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Slipped epiphyses in renal osteodystrophy

OTTO MEHLS, EBERHARD RITZ, BURKHARD KREMPIEN, GIULIO GILLI, KURT LINK, EBERHARD WILLICH, and KARL SCHÄRER

From the Medical School, Departments of Paediatrics, Internal Medicine, and Pathology, University of Heidelberg

Mehls, O., Ritz, E., Krempien, B., Gilli, G., Link, K., Willich, E., and Schärer, K. (1975). *Archives of Disease in Childhood*, **50**, 545. **Slipped epiphyses in renal osteodystrophy.** Clinical, biochemical, roentgenological, and histological features of slipped epiphyses (epiphysiolysis) in 11 out of 112 children with renal osteodystrophy have been analysed. Characteristic age-related patterns of involvement of different epiphyses are described. Quantitative measurements of iliac bone histology, serum parathyroid hormone levels, and clinical history show the presence of more advanced osteitis fibrosa in children with epiphysiolysis than in those without. A good correlation was found between serum parathormone levels and osteoclastic resorption, endosteal fibrosis as well as osteoid.

Histological studies show that the radiolucent zone between the epiphyseal ossification centre and the metaphysis in *x*-rays is not caused by accumulation of cartilage and chondro-osteoid (as usually found in vitamin D deficiency rickets) but by the accumulation of woven bone and/or fibrous tissue. The response to vitamin D therapy in most cases was good. Parathyroidectomy was required in only one case.

Skeletal changes secondary to renal osteodystrophy are localized in cortical and cancellous bone. In the growing skeleton, additional lesions are found in the growth zone. The most severe manifestations of renal osteodystrophy in the growth zone are slipped epiphyses (epiphysiolysis). The pathogenesis of the lesion has remained obscure and detailed information about underlying morphology, natural history, and response to therapy is almost entirely lacking. Therefore, a detailed analysis of our case material was undertaken.

Materials and methods

Thirty patients, 18 boys and 12 girls, with chronic renal failure under conservative treatment were investigated. The ages ranged from 2 to 16 years (mean age 10½ years). The average duration of chronic renal insufficiency, as defined by a serum creatinine level >2 mg/100 ml, was 25 months. The data from these children were compared with data obtained from 41 children being dialysed in several centres. This latter group consisted of 40 boys and 42 girls whose ages ranged from 2 to 17 years (mean age 11 years). The average duration of renal insufficiency in this group was 41 months. The underlying renal diseases in children of both groups are shown in Table I. Detailed information about the dialysis group has been previously reported (Mehls *et al.*, 1973a). Serum Ca and Mg were measured by atomic absorption spectrophotometry, and serum phosphorus and alkaline phosphatase were determined with an autoanalyser.

Serum parathormone (PTH) levels were measured with a bovine PTH antibody by the method of Schopman, Hackeng, and Lequin (1970). Transiliac bone biopsies were obtained under local anaesthesia with a Bartelheimer-trephine. Undecalcified sections embedded in methyl-methacrylate were stained after the method of Masson Goldner and evaluated by micromorphometry according to Schenk and Merz (1969).

X-rays were obtained at regular intervals of the lateral skull, clavicles, forearms and hands, lumbar spine, pelvis, knees, and feet.

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Results

Clinical findings. Slipped epiphyses were found in 10 of 30 (33%) nondialysed, and in only one of the 82 (1%) dialysed children. Diagnosis was suspected on clinical grounds in the nondialysed children, whereas only *x*-ray examination revealed epiphysiolysis in the dialysed patient (Case 7). It is noteworthy that all the children with the exception of the dialysed patient had congenital renal

TABLE I

Primary renal disease in 112 children with chronic renal failure

	Uraemic children (conservative treatment)	Dialysed children
Glomerulonephritis	7 (23)	39 (48)
Obstructive uropathy	11 (37)	22 (27)
Juvenile nephronophthisis	5 (17)	8 (10)
Polycystic disease	1 (3)	4 (5)
Oligomeganephronic hypoplasia	2	1
Renal hypoplasia	2 (17)	1 (2)
Segmental hypoplasia	1	—
Alport's syndrome	—	2 (2)
Oxalosis	1 (3)	1 (1)
Others	—	4 (5)
Total	30	82

Note: Percentage in parentheses.

disease (Table II). The mean duration of renal disease was 10 years, and that of renal insufficiency 49 months.

In 3 patients (Cases 1, 4, and 5) with *x*-ray changes indicating only a moderate degree of renal osteodystrophy and initially not treated by vitamin D, slipped epiphyses developed within a 6-month period. The pattern of epiphyseal involvement seemed to be age-related (Table III). In preschool children slipping was observed in both upper and lower femoral epiphyses but not in the distal radial and ulnar epiphyses, while in older children only the upper femoral and/or the radial and ulnar epiphyses were involved. In patients with the highest degree of slipping of the femoral, radial and ulnar epiphyses, slipping was also found in other epiphyses, such as metatarsal, metacarpal, humeral, lower tibial, and fibular.

In 4 of 9 children (Cases 3, 5, 6, 9), epiphyseal slipping in the ulna and radius was manifested clinically by swelling of the wrists and visible ulnar deviation of the hands (Fig. 1), but without functional impairment. Contrary to expectations, handedness did not appear to influence the degree of involvement. Lesions were distributed equally and bilaterally in all but one patient (Case 9).

Locomotion was uniformly impaired in children with slipped upper femoral epiphyses. 2 children (Cases 1 and 2) were unable to walk, probably due to coexisting involvement of the knees. 5 children (Cases 3–6 and 8) had waddling gait. None of the children complained of pain in the involved epiphyses. However, 3 children (Cases 4, 5, and 10) complained of moderate pain in the knees with exertion. These 3 children did not have slipped lower femoral epiphyses, but they were knock-kneed so that the complaints could have been related to faulty static loading.

Severe dorsal deviation of the distal tibial epiphyses was seen in a 2½-year-old boy (Case 1)

TABLE

Serum biochemistry of uraemic

Case no.	Sex	Age (yr)	Age (m)	Diagnosis
1	M	2	6	Obstructive uropathy
2	M	3	7	Renal hypoplasia + dysplasia
3	F	7	7	Oligomeganephronic hypoplasia
4	M	8		Obstructive uropathy
5	F	9	11	Renal hypoplasia
6	F	12	3	Obstructive uropathy
7	F	12	3	Glomerulonephritis
8	F	12	4	Juvenile nephronophthisis
9	M	12	6	Oligomeganephronic hypoplasia
10	F	13		Glomerulonephritis; infantile familial nephrotic syndrome
11	M	16		Juvenile nephronophthisis

For normal biochemical values, see Table IV.

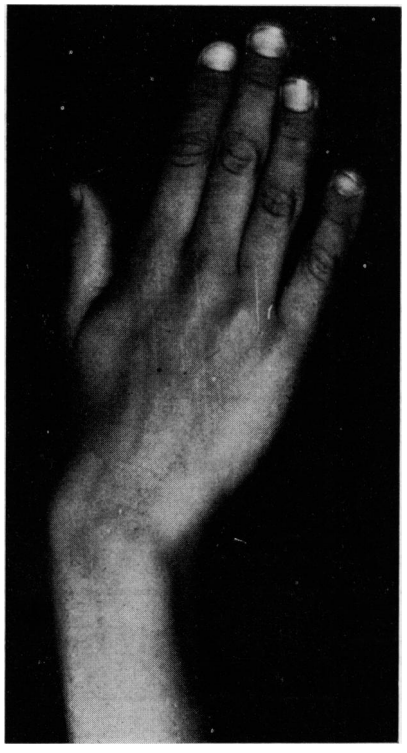


FIG. 1.—*Case 9. Ulnar deviation of right hand as a clinical sign of slipped ulnar and radial epiphyses in severe renal osteodystrophy. No trauma, no pain, no functional impairment. Note pseudoclubbing of the fingers, caused by excessive acro-osteolysis.*

who stopped walking and reverted to crawling after renal osteodystrophy developed. A 12-year-old boy (Case 9) with far advanced renal osteodystrophy had lateral displacement of the lower fibular epiphyses which caused marked pronation of his feet.

Extraskeletal signs of uraemic bone disease, such as vascular or other extra-osseous calcifications, were never detected. Pruritus was observed equally in children with and without epiphysiolysis.

Serum biochemistry. Our patients with epiphysiolysis tended towards hypocalcaemia before treatment with vitamin D (Tables II and IV). Serum alkaline phosphatase and serum PTH levels were found to be significantly higher ($P < 0.01$) than in uraemic children without epiphysiolysis. There were no significant differences in the serum Mg and phosphate levels.

Bone histology. Iliac crest biopsies were obtained from 8 patients with epiphysiolysis. This group was compared with a group of non-dialysed uraemic children without epiphysiolysis and with a control group of children matched for age and sex without skeletal or renal disease who had died accidentally (Table V). All uraemic children showed histological evidence of renal osteodystrophy. An increased fraction of bone per unit spongiosal volume (V_v), which is evidence of more advanced osteosclerosis, was noted in patients with epiphyseal slipping. Both the osteoid volume fraction (V_o) and the osteoid surface fraction (OS) were increased. This may reflect increased bone turnover, impaired mineralization, or appearance of poorly mineralized woven bone (Ritz *et al.*, 1975). The specific bone volume (S/V) was clearly decreased, indicating coarse trabeculae with increased trabecular diameters. In contrast, surface density (SD), i.e. the interface between the bone and marrow cavity, was slightly

II

children with epiphysiolysis

Creatinine (mg/100 ml)	PTH (pgEq bovine PTH/ml)	Ca (mEq/l)	Mg (mEq/l)	P (mg/100 ml)	Alkaline phosphatase (IU/ml)
2·5	2640	3·8	1·9	4·8	450
3·6	1290	4·3	2·2	5·3	103
6·5	1370	5·0	1·6	5·6	249
9·8	2240	3·0	1·8	6·6	280
10·2	2500	3·6	1·9	6·0	386
8·9	—	4·1	1·7	7·2	478
Dialysis	1160	4·5	2·2	6·7	435
7·8	—	4·0	1·4	6·6	415
9·2	3500	4·8	2·6	6·6	220
12·1	2045	2·6	2·3	8·7	876
7·2	2020	5·0	2·4	5·4	180

TABLE III

Major sites of slipped epiphyses

Case no.	Age (yr)	(m)	Upper humerus	Distal radius	Distal ulna	Proximal femur	Distal femur	Distal tibia
1	26		—	—	—	+	+	+
2	37		—	—	—	+	+	—
3	7	7	+	+	+	+	—	—
4	8		—	+	+	+	—	—
5	9	11	—	+	+	+	—	—
6	12	3	+	+	+	+	—	—
7	12	3	—	+	—	—	—	—
8	12	4	—	+	+	+	—	—
9	12	6	+	+	+	+	—	—
10	13		—	+	+	—	—	—
11	16		—	+	+	—	—	—

+, epiphysis slipped; —, epiphysis not slipped.

TABLE IV

Comparison of serum biochemistry in uraemic children with and without epiphysiolysis

	Uraemic children		P*	Normal range
	Without epiphysiolysis (n=19)	With epiphysiolysis (n=11)		
Serum Ca (mEq/l)	4·5±0·57	4·0±0·8	<0·1	4·5–5·5
Serum P (mg/100 ml)	6·1±1·8	6·3±1·1	NS	4·3–5·1
Serum alkaline phosphatase (IU/ml)	134±80·3	370±208	<0·01	38–138
Serum Mg (mEq/l)	1·9±0·44	2·0±0·35	NS	1·6–2·0
Serum PTH (pgEq bovine PTH/ml)	1229±578	2107±668	<0·01	<420

*Difference between children with and without epiphysiolysis (Wilcoxon test).

TABLE V

Micromorphometric measurements of iliac bone (mean ± SD)

	I Control group (n=9)	II Chronic renal failure without epiphysiolysis (n=19)	III Chronic renal failure with epiphysiolysis (n=8)	Significance (P) of difference between*		
				I & II	I & III	II & III
V_V (%)	17·7±4·9	26·5±9·0	33·7±7·1	0·01	0·01	0·1
V_O (%)	0·4±0·2	2·3±1·9	5·3±4·8	0·01	0·01	0·05
S/V (mm^2/mm^3)	17·1±3·1	12·7±2·6	11·8±3·2	0·01	0·01	0·1
SD (mm^2/cm^3)	2752±813	3175±770	3855±908	NS	0·05	0·05
HO (%)	0·3±0·7	3·89±7·7	12·3±7·7	0·01	0·01	0·01
HO (mm^2/cm^3)	4·1±7·7	126±17·7	444±279	0·01	0·01	0·01
OS (%)	7·2±4·0	43·6±19·0	52·0±24·9	0·01	0·01	NS
OS (mm^2/cm^3)	160±90	1430±739	2000±1125	0·01	0·01	0·01
S (μm)	11·6±2·8	17·1±6·7	21·4±11·7	0·01	0·01	0·01
EOF (%)	0	45·3±35·2	88·8±13·3	0·01	0·01	0·02

V_V, fraction of bone volume contained in unit volume of spongiosa; V_O, fraction of osteoid volume contained in unit volume of spongiosa; S/V, trabecular surface/unit volume of bone; SD, interface between trabecular bone and marrow cavity; HO, active Howship's lacunae (given as % trabecular surface and given as surface/unit spongiosa volume); OS, osteoid seams on travecular surface (given as % trabecular surface and as surface/unit spongiosa volume); S, seam thickness of osteoid; EOF, fraction of trabecular surface covered by endosteal fibrosis.
*Wilcoxon test.

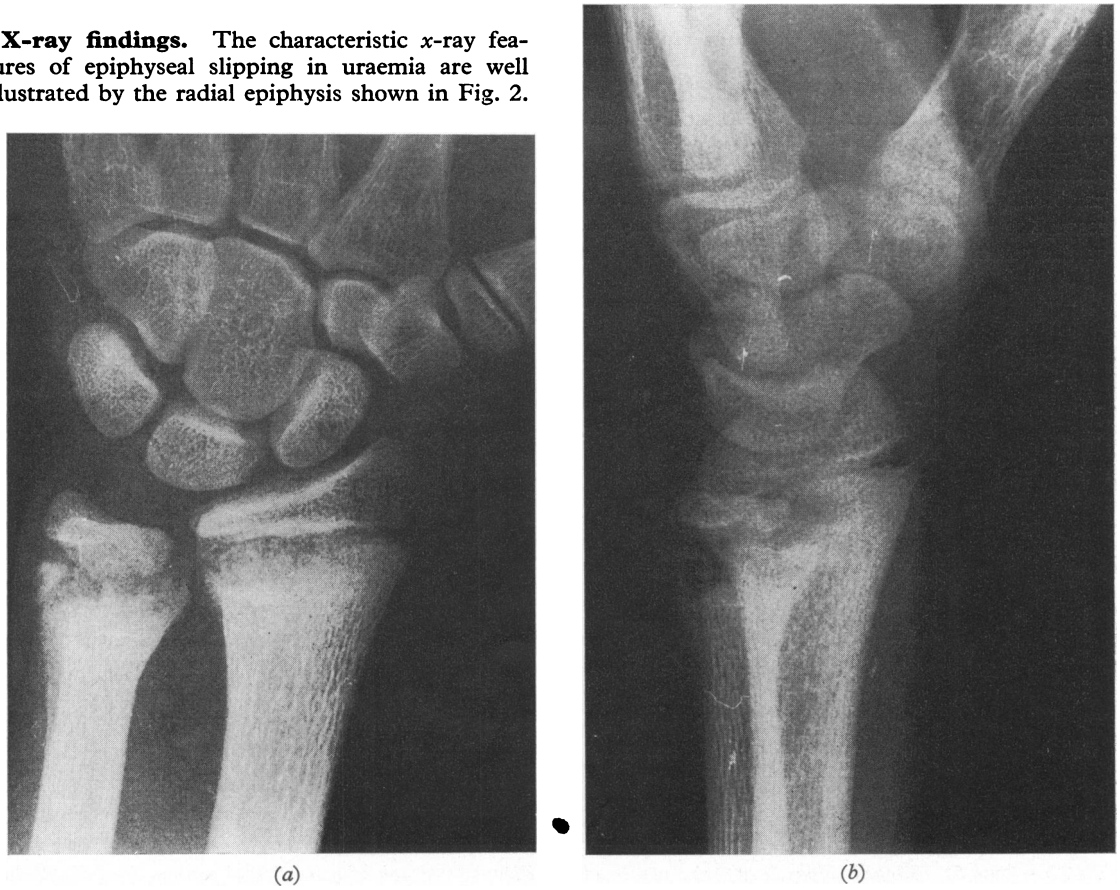
increased. The surface fraction of active Howship's lacunae (HO) was markedly increased, and endosteal fibrosis (EOF) was prominent.

In uraemic children both with and without epiphysiolysis good correlations were observed between serum PTH levels and the surface fraction of active Howship's lacunae, the surface fraction of endosteal fibrosis, and the surface fraction of osteoid (Table VI). In children with epiphysiolysis there was more advanced osteitis fibrosa, as manifested by higher PTH levels and increased numbers of active Howship's lacunae and marked endosteal fibrosis (Tables IV and V).

The histological findings in the growth zone, previously described by Krempien, Mehls and Ritz (1974), confirm the advanced stage of hyperparathyroidism in these patients.

X-ray findings. The characteristic *x*-ray features of epiphyseal slipping in uraemia are well illustrated by the radial epiphysis shown in Fig. 2.

The structure of the epiphysis proper with its slow rate of growth was usually not affected. Judging from *x*-ray, the radiolucent band of the growth cartilage was narrow. This interpretation is confirmed by our histological findings (Krempien *et al.*, 1974) (Fig. 3). The underlying zone of metaphyseal spongiosa was poorly trabeculated and devoid of trajectorially orientated longitudinal trabeculae. The metaphysis adjacent to the growth plate showed zones of diminished density, particularly in the long tubular bones. At times the metaphyses had the appearance of irregular radiolucent bands and at other times that of cyst-like defects. The latter were especially prominent during vitamin D treatment. In contrast to the metaphyseal zone near to the growth plate, the



(*a*) (*b*)

FIG. 2.—*Case 11.* X-*rays of incipient displacement of radial epiphysis of left forearm.* (*a*) *Dorsovolar and* (*b*) *lateral views, technique after Meema. Incipient displacement of radial epiphysis towards* (*a*) *ulnar and* (*b*) *dorsal sides. Narrow, irregularly outlined growth plate. Wholly disorganized structure of upper metaphysis with large irregular defects. Subperiosteal resorption zone at ulnar cortex. Console formation at ulnar side of radial metaphysis. Dense coarse trabeculae in lower metaphyses.*

TABLE VI

Correlation between micromorphometric parameters and serum PTH levels (x=mgEq bovine PTH/ml) in untreated uraemic children before dialysis, with and without epiphysiolysis (n=20)

(1)	Surface fraction of active Howship's lacunae (HO) y=HO (%) y=(−1·735+0·005)x; r=0·758; P <0·01
(2)	Surface fraction of endosteal fibrosis (EOF) y=EOF (%) y=(9·673+0·032)x; r=0·732; P<0·01
(3)	Surface fraction of osteoid (OS) y=OS (%) y=(24·2+0·013)x; r=0·45; P <0·05
(4)	Bone volume/unit spongiosal volume (V_V) y=V_V (%) y=(27·9+0·0001)x; r=0·041; NS

inner metaphysis usually was denser with coarse, irregular, poorly delineated trabeculae. The metaphyseal cortex was irregularly thinned by endosteal erosion and showed longitudinal striations caused by the enlargement of the haversian channels. In addition, the metaphyseal cortex was eroded at the periosteal surface by deep scalloping subperiostal resorption defects which were predominantly seen at the external aspect of the ulna. It should be remembered that some 'subperiosteal resorption' caused by deposition of woven bone can also normally be seen at this site in healthy rapidly growing children (personal observation). Usually the displaced epiphyses jutted beyond the lateral margin of the metaphysis. Sometimes they were supported by a console of newly formed bone.

In the radial and ulnar epiphyses ulnodorsal deviation was the rule, but one child (Case 3), with a traumatic fracture of the right humerus and right radial metaphysis, had slipping to the radial side. Interestingly, the displaced epiphysis slipped to the ulnar side soon after the humeral fracture was immobilized.

The upper femoral epiphyses slipped into lateral or posterior-inferior position (best seen in the Lauenstein-position) (Fig. 5). In addition, the collodiaphyseal angle was decreased (coxa vara epiphysaria). Particularly severe subperiosteal resorption was seen at the femoral neck, where the ileofemoral and pubofemoral ligaments insert (rotten fence post sign). The lower femoral epiphysis slipped posteriorly (best seen in lateral view, Fig. 6). This displaced epiphysis is particularly prone to traumatic fractures.

Slipping of other epiphyses such as at the proximal humerus (Fig. 4), metacarpal or metatarsa

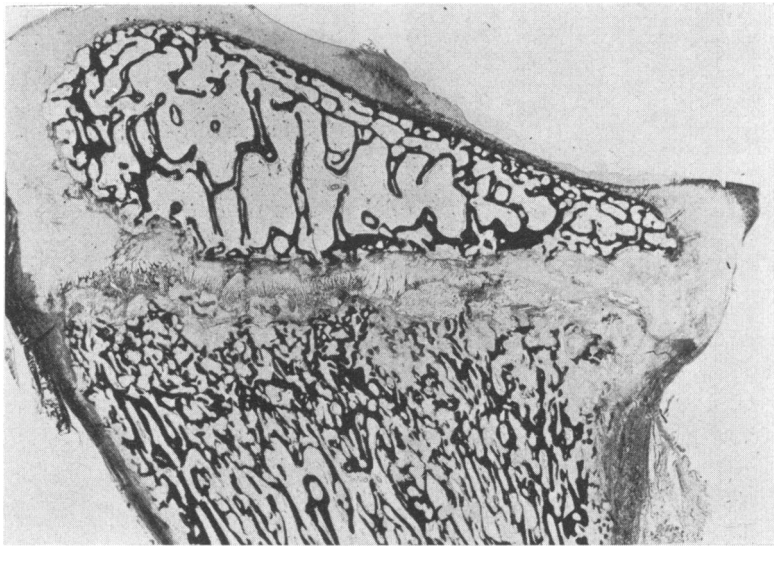


FIG. 3.—*Case 5. Slipped epiphysis of right distal radius. Photomicrograph of undecalcified section, Masson-Goldner stain. Narrow irregular cartilage plate. Severe fibro-osteoclasia with destruction of primary spongiosa and dissecting cancellization of cortical bone. Incipient displacement of epiphysis with erosion of its lower radial border; epiphysis at ulnar side riding over metaphysis, supported only by a console of fibrous tissue and woven bone. No evidence of traumatic separation. Intensive subperiosteal resorption with complete dissolution of cortex at ulnar side. Note resorptive defects in metaphysis filled with fibrous tissue or poorly mineralized woven bone.*

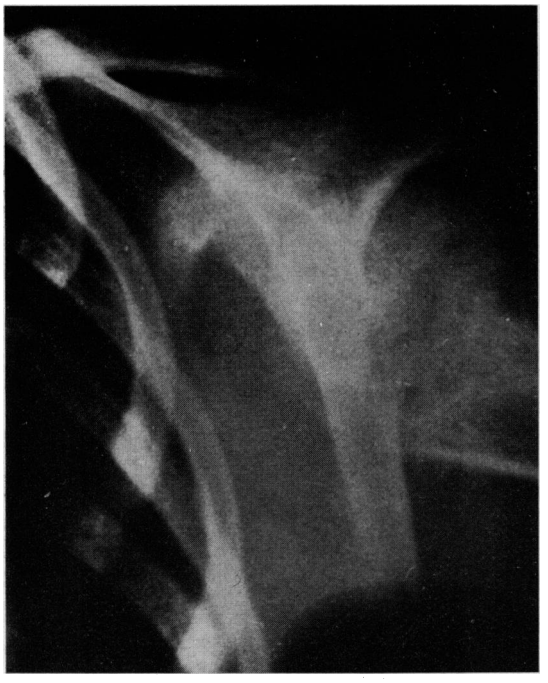


FIG. 4.—*Case 6. X-ray of slipped upper humeral epiphysis of left shoulder. Extensive metaphyseal changes of humerus. Displacement of humeral shaft with dorsolateral position of upper humeral epiphysis.*

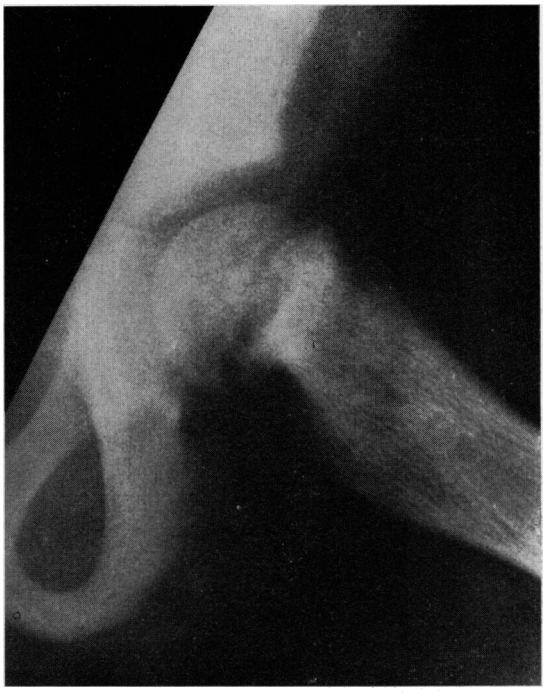


FIG. 5.—*Case 3. X-ray of slipped upper femoral epiphysis of left hip. Axial view (Lauenstein-position). Epiphysis is placed downwards and posteriorly. Severe resorptive defects at femoral neck (rotten fence post sign).*

areas was seen only in the most severely affected children (Fig. 7). Usually no major abnormalities were found in epiphyses subject to axial compression, e.g. in the upper tibia.

Treatment. Most children responded to vitamin D_3 given in high doses from 20 000 to 100 000 IU per day (Mehls *et al.*, 1975). Increased mineral density was evident on *x*-ray after 4 weeks and almost complete normalization of the trabecular structure of the primary spongiosa was seen after nearly 6 months. During this interval the position of the epiphysis relative to the metaphysis had reverted more towards normal in both the radius and ulna, particularly in young children. Slipped femoral epiphyses were consolidated but remained out of normal position. Apart from 3 cases of transient moderate hypercalcaemia, no vitamin D intoxication was seen. Only one child (Case 9) required subtotal parathyroidectomy to correct his metaphyseal lesions.

While the lesions in the fast growing epiphyseometaphyseal region always healed with vitamin D and calcium supplements alone, orthopaedic surgery was subsequently required in one patient to correct metaphyseodiaphyseal deformities (Case 1). In contrast to idiopathic epiphyseal slipping, attempts at stabilizing epiphyses by screws or nails are ill-advised, because of the different underlying metabolic and anatomical abnormality in patients with advanced renal failure (Mehls *et al.*, 1975).

Discussion

Epiphyseal slipping is a frequent complication in the natural course of uraemic bone disease. It may develop quite rapidly, as shown by some of our patients (Cases 1, 4, and 5). In young children it may occur at an early stage of renal insufficiency (glomerular filtration rate in Cases 1 and 2, 17 ml and 18 ml/min per 1·73 m^2, respectively). In contrast, epiphysiolysis was considerably less frequent in the dialysed children (1%). The lower incidence of the lesion in the dialysis group can be partly ascribed to the selection of patients referred for dialysis, furthermore it shows that the lesion is preventable and/or susceptible to therapy (Mehls *et al.*, 1975).

Slipping of the epiphyses in renal patients is caused by mechanisms different from those operat-

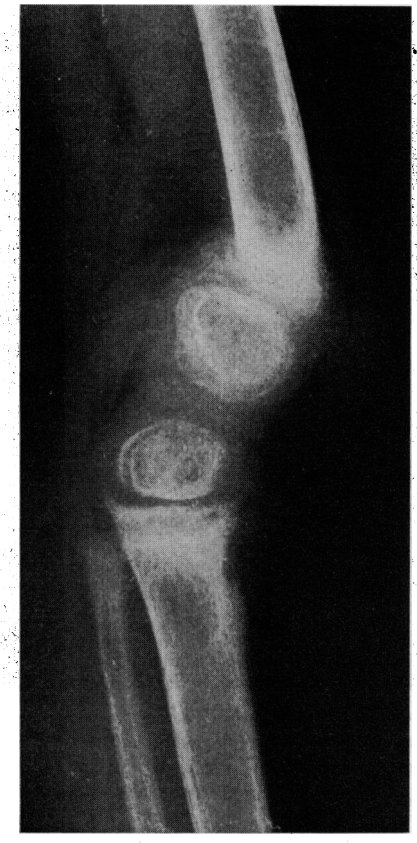


FIG. 6.—*Case 1. X-ray of slipped lower femoral epiphysis of left knee. Lateral view of left knee. Ventral displacement of femoral shaft. Intensive destruction of metaphysis with resorption at the ventral side and console formation at the dorsal side. Intensive resorptive lesions of the upper tibia and fibula.*

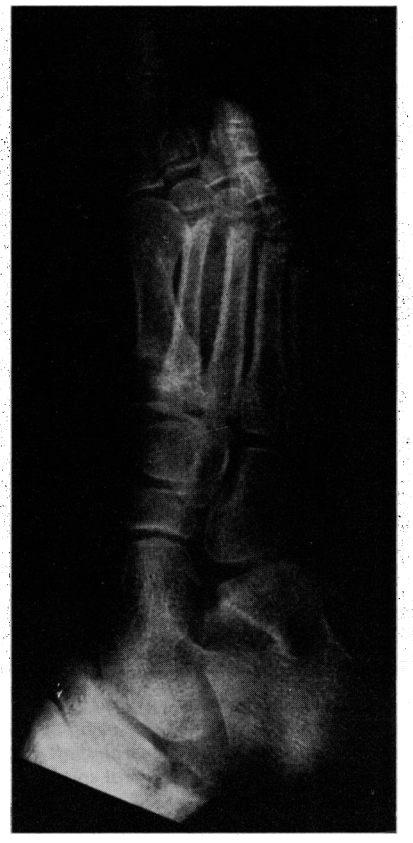


FIG. 7.—*Case 5. X-ray of slipped metatarsal epiphyses of left foot. Oblique view of left foot. Plantar displacement of metatarsal shafts with dorsal position of distal metatarsal epiphyses. Marked acro-osteolysis of distal phalanges.*

ing in slipping of upper femoral epiphyses in adolescence ('idiopathic epiphysiolysis'). In the latter case, clefts are initially found through the layer of hypertrophic cartilage adjacent to the zone of calcification which is possibly the consequence of disturbed vascular invasion (Lacroix and Verbrugge, 1951; Ponseti and McClintock, 1956; Morscher, 1968). The subsequent disorganization of metaphyseal structures is secondary to disturbed differentiation of cartilage cells and faulty mechanical loading.

In children with renal failure, slipped epiphyses are not the result of a local process but the consequence of generalized metabolic bone disease. Our histological studies show that epiphysiolysis in uraemic children is the ultimate consequence of osteitis fibrosa (Krempien *et al.*, 1974). It has to be noted that slipped epiphyses have also been found in primary hyperparathyroidism (Kirkwood, Ozonoff, and Steinbach, 1972). In the growth zone of uraemic children we found that the cartilaginous growth plate is separated from the metaphyseal spongiosa by chondroclastic removal of hypertrophic cartilage. Then dense fibrous tissue is interposed between growth cartilage and the adjacent metaphysis providing a plane of slippage. The trabeculae of the primary spongiosa show evidence of intensive remodelling, are devoid of a core of chondroid and consist entirely of woven bone. In roentgenological studies, microtraumata have been implicated as the final event triggering the slippage of epiphyses (Kirkwood *et al.*, 1972). However, in our histological studies in early stages of epiphyseal slipping, neither haemorrhage nor metaphyseal microfractures were found as histo-

logical evidence of microtraumata. However, *x*-ray findings in some patients with advanced slipping suggest that superimposed eccentric metaphyseal fractures further contribute to slipping.

Unlike the usual finding in vitamin D deficiency rickets, accumulation of chondro-osteoid in the growth zone (i.e. poorly mineralized chondroid with chondrocytes covered by unmineralized osteoid) was not observed. This might be the consequence of poor growth which prevents the appearance of chondro-osteoid. Similarly in vitamin D deficiency, rachitic histology with chondro-osteoid is not observed in the presence of absolute starvation (Park, 1954). Alternatively, in endstage renal failure hyperparathyroidism of greater intensity than usually encountered in nutritional vitamin D deficiency might have led to removal of pre-existing chondro-osteoid by aggressive chondroclastic resorption.

Consequently on *x*-rays the broad radiolucent zone between the ossification centre and the metaphysis does not reflect the accumulation of cartilage and chondro-osteoid (Park, 1939), but the appearance of poorly mineralized woven bone and/or fibrous tissue in the primary spongiosa.

The absence of rachitic histology does not necessarily mean that respective biochemical abnormalities are not present. However, the actual process of slipping is brought about exclusively by fibro-osteoclastic and chondroclastic resorption as described above. In support of the contention of the fibro-osteoclastic nature of the lesion, children with slipped epiphyses have also more severe fibro-osteoclastic lesions elsewhere in the skeleton (as shown by our micromorphometric studies of the iliac bone) and had more raised serum PTH levels. They had a longer duration of renal insufficiency and more frequently had congenital renal disease. The therapeutic response to vitamin D in the presence of fibro-osteoclastic histological lesions parallels the findings of Verberckmoes in adult dialysis patients with renal osteitis fibrosa (Verberckmoes, Bouillon, and Krempien, 1973). Even in primary hyperparathyroidism administration of vitamin D has been shown to improve resorptive bone lesions (Woodhouse, Doyle, and Joplin, 1971).

Two factors seem to determine the extent of involvement of a given epiphyseal growth plate: the inherent growth rate and the mechanical load. Differences in growth rate may explain the characteristic pattern of involvement in different age groups. Growth rate alone, however, is not the only determinant. Whereas in vitamin D deficiency rickets the distal fast growing end of the femur is predominantly involved, in renal osteodystrophy more striking changes are seen in the upper femoral epiphyses (Dent, 1973). This is presumably the consequence of mechanical forces which induce excessive remodelling. The different behaviour of the proximal tibial and the proximal femoral epiphyses illustrates the importance of this factor. In contrast to the epiphyseal growth plates of proximal tibiae which are subject to axial compression and show narrowing without dislocation, the upper femoral epiphyses are subjected to shearing forces and exhibit a marked tendency to slip. In a diseased epiphysis, displacement is brought about by the constant action of shearing forces, either muscular as in the ulna and radius, or gravitational as in the upper femur.

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Correspondence to Dr. O. Mehls, Universitäts-Kinderklinik, 69 Heidelberg, Im Neuenheimer Feld 150, Federal Republic of Germany.

Addendum

After submitting this article a second child (referred to as Case 1) underwent subtotal parathyroidectomy. In this child the dose of vitamin D_3 necessary to prevent relapse of bone lesions was 10 000 IU/d. This resulted in recurrent hypercalcaemia. Therefore, 12 months after starting vitamin D treatment parathyroidectomy was performed. Afterwards, doses of 2000 IU/d vitamin D_3 were sufficient to sustain healing. This case stresses further the importance of hyperparathyroidism in the genesis of epiphysiolysis in uraemia.