month of pregnancy she fell heavily on to her abdomen when going upstairs.

The appearance of the radiograph (Fig. 3) suggested that the upper end of the femur was present but still cartilaginous. This was later confirmed by arthrography (Fig. 4) which showed a normal head, neck and acetabulum, and by the finding at subsequent exploration of trochanters and of the upper end of the femoral shaft. If a fracture had in fact occurred at the time of the accident, when the femoral shaft would be as much cartilage as bone, it would have taken place either through the cartilaginous shaft or at the place where cartilage and bone joined. Whichever was the case the rhythm of ossification previously described—which we believe to be essential for normal growth in length—would have been interrupted. The failure of the spread of ossification from below would have prevented the development of a normal epiphysial plate, so that if the upper end of the femur were eventually to undergo
FIG. 2
Radiograph of child on the day of birth, showing typical congenital coxa vara.

FIG. 3
Figure 3—Same child eight months later. The ossific nucleus of the femoral head is now visible in the acetabulum and the upper end of the femur is appearing as a "ghost." Its relationship to the shaft is similar to that in cases of birth fracture.

FIG. 4
Figure 4—An arthrogram of this hip outlines the cartilaginous head and neck.
Figure 5—At a later stage the capital nucleus is extending into the neck and irregular nuclei are developing.

Figure 6—The irregular nuclei are fusing together.

Figure 7—A similar case at a later stage. Figure 8—The same case after the varus deformity was corrected by osteotomy. The whole of the upper end of the femur has fused into one solid mass, without the property of growth in length.
ossification it could only do so in an irregular manner. Serial radiographs showed that patchy ossification occurred in the cartilaginous part which eventually fused into a solid mass which, in its turn, fused with the ossific nuclei in the head and trochanter (Figs. 5 and 6). Thus, the upper end of the femur became one solid mass of bone without a metaphysis, and without hope of longitudinal growth. This solid upper end of the femur will eventually join the shaft spontaneously; in this case it was assisted by an osteotomy (Figs. 7 and 8).

FIG. 9
Congenital coxa vara. In these hips the defect is in the femoral neck. The trochanteric part of the epiphysial plate is intact so that growth in length can continue here normally. Note the triangular piece of bone.

FIG. 10
Same case as Figure 9. The femoral head has got left behind. The "defect" has closed spontaneously.
The lesion causing the interruption in ossification just described was below the trochanter but it may be in the neck. In that case the ossification of the shaft will spread upwards forming a normal epiphysis with the greater trochanter. It cannot, however, extend into the neck, so that no metaphysis will develop for the head, no growth will take place, and the head will get left behind. In this instance a gross and increasing coxa vara results (Figs. 9 and 10).

There are certain instructive features about this lesion. Trueta (1957) has demonstrated the blood supply of the upper end of the femur by the injection method. The growing head of the femur derives its blood supply from two sources: the more lateral part of the head from a leash of vessels on the upper surface of the neck; and the medial part by a smaller leash on the under surface. This lower part of the head is mainly proximal to the epiphysial line but a small portion lies distal to it. Thus the ossific nucleus of the head, with a small triangular portion of the neck adjacent to the medial part of the head, will have a blood supply of its own which is unaffected by the lesion in the neck. The whole segment may ossify quite normally, giving the appearance of a normal femoral epiphysis and a triangular piece of bone (Fig. 9).

These observations on the etiology of this condition suggest certain points which may be of value in treatment. There are the two types of coxa vara. In the first the lesion is distal to the trochanter, and in the second it is proximal to it. There are two problems: firstly, the deformity. This is static in the first type throughout the period of growth and is progressive in the second type. Secondly, the failure of normal growth in length: in the first type this will be extreme, there being no upper metaphysis or plate at all, but in the second it will be minimal because the trochanteric metaphysis and portion of plate will continue to grow uninterruptedly.

Whereas the deformity presents no problem (it can be corrected easily by osteotomy) the problem of growth may be insuperable. Nevertheless, there are certain possibilities worth considering even with this.

If ossification could be made to spread across the defect from the ossified shaft into the cartilaginous part, so that it might continue upwards in an orderly manner before the

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**FIG. 11**
Operative findings in case shown in Figures 2 to 6. The fibrous union between the bony shaft and the cartilaginous upper end can be seen.
disorganised ossification had begun, it might be possible for an epiphysial plate to form and for growth in length to be restored. With this possibility in mind we explored the upper end of the femur in the case illustrated in Figure 2. We found the cartilaginous upper end of the femur joined to the ossified shaft by a fibrous union at which movement could take place (Fig. 11). An osteotomy was carried out close to the end of the bone, a drill was passed across the fibrous union from the bone into the cartilaginous mass and the deformity was corrected at the osteotomy. The procedure was unsuccessful.

The cause of failure may have been the great difficulty of maintaining apposition at the osteotomy in such a short, fat leg; it may have been that at ten months of age we were already too late; or it may have been that we were attempting the impossible. But in spite of this failure we feel that further attempts should be made to get bone to fuse to cartilage in order that normal ossification may progress from the bone into the cartilage.

We do not wish to convey the impression that we believe that congenital coxa vara is always the result of trauma such as recorded here, but rather to suggest that any lesion that will in some way prevent the upward spread of ossification will cause this condition. The spread of ossification is dependent entirely on blood supply. If the blood supply is interrupted the spread will cease. Trauma is only one way by which the blood supply may be stopped.

Other conditions affecting the upper end of the femur—Less spectacular changes in the upper femoral epiphysis may follow paralysis of the abductors, as commonly occurs after anterior poliomyelitis, the loss of tension in the trochanteric epiphysis tending to close the lateral portion of the plate so that the medial or subcapital part of the plate overgrows with resultant coxa valga (Fig. 12).

In most patients with Perthes' disease deformity occurs with little if any shortening of the thigh. In this condition revascularisation of the ischaemic capital epiphysis leads to the maturity of a larger piece of the crescentic pre-plate than normal, so that the wide incongruous head is supported on a short, thick neck, while at the unaffected trochanteric portion of the plate growth continues steadily and is thus able to maintain the length of the shaft (Fig. 13).

Some of the most severe and bizarre abnormalities of growth at the upper end of the femur follow acute infections of that region in early life. In such cases the infective process destroys part or whole of the capital anlage or epiphysis and as a result either the plate is never formed or, if present, it is closed. In some cases only the medial (subcapital) part of the plate is destroyed. When this happens no head or neck is formed, but the length of the femoral shaft is maintained undisturbed by the activity of the undamaged lateral (trochanteric) part of the plate (Fig. 14). In other cases the damage to the subcapital epiphysis is incomplete and the viable portions of the plate grow a long, thin and irregular neck which may even have a head at its summit (Fig. 15). Lastly.

**FIG. 12**
Radiograph of the femora after poliomyelitis, showing shortening of the right femur and coxa valga.
Perthes' disease of left hip, showing short, thick neck with normal trochanteric epiphysis.

Figure 14—Infection has destroyed the femoral head; the neck is absent but the length of the shaft is normal. Figure 15—Infection has injured the whole of the upper epiphysis. Irregular neck and trochanter formed with much shortening.
partial subcapital damage may distort the head, because one part of the plate is closed and another still open (Figs. 16 and 17).

Finally, it might perhaps be true to describe adolescent coxa vara as the swan song of the upper femoral plate because, goaded by hormones, the epiphysis and plate slip away.

SUMMARY
1. A brief description is given of normal epiphysial growth of the human femur.
2. Some ways in which abnormality of the growth plates may affect the shape and length of the human femur are described.
3. The influence of the blood supply on growth is discussed with particular reference to the etiology and treatment of congenital coxa vara.

We would like to thank our colleagues for allowing us access to this material, and Professor J. Trueta for his enthusiastic and unfailing help.

REFERENCE