T. GORDH, STOCKHOLM:

ON RENAL RICKETS

Most cases of the so-called renal rickets (renal dwarfism, renal infantilism) have been reported by English and American authors. In recent years, however, cases of this rather uncommon diseases have been reported also from other countries, especially Germany and France. In the Scandinavian countries cases have been described by Svensgaard and by Bichel, Denmark, by Salvesen and by Sundal, Norway. In Sweden one case has been reported by Faxén in Acta paediatr. scandinav 1932 and in Hygiea, same year. About 100 cases altogether have been described so far (16, 29).

As early as 1883 (24) the clinical picture of the disease was described the first time, and now it is clearly differentiated clinically. But as such cases are relatively rare, and as the etiology has not yet been established, it is important that every case which is diagnosed as renal rickets should be examined thoroughly and reported so completely as possible. Now the term "renal rickets" has been adopted generally for the syndrome that, to begin with, was made up by the appearance of chronic nephritis in the latter part of childhood combined with skeletal changes of rachitic type. The disease does not involve any rickets due to vitamin D deficiency, however, and it is only morphologically that it is comparable to genuine rickets (2, 17). Through recent investigations the clinical picture of the disease has been complicated by the presence of parathyroid hyperplasia as revealed on autopsy. So, at present the syndrome of renal rickets may be set forth as: 1) renal insufficiency of the chronic nephritic type, with or without congenital malformation of the

urinary passages; 2) skeletal changes of the rachitic-osteodystrophic fibrous type; 3) parathyroid hyperplasia (34).

Renal rickets is a disease occurring at the age of growth and characterized clinically by disturbances of growth, with skeletal deformities resembling those encountered in pronounced rickets. A chronic kidney lesion is manifest at the same time, but often the renal symptoms are masked by the skeletal changes and not discovered till renal incompensation develops. Usually the disease sets in with the earliest symptoms at the age of 7—8 years (2, 27). Not infrequently, however, the kidney lesion may be traced far back, even to the first year of life (5). Usually the diagnosis is not made till the appearance of skeletal changes and signs of advanced damage to the kidneys, being based on the common symptoms: skeletal, renal and blood changes.

General Symptoms.—Dwarfism is a constant symptom, and it is of proportional type. Usually the skin is dirty yellowish in colour, reminding somewhat of Addison's disease. Mentally these children are strikingly well-developed. On the other hand, sexual retardation is the rule (16, 27). Polyuria and polydipsia are reckoned as early symptoms.

Skeletal Changes.—In many cases medical advice is sought first on account of the characteristic skeletal changes that appear in the latter part of childhood or after puberty in the form of genua valga, vara, coxa plana-like phenomena, backache, thickening of the extremities, slipped epiphyses and liability to fractures (27). The X-ray picture is decisive of the diagnosis, and roentgenologically distinction is made between different types. Brailford's classification of the cases in types A and B is the most clear-cut (7, 27). Type A reminds more of florid rickets, only that the latter shows universal osteoporosis with blurred corticalis borders, widening of the epiphyses and deformities due to softening of the long bones, while the changes in Type A are localized chiefly to the metaphyses, which become extraordinarily wide through the formation of a large amount of osteoid tissue between the epiphysis and the diaphysis. The ends of the diaphysis are irregular and "woolly", while the border between the epiphysis and the metaphysis is more sharp-

ly defined. These changes are most pronounced in the hip-joint, sacroiliac joint, knee- and shoulder-joints, but they are distinct also in the bones of the hand and foot. The long bones show normal density and form, without osteoporosis or malacia, and thus the typical changes of classical rickets are completely absent here. In this type no changes are seen in the skull. The deformities in Type A are pronounced but seldom; they arise as the softened metaphyses yield to strain and loading. giving rise to an angle between the epiphysis and the diaphysis. Brailsford holds that renal rickets is a not infrequent cause of slipped epiphyses in the hip-joints, and in a couple of cases he has been able to make the diagnosis merely on the features of the roentgenograms; later the diagnosis was verified on examination of the kidneys (7).

Type B presents the features of more serious metabolic disturbances with pronounced deformities of bones, osteoporosis of the entire skeleton, greater thickening of the metaphyses and even involvement of the flat bones. The X-ray picture of the skull is "woolly" and honeycomb-like, with cyst-formation, reminding of fibrous osteodystrophy (8); and in many cases the two conditions cannot be distinguished from each other. Here the osteomalacia also causes bending of the shafts and more pronounced deformities of the bones. According to Price and Davie, Type B is the more common form, and it does not arise without hyperactivity of the parathyroids (16, 27).

Renal Changes.—As mentioned already, the kidney phenomena take a more inconspicuous course, and usually they are not discovered till there has developed a renal insufficiency of the chronic nephritic type. In many cases the lesion involves congenital malformations of the urinary tract: cystic kidney, hydronephrosis, hydro-ureter. Distinction is made between congenital and acquired kidney lesions (16). The latter often arise through pathological changes in the lower urinary passages: strictures, diverticula, phimosis (16). In most accounts of renal rickets the occurrence of malformations in the urinary tract is strikingly common, and several authors look upon these as the primary factor in the development of the disease (2, 11, 16, 21,

25, 30, 33). Clinically we meet the features of a chronic nephritis with a high non-protein nitrogen concentration, which often keeps at a level of 150—200 mg.%. Values above 300 mg.% are not uncommon. Functional kidney tests show pronounced insufficiency, and intravenous urography generally turns out a failure. As revealed by many autopsies, the pathologic-anatomical changes are those pertaining to chronic interstitial nephritis with atrophic, cicatricial, kidneys in which histological examination shows a diffuse sclerosis and hyalinization of the glomeruli. The low blood pressure in these patients is to be pointed out; often it keeps within normal limits for a long time, showing hypertension only in the terminal stage (16). Examination of the urine shows more or less pronounced polyuria, isostenuria. As a rule, the albuminuria is moderate, showing about 1—2 ‰ albumin.

Blood Changes.—While genuine rickets as well as fibrous osteodystrophy show hypophosphatæmia and normal or increased calcium concentration, these conditions are reversed in renal rickets, where hyperphosphatæmia and hypocalcæmia is the rule. Further, in renal rickets we find a low alkali reserve and uncompensated acidosis. Usually there is a more or less pronounced anæmia.

In spite of these findings the patients feel surprisingly well, and it is difficult to understand that they can live so long in the shade of an impending uræmia, which must come sooner or later and lead on to exitus, usually before the age of 18—20 years. As shown by Salvesen (29) large doses of calcium phosphate may give a temporary improvement. The usual antirachitic therapy is quite ineffective. Still, as long as the skeletal changes keep within the roentgenographic Type A, there is thought to be some chance of recovery (27). Surgical measures are inadvisable. Before the disease was known sufficiently, osteotomy was performed not infrequently for the bone deformities, always with disastrous results.

In his investigation of 200 cases of nephrosclerosis in children, Mitchell found that 34 % of these cases presented clinical and roentgenographic changes in the bones of the type seen in renal

rickets, and in several of these cases the kidney lesion was discovered first on autopsy. In many cases the operation was soon followed by uræmic coma. The skeletal changes may appear at such an early stage that there are as yet no clinical signs of renal insufficiency, and still the autopsy reveals interstitial changes in the kidneys, notwithstanding normal findings on examination of the blood and urine (26, 33). Here the renal insufficiency is present in a latent form, manifesting itself on increased strain from intercurrent disease or operative treatment

Case Record.

This was the case of a girl, 15 years old, with a negative family history as to hereditary abnormalities. She was breast-fed during the first 5 months of life. The dentition was delayed, the first tooth appearing at the age of 12 months. She was growing but poorly. At the age of 1 year albuminuria and rachitic changes were ascertained. She was given cod-liver oil, and her condition improved. In 1936, at the age of 13 years, she had pain in her right hip, and she began to limp. She was admitted to a hospital under the diagnosis: nephritis chronica; coxa plana. The pain in the hip increased, and she had to get about on crutches. She entered the Stockholm V.F.A. (orthopedic hospital) on May 15, 1938. It is not known whether there has been any albuminuria during the period of 1925—1936.

Physical examination: General condition good. Height: 137 cm. (19 cm. shorter than normal for her age). Weight: 37.4 kg. Build fully proportional. Skin pale, with a dirty yellowish hue. Face of pentagonal type. Mental development normal for her age. Teeth typically rachitic.

Skeleton: No particular deformity except for slight genu valgum; especially, no deformity of the long bones. She is limping, and has to use a cane. Moderate reduction in the mobility of the right hip-joint, with decreased extension, rotation and abduction. No shortening of the extremity.

Heart, lungs and abdomen: No abnormality. Blood pressure: 105/60.

Urine: Diuresis 1000—1500 g. Isostenuria about 1008. Albuminuria (Esbach: 0.1 %). Acid reaction. Sediment, containing a moderate amount of red and white blood cells; no bacteria. No casts.

Blood: Anæmia; Hb. 50 %. Red cell count: 2.8 millions. White cell count: 8600. *Non-protein nitrogen: 177 mg.%. Blood calcium: 5.5 mg.%. Blood phosphorus: 10.4 mg.%.*

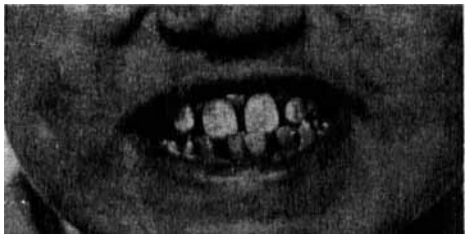
Roentgenography: The skeleton shows a systemic disease

Fig. 1. a. The patient compared with a normal girl of 15 years.

with changes in most of the epiphysial junctions. Nearly all metaphyses are widened and irregular. The most pronounced changes are seen in the hip-, shoulder- and knee-joints, besides in the distal ends of the radius and ulna, and in most of the fingers and metatarsal bones, but are absent in the elbow-joints. The sacro-iliac joints show a regular loosening. *Left hip-joint:* The epiphysial connection is loosened, the caput condensed. *Right hip-joint:* Caput flattened and widened (mushroom-like). In its central part absorptive changes have led to fragmentation, so that the picture has the character of coxa plana. A previous roentgenogram, taken in 1937, showed merely a condensation of the epiphysis and thinning of its lateral part (Fig. 2).

No changes in the vertebral column except for some blurring that is common to all parts of the skeleton. In profile roentgenograms of the skull the sella turcica is found to be a little large, but normal in outline. Roentgenography of the neck reveals no shadow of soft parts that might be suggestive of enlargement of the parathyroids.

Urinary Tract.—Cystoscopy shows normal ureteral orifices in the mucous membrane of the trigonum. The catheter is introduced easily on the right side. On the left side the catheter



b. Irregularities of the teeth, rachitic type.

meets an obstruction at the level of the sacro-iliac joint; and this obstruction cannot be forced, neither with the catheter nor with the injected contrast substance. Retrograde pyelography shows a small right kidney (4.5×9 cm.) and obstruction of the left ureter at the level of the 4th lumbar vertebra. No sharply defined outline of the left kidney shadow is obtained. On account of the high non-protein nitrogen value, intravenous urography is not performed; nor could a contrast-giving concentration be expected in the presence of this renal insufficiency.

This case of renal rickets is thus to be reckoned as belonging to Brailford's Type A; and it probably is to be entered in the group of congenital kidney lesions, inasmuch as the presence of albuminuria has been tracked back to the first year of life. Parathyroid hyperplasia could not be demonstrated. The patient has later been transferred to Crownprincess Lovisa's Children's Hospital, Stockholm, for further examination of the renal insufficiency, tolerance tests, parathyroid hormone deter-

mination, etc. The findings in these examinations will be published later by Dr. Kurt Kaijser, on the staff of that hospital.

Etiology and Pathogenesis.

While these aspects of genuine rickets have been established through the vitamin research, the etiology of renal rickets is still obscure. Many theories have been advanced concerning this question, and these have been complicated further through the circumstance that autopsy has revealed the presence of parathyroid hyperplasia in some of these cases, while this phenomenon was absent in other cases. This hyperplasia now constitutes the central point in the discussion of the etiology, and the main problem is, whether the hyperplasia is primary or secondary. Price and Davie have set up two cardinal theories: a renal and an endocrine (27). The former presupposes a primary kidney lesion, the latter a primary parathyroid disturbance.

All forms of chronic nephritis imply a retention of high-molecular salts, above all the phosphates. A phosphate retention in the blood arises with P values up to 10—12 mg.%, and there appears a relative Ca deficiency. The excess of phosphates is excreted by way of the intestines (26 *et al.*), where insoluble calcium phosphate is formed with the alimentary calcium, resulting in a true calcium deficit in the blood. In renal rickets this hypocalcæmia may show calcium values as low as 4 mg.%.

In this connection it may be of interest to point out the rare occurrence of tetany with these low calcium values (2, 29). We know that calcium is found in the blood in two forms: loosely combined with plasma albumin, and free. Ionisation of the free calcium depends upon the pH value of the plasma, and the more this is decreased on the acid side, the greater is the amount of free calcium. By ultrafiltration through a colloid membrane Pinkus, Peterson and Kramer have shown that even though the calcium content of the blood is very low it is chiefly the combined calcium that is reduced in chronic diseases of the kidney, while the free form gives off but very little in the filtration, probably this applies also to renal rickets (27). As, fur-

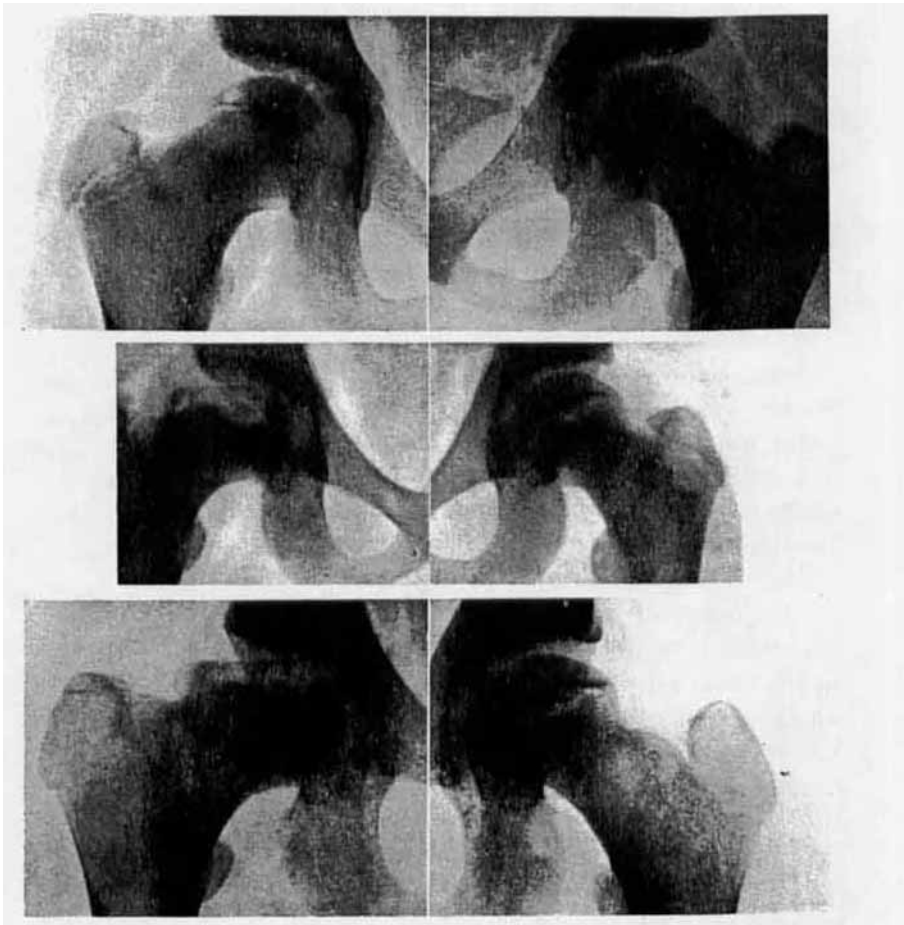


Fig. 2.

Development of the changes in the hip-joints.

a. (January 1937). *Right:* Semilunar epiphysis with decrease in height and rarefication of the lateral part. *Left:* Semilunar epiphysis.

b. (January 1938). *Right:* The process has advanced, with pronounced absorbtive changes within the outer ¾ of the epiphysis, softening and flattening of the caput within the corresponding area. Picture resembling coxa plana. *Left:* Loosening and widening of the metaphysis. The normal form of the epiphysis is preserved, sharply defined. No slipping.

c. (August 1938). *Right:* No particular progress since last examination. Caput deformed to the same extent as before. The resorbtive changes have come to a standstill; here and there the sclerosis is somewhat increased. *Left:* No changes. No slipping of the epiphysis (cf. Fig. 3 b).

thermore, the pH values of the blood are suggestive of uncompensated acidosis, the relatively rare occurrence of tetany is easier to understand even with very low calcium values.

The renal insufficiency thus gives a disturbance in the Ca-P balance, and this is subject to the regulatory influence of the

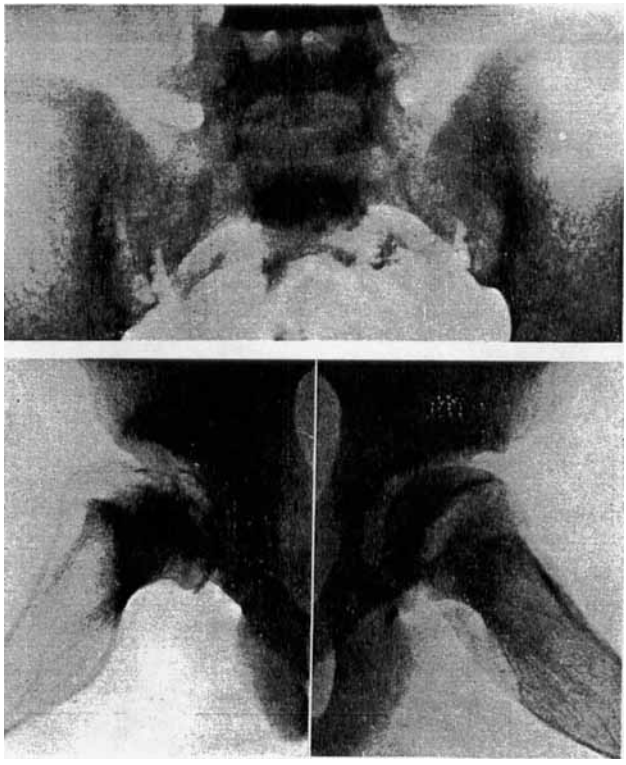


Fig. 3.

a. Marked loosening of the sacro iliac joint. b. Same as Fig. 2 c, in Lauenstein's position, showing more distinctly that there is no slipping of the epiphysis.

parathyroids, which here enter into the chain of processes involved in this disease. First through hypersecretion of parathyroid hormone, then successively through hyperplasia. Indeed, an increase in the parathyroid hormone content of the blood has been demonstrated in renal rickets (4, 30). This hyperplasia

of the parathyroids is thus to be looked upon as compensatory. Parathyroid disturbances have been demonstrated even in older patients with protracted chronic kidney lesions (Bergstrand and others, 3, 4, 13). Also by experimental reduction of the

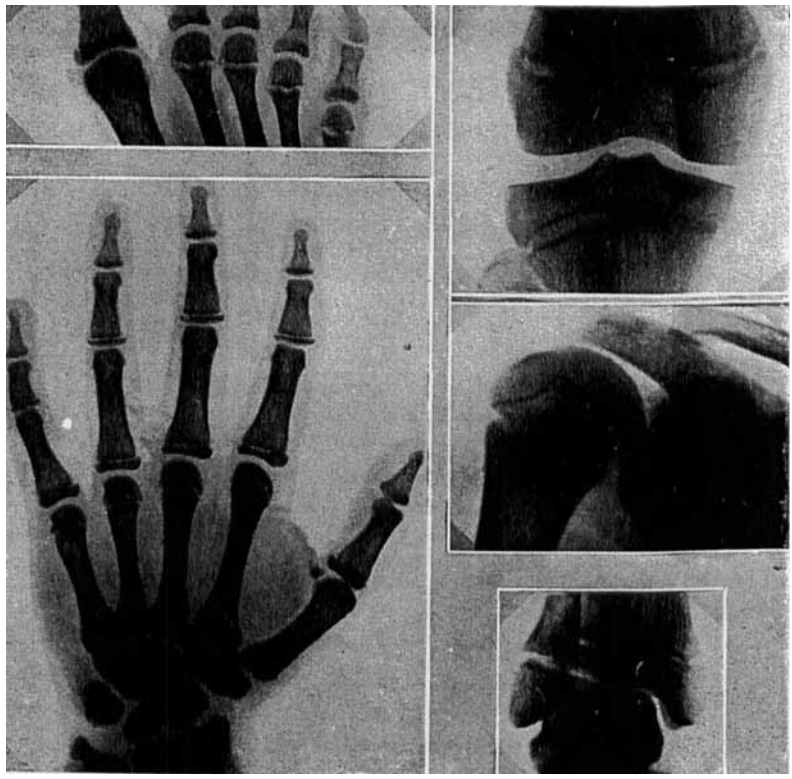


Fig. 4.
General widening of epiphyses.

kidney substance in rats, by nephrectomy or partial cauterization, it has been shown that a marked decrease in the amount of secreting kidney parenchyma gives rise to parathyroid hyperplasia (20). On the other hand, Hamilton and Highman have produced renal insufficiency in dogs by injection of large amounts of parathyroid hormone (46).

The increased secretion of parathyroid hormone now mobilizes the calcium of the bone system, and the skeletal changes of Type A go on to the more serious Type B, depending upon the amount of hormonal secretion and the severity of renal insufficiency. In extreme cases this aggravation may result in a clinical picture similar to that of generalized fibrous ostitis or fibrous osteodystrophy complicated by chronic nephritis (4, 30). The differential diagnosis between these diseases will be based largely on the anamnesis, onset of the renal insufficiency, presence and absence of nephrolithiasis and nephrocalcinosis, besides the blood chemistry (4, 8, 10, 11, 15, 18, 23, 33). In fibrous osteodystrophy the depositing of calcium in the kidneys is due to the excretion of large amounts of calcium salts through these organs, while in renal rickets the capacity for excretion is lowered through the preexisting kidney insufficiency, and hence metastatic depositing takes place in other parts of the body, mostly the blood vessels. If, besides, there is a congenital defect of the kidneys with reduction in the amount of kidney substance, the kidneys may be said to have been prepared beforehand for the insufficiency. In this way the phosphate retention will arise more rapidly, and the hypercalcæmia—so characteristic of fibrous osteodystrophy—will not always be established in renal rickets, when the secondary parathyroid hyperplasia sets in with increased secretion of hormone and mobilization of the skeletal calcium.

The renal theory thus explains the development of the skeletal changes, whereas the dwarfism, which is a constant phenomenon, may hardly have any plausible explanation, especially as it manifests itself long before the renal symptoms become far advanced. According to György, chronic acidosis is supposed to have an inhibitory effect on the growth (17); and a substance with a similar effect is said to have been isolated from hyperplastic parathyroids (30).

The endocrine theory assumes a primary parathyroid hyperplasia, with or without influence from the hypophysis. In general the other organs show no particular changes. Some cases have shown atrophy of the thymus (32), fatty degeneration of

the suprarenals (16), and depositing of calcium in the suprarenal cortex (4), and more or less significance has been attributed to these findings. Chown has advanced the theory that the hypophysis may be the origin of the disease, and he has found pathological changes in this organ (9). On assumption of a primary parathyroid hyperactivity the disease takes a similar course as that of fibrous osteodystrophy, and the skeletal changes are fully explained, whereas the dwarfism and infantilism more likely are due to hypophyseal dysfunction, and thus the problem becomes far more simple when we assume that the hypophysis is contributory to the development of the disease. According to this theory, the renal changes would be secondary —and this question has been discussed in part in the preceding.

Through the established hypercalcæmia and increased excertion of calcium with the urine, a part of this calcium is reabsorbed in the kidneys and deposited in the peritubular tissue, where it gives rise to a true chronic interstitial nephritis. It seems unquestionable that progression of this condition will be associated with destruction of the kidney parenchyma, and this may explain the chronic renal insufficiency. In objection to this theory it has been emphasized that there are a number of cases in which the damage to the kidneys was combined with congenital anomalies of the urinary tract, and that these cases make up an all too large percentage to be overlooked or disregarded (2, 11, 14, 16, 21, 25, 30, 33). Ellis and Evans have given an account of 20 cases, among which 14 showed the presence of cystic kidney, hydronephrosis, hydro-ureter or ureteral stenosis (27). A theoretically satisfactory explanation may be found, however, by assuming a disturbance in the ganglion centers controlling the hypophysis; these centers are located in the diencephalon, supposedly near the centers that regulate the development of the renal system, and where an injury might explain the development disturbances in the urinary tract (27).

Thus the syndrome skeletal changes together with renal insufficiency, congenital or acquired, has gradually developed into a more complicated symptom complex with pronounced disturbances in the metabolism. Also disturbances in the cystin dia-

thesis (6, 22, 28) and glucose metabolism (11) have been demonstrated. Several plausible theories have been advanced, and future will show whether the conclusive solution of the problem of renal rickets falls within the working field of the urologist, the pediatrician or the endocrinologist.

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DISCUSSION:

Bentzon, Aarhus:

In connection with the paper read by Dr. Gordh I may mention a case that in all essentials is similar to his.

The patient is a young girl (Ruth S.), born May 24, 1921. The history of her case has been published in detail before, by internists, in Hospitalstidende. The prognosis quoad vitam was made by these colleagues as being poor. Nevertheless, the patient keeps living, and in spite of the pronounced dwarfism and

infantile habitus, the menstruation commenced at about normal time—at the age of 15 years. She is rather hard of hearing, what I think is not infrequent in this disease.

Her renal affection was demonstrated the first time when

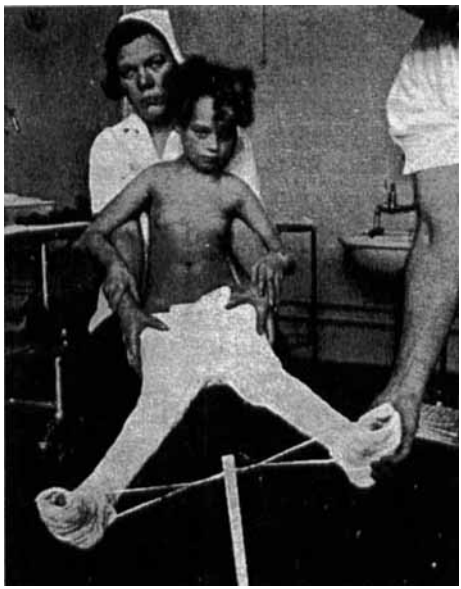

Fig. 1.

she was 1 year old, and there has always been albuminuria since. As to the functional kidney tests, determination of serum calcium, etc., I beg to refer to the publications I mentioned before. Here I shall limit myself to the orthopedic problems of the case.

It is the general consensus of opinion that these patients are able to stand true operative measures under anæsthesia but very poorly. When this patient entered the Aarhus Orthopedic Hospital because the genua valga gradually had become so monstrous that they made her entirely unable to walk, I was afraid therefore to submit her to the bilateral osteotomy which orthopedically was indicated for this deformity. On the other hand, it seemed a pity not to try to correct the deformity so much

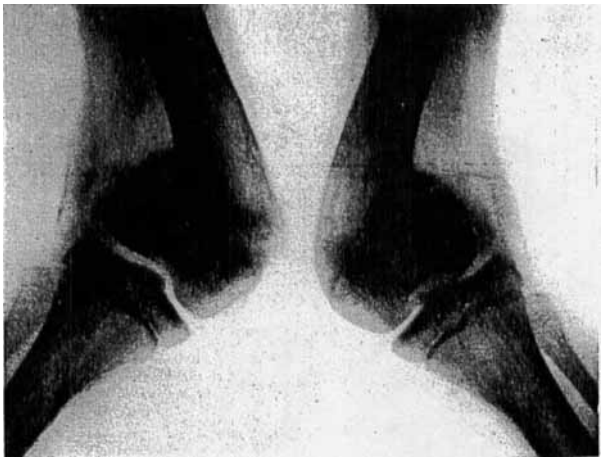

Fig. 2.

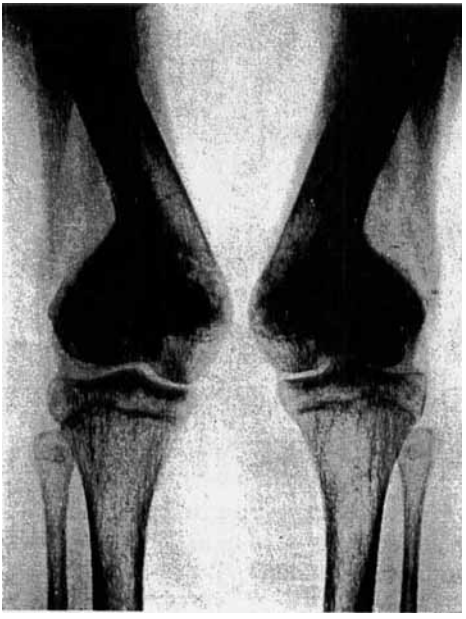

Fig. 3.

that she would again be able to walk. So she was admitted to the hospital, and an attempt was made at bloodless correction of her deformity ad modum Mommsen. She was put in plaster as shown in Fig. 1, with both thighs kept together in a "common" cast, with the crura branching off like a fork. Corresponding to the knee-joints, linen straps were embedded medially in the

Fig. 4.

plaster cast, and a partial transverse incision was made in the lateral surfaces of the cast so that the Mommsen traction applied to the lower part of the legs could have a correcting effect on the valgus position.

I really had been uneasy about the possibility that this arrangement might be likely to cause a looseness of the knee-joints, but it was found that the correction took place in the cartilage of the distal femoral epiphysis, which had undergone pronounced pathological changes.

Thus, by repeating the plaster bandaging a couple of times,

we succeeded within a little over 2 months in reducing the valgus deformity from the degree presented in Fig. 2 to that shown in Fig. 3, whereafter the patient was able to walk fairly well with the support of leather capsule bandages and canes.

Later, on reexamination, this condition was found to have kept unchanged, and her general condition appears at any rate not to have got worse—on the contrary, she seems to look a little better and feel a little stronger. As shown in Fig. 4, her valgus deformity is now very slight.

Guildal, Copenhagen:

G. mentioned the case of a male patient suffering from renal rickets with genua valga, on whom osteotomy was performed in the Orthopedic Hospital, Copenhagen (V. B. 58/32).

Family history negative as to dwarfism. Past history of good health; no rickets in early childhood. From the age of 10 years, increase in valgus deformity of the knees. In 1928 bilateral osteotomy on the femur, performed in the Aarhus City Hospital. Plaster cast for 4 weeks.

During the first half year the position of the knees was very good, but then the deformity was reproduced gradually, and now it is so pronounced that it is a hindrance to walking, and it causes pain in the knee-joints and feet. No symptoms from the other organs; never any spontaneous fracture. No cardiac symptoms.

The patient is retarded in growth, measuring only 133 cm. in height, but the proportions are normal. The mental faculties appear to correspond to his age.

Moderate cyanosis of the cheeks. Skin dry.

No enlargement of the thyroid or thymus.

In erect posture, marked valgus of the knee-joints, about 25°. The medial condyle appears to be more developed than normally. Free mobility of the joints; no looseness; no abnormal amount of fluid; no crepitation. On the lateral surface of the thighs, linear scars from the operation. On palpation, the site

of osteotomy is strikingly inconspicuous; in particular, there is no deviation. Good firmness. No tenderness.

Feet in marked plano-valgus position.

The patient is referred to the Dep. of Pediatrics, Rigshospital, wherefrom a report is received, stating that a preliminary examination has revealed no abnormality with regard to the *blood calcium* and *blood phosphorus* (blood calcium on 21/1: 10 mg.%; 13/3: 10.6 mg.%; blood phosphorus on 28/1: 3.92 mg.%; 13/2: 4.08 mg.%). But there was an increase in the blood urea concentration (3/2-32: 65 mg.%; 10/2: 68 mg.%).

As the valgus deformity increased, the Pediatric Department of the Rigshospital was consulted again. On 2/3-33: Osteotomia cuneiform. fem. utr., with the best possible correction of the position.

In 1934 reexamination showed some varus position of the thighs, but the general impression was fairly good, and the patient was able to flex the knees to a right angle.

Height: 146 cm.

The patient is not suited for hard manual labour; otherwise he is able to be occupied with almost any kind of work.

In 1936 it was stated that the patient had become an apprentice in an office, and that he tended to his work without any inconvenience. In March 1938 it was stated that the patient was feeling well and doing his work well as office clerk.

There was no recurrence of the varus position.

We have not had any occasion to see the patient since 1934.