Coxa plana--the fate of the physis

D Keret, MH Harrison, NM Clarke and DJ Hall


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Coxa Plana — The Fate of the Physis


From the Royal Orthopaedic Hospital, Birmingham

ABSTRACT: Eighty patients who had unilateral coxa plana and who had been treated conservatively were followed to a mean age of sixteen and one-quarter years, and their cases were reviewed for radiographic evidence of a disturbance of the femoral capital growth plate. This physeal involvement was inferred by the presence of one or more of five findings: premature physeal closure, overgrowth of the greater trochanter, change in physeal shape, lateral protrusion of the capital nucleus, and medial bowing of the femoral neck. Premature physeal closure, which was more common in the girls than in the boys, occurred in 25 per cent of the affected femoral heads, and 90 per cent of the patients showed some interference with normal physeal growth in the affected femoral head. A direct correlation was found between the severity of the physeal involvement and the ultimate deformity of the femoral head. The possible causes of interference with physeal growth and damage are discussed.

The data on which our understanding of coxa plana is based are essentially radiographic. The changes affect predominantly the femoral neck, metaphysis, and acetabulum, in addition to the principal lesion in the capital epiphysis. Edgren, in an extensive survey of coxa plana, clarified the significance of growth arrest in the production of deformity of the head and neck of the femur; Mindell and Sherman as well as Kamhi and MacEwen reported confirmatory studies. Both Trias and Barnes showed that premature fusion of the femoral capital physis could occur even if containment of the femoral head had been achieved by femoral osteotomy. Langenskiöld and Salenius corrected the trochanteric deformity that followed premature fusion in thirty-two patients by epiphysodesis of the greater trochanter. In a previous study of thirty-one young patients who had a painful hip after coxa plana, two of us were surprised to find that fifteen hips had premature fusion of the femoral capital physis. All except one of these children in whom premature fusion developed had been treated by methods that would not provide containment of the femoral head (such as observation or a caliper). We suspected that this omission might have resulted in premature fusion of the epiphysis. Therefore, we decided to survey the degree and frequency of physeal involvement in a group of children whose coxa plana had been treated by conservative con-

![Histogram showing the age at diagnosis of eighty children with coxa plana. The hatched areas indicate the age at diagnosis of the twenty children who had premature physeal closure.](image)

**FIG. 1**

Observations

All of the patients who were selected for study had had unilateral coxa plana. They had been treated conservatively by the Birmingham containment splint* and either had reached the age of fifteen, if male, or fourteen, if female; had reached skeletal maturity by the time of final follow-up; or had had premature closure of the physis of the affected

* 4a Mapu Street, Afula, Israel.
† 25 Frederick Road, Edgbaston, Birmingham B15 1JN, England.
‡ Royal Orthopaedic Hospital, The Woodlands, Bristol Road South, Northfield, Birmingham B31 2AP, England. Please address reprint requests to Mr. Clarke.
The rate of increase in standing height of one patient (Case 17, Table I) plotted on the standard chart of Tanner et al. The broken and solid lines show average growth rates for normal boys (ninety-seventh, ninetieth, seventy-fifth, fiftieth, twenty-fifth, tenth, and third percentiles). The shaded curves show the average growth rates that can be expected in individual children during the adolescent growth spurt. Individual curves have been superimposed so that their peak height velocities coincide, thereby avoiding the smoothing effect of cross-sectional averages. Premature fusion of the affected capital femoral epiphysis was first observed at the time of peak height velocity, when a further two years' worth of growth could be expected on the unaffected side.

We sought evidence that physeal activity had been interfered with consequent to the coxa plana by the presence of at least one of five radiographic findings.

**Premature Physeal Closure**

Our evidence here was replacement of at least 50 per cent of the physis by bone trabeculae so that continuity was established between the epiphysis and the metaphysis at least one year before similar physiological changes took place in the unaffected hip. Twenty of the eighty patients showed premature physeal closure according to this criterion. The closure commenced at a mean age of twelve years and three months (range, thirteen to twenty-seven years). This group of patients was a selected one in three respects. Firstly, during the years when this cohort was treated most children who were more than ten years old were deemed unsuitable for splintage therapy. Secondly, bilateral or mild disease (Catterall Group I) was not treated with the Birmingham splint. Thirdly, children who were lost to follow-up after treatment was completed could not be included in the series.

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Premature fusion observed

A: The lengths of the lower limbs (in centimeters) as measured by a tapemeasure for one patient (Case 17, Table I), whose right hip was treated for coxa plana by the Birmingham orthosis. B: Tibial lengths were measured by a Herpenden anthropometer. The affected right lower limb was 0.5 centimeter short at diagnosis and was two centimeters short at the end of the orthotic treatment. The shortening was equally distributed between the tibia and femur. After removal of the orthosis, a catch-up in growth of about 0.5 centimeter occurred in the right tibia. This catch-up, shown as a greater increase in the slope of the curve for the right tibia as compared with that for the left one, is best seen from the ages of twelve and one-half to fourteen and one-half. C: The catch-up masked an increase of about 1.5 centimeters in femoral shortening during that two-year interval, due to premature fusion of the capital physis. After fusion was observed radiographically on the affected side at the age of thirteen years, the discrepancy increased because of continuing growth of the unaffected contralateral capital physis.

Fig. 3

Fig. 4

Anteroposterior radiograph of the proximal end of a normal femur, showing the shape of the physis and the height of the greater trochanter.
and hormonal development. Such estimates could only be approximations, because anthropometry did not always synchronize with the radiographic findings. It appears that premature fusion occurred at the time of the adolescent growth spurt (Fig. 2) — later in the spurt in girls and earlier in boys. The menarche in girls and voice change in boys occurred almost at the same time as the observed premature fusion. Our measurements of lower-limb lengths showed that the increase in limb-length discrepancy often followed premature fusion of the capital physis (Fig. 3).

### Overgrowth of the Greater Trochanter

Severe trochanteric overgrowth is obvious radiographically but is difficult to quantitate. For that purpose, we measured the distance between the tip of the greater trochanter and the summit of the physis, the physeal-trochanteric distance, in preference to a measurement from the summit of the femoral head to the trochanteric tip. We did

![Anteroposterior radiograph of the proximal end of a femur, showing + trochanteric elevation and ++ curvature of the physis.](image)
Anteroposterior radiograph of the proximal end of a femur showing +++ elevation of the greater trochanter.

this because flattening of the femoral head consequent to the unilateral coxa plana would reduce the articulotrochanteric distance, and therefore a shorter distance would not necessarily indicate a growth-plate disturbance. We made a visual comparison of the physeal-trochanteric distances of both hips seen on the same radiograph. If the distance was the same in both hips, then the affected hip was considered to be normal. Slight (+) elevation of the trochanter meant that its tip did not reach the level of the physis (Fig. 5). If the tip of the greater trochanter reached or passed just rostral to the physis, then elevation was graded ++, and if the trochanter rose rostrally. In fourteen of the twenty hips that had had premature fusion of the capital physis, important overgrowth of the greater trochanter was evident (Table I).

Alteration of Physeal Shape

The normal appearance of the capital physis is an irregular linear translucency, two to three millimeters wide, with a slight inferior concavity at each edge. Its appearance is similar on both anteroposterior (Fig. 4) and lateral radiographs. We observed that in many femoral heads previously affected by coxa plana there was a more curved appearance on both radiographs.

We considered a physis to be normal when the curvature of the physis was the same as that in the unaffected hip and as ++, +++ (Figs. 5 and 9), and ++++ (Fig. 7) as it became increasingly curved. The physis retained its normal curves on anteroposterior and lateral radiographs in only eight of the eighty femoral heads. These eight patients and seven others in whom the shape of the physis was normal on only one radiographic projection, and in one such femoral head the physeal-trochanteric distance was ++++. Twenty-three patients had severe (++++) deformation of the physis, and twenty-four had severe deformation of the femoral head. Seventeen of the twenty-three with severe deformation of the physis had a severely deformed femoral head at final follow-up, and thirteen of the twenty-three had premature closure of the growth plate (Table I). The physeal deformation was always

<table>
<thead>
<tr>
<th>Position of Trochanter</th>
<th>Final Result</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Excellent</td>
</tr>
<tr>
<td>Normal</td>
<td>16</td>
</tr>
<tr>
<td>Slight elevation (+)</td>
<td>3</td>
</tr>
<tr>
<td>Moderate elevation (+++)</td>
<td>4</td>
</tr>
<tr>
<td>Severe elevation (+++)</td>
<td>0</td>
</tr>
</tbody>
</table>
FIG. 7

Anteroposterior radiograph of the proximal end of a femur, showing ++ curvature of the physis. The lateral edge of the epiphysis seems to have grown over the upper surface of the neck, producing a notch between it and the greater trochanter (the lateral protrusion deformity).

The same on the lateral as on the anteroposterior radiograph except in Cases 2, 15, and 20, for which the rating was one grade lower on the lateral radiograph, and in Case 12, for which the reverse was true.

**Lateral Protrusion of the Capital Nucleus**

This is a relatively late deformation of the capital femoral epiphysis, whose lateral edge is seen to bulge progressively inferolaterally, making the epiphysis tilt into valgus angulation and in severe examples to overlie the superior surface of the femoral neck (Fig. 7). Thirty-three femoral heads (41 per cent) showed clear evidence of protrusion, and thirty of them showed a major curvature of the plate (+ + in eleven and + + + in nineteen). Only three femoral heads with protrusion showed a physis that was normal or that had only a mild curvature.

FIG. 8

Anteroposterior radiograph of the left hip of a child who was nine years and ten months old, two years and three months after the diagnosis of coxa plana. The unaffected right hip is shown in Fig. 4. The medial border of the neck in this affected hip is more highly arched than its normal fellow, and leads superiorly to a more prominent, squared-off medial metaphysis. This is the medial bowing deformity of the femoral neck.
Anteroposterior radiograph of the hips of a boy, eleven years and seven months old, who had had coxa plana of the right hip at the age of eight. A medial bowing sign and a + + curvature of the physis are seen as residues of that condition.

**Medial Bowing of the Femoral Neck**

On the anteroposterior radiograph, the medial border of the femoral neck normally runs superiorly in a gentle arc from the lesser trochanter to the metaphysis. This curve is the lateral component of Shenton’s line. In forty-seven patients this line became shorter and more concave than in the contralateral, unaffected hip. This appearance was frequently associated with prominence of the medial border of the metaphysis (Fig. 8).

In twenty-five patients this medial bowing was apparent in the early stages after diagnosis and then disappeared. In thirty-three patients it was absent throughout their course and in twenty-two patients it persisted once it had appeared (Fig. 9), at an average age of 8.8 years — 0.25 to five years (mean, 1.5 years) after the diagnosis of coxa plana. No difference was found in the age at diagnosis in the patients with or without this sign. The patients with medial bowing, compared with the patients who did not have this sign, showed greater incidences of premature closure (59 compared with 12 per cent), important elevation of the greater trochanter (77 compared with 38 per cent), and lateral protrusion of the capital nucleus (77 compared with 30 per cent).

**Discussion**

This study has shown that interference with the growth of the physeal plate of the femoral head is common in coxa plana — much commoner than we had previously suspected. We believe that in the past this was overlooked, firstly because most patients are not followed until maturity once the femoral head has been reconstituted, and secondly because follow-up usually is terminated because of apparent clinical normality. Several months, and for many patients years, may precede the radiographic evidence of disturbance of growth. It has been our practice to see these children annually or biennially until they reach at least the age of fifteen, and this has given us the opportunity to assess the growth plate and to determine whether any damage has been manifested in late adolescence. Twenty-five per cent of our patients showed evidence of premature physeal fusion. Some degree of interference with normal physeal growth was found in 90 per cent of the patients who were examined. When we consider the possible cause of the growth restraint that we noted, we attribute it to a local lesion, not part of the generalized interference with skeletal growth in coxa plana that has been described elsewhere. The growth restraint is not always evident; in many patients with a mild lesion (Catterall Grade I) the lesion will not develop. Such patients were not included in this series because they were not considered to need treatment. We have studied them and they have not shown any evidence of physeal involvement. A third point is that the lesion is not exclusively related to containment therapy, as we have seen it develop in children who were treated by methods other than containment and in those who were not treated at all. Finally, the growth plate is not involved symmetrically. The interference with growth is usually in the area of the physis underlying the maximally involved segment of the capital epiphysis. In 25 per cent of such patients a premature osseous fusion between the epiphysis and the metaphysis becomes evident. Coxa plana often spares the posteromedial segment of the head and affects predominantly the central
and anterolateral areas. These are segments of the physis that usually sustain growth restraint and possibly premature fusion.

How can interference with physeal growth be related to changes in the overlying osseous epiphysis? A concept that coxa plana is the result of epiphyseal stress fractures has been explained at length elsewhere. In some children such a fracture may heal rapidly and produce minimum bone infarction. However, such a fracture may be followed by additional fractures and infarctions. Thus, the volume of necrotic epiphyseal bone may be small and may involve only the summit of the head, or it may constitute a major part of the epiphysis, involving its borders as well as its center and depths, all of which may become necrotic. In the former event we envisage that the germinal cells of the physis may undergo little or no disturbance in their metabolism, but if major epiphyseal compression and distortion take place then damaging loads will fall on the germinal cells of the physis, which are normally shielded by the intact articular cartilage and bone of the capital epiphysis. Thus, we envisage the physeal damage to be directly related to the extent and geography of the involvement of the overlying bone, a load-directed injury. Such loading may damage the physis in more than one way. The possibility of germinal-cell infarction is easily envisaged as the epiphysis progressively flattens. The arterioles and venules serving the physis from its epiphyseal surface will be progressively vulnerable. However, the evidence for physeal infarction is not strong. An infarcted plate would be likely to disappear within months after injury, but in our study the mean age at which premature physeal fusion occurred was twelve years and eleven months, compared with a mean age at diagnosis of seven years and eleven months.

We suggest an alternative pathophysiological explanation, whereby epiphyseal flattening and distortion may result in retardation of germinal-cell growth. In patients with the most severe epiphyseal flattening, the fractured and flattened bone will allow increased loads on the physis that may distort or even fracture it. We suspect that such fracture lines may not heal and may cause few or multiple micro-epiphyseodeses, varying in extent. If there are few such lesions they may result in growth restraint, but they may exert a retarding influence that is strong enough to cause premature physeal fusion. Barnes also made this suggestion, and a lesion such as is postulated here can be found in the description of a necropsy. Whatever the pathogenesis, premature fusion may be important clinically. An increase of as much as 1.5 centimeters in the length of the lower limb may be superimposed on the deformity of the femoral head resulting from the disease process and its treatment. This superimposed complication, occurring quite suddenly many years after an apparently satisfactory result, should be watched for.

How can deformation of the proximal end of the femur follow physeal damage? Growth abnormalities producing change in the shape of a bone are usually considered to result from so-called differential growth rates, as when the damaged portion of a physis grows more slowly than the rest, which grows at the normal velocity. The Salter-Harris Type-IV or V fracture at the knee or ankle is an example of this phenomenon. When we consider the deformities described in this paper, their pathophysiology requires some further clarification. Progressive elevation of the greater trochanter will occur as capital physeal growth diminishes while the physis of the greater trochanter grows at normal velocity. The former change will result in shortening of the neck, with consequent alteration of the forces that the abductors exert on the greater trochanter, whose shape will be modified and whose length will increase disproportionately relative to the femoral head and neck.

It would be convenient to ascribe the convex deformity of the physis that we have described to a disproportionate physeal growth rate, impaired centrally and normal peripherally. This, however, would lead to the exact reverse deformity, namely a concave or cupped physis similar to the type that results from a central growth arrest in, for instance, the distal end of the femur.

We therefore advance a hypothesis that the primary deforming force that causes the physis deformity in coxa plana originates on the epiphyseal side. Differential growth within the epiphysis related to the loads placed on it cause secondary physeal deformity.

References