feature here was the apparent metabolic "balance" produced by the concomitant hypersecretion of glucocorticoids, mineralocorticoids and androgens. Thus there was no disturbance in muscular strength, bone growth, or electrolyte balance. The marked body odour probably was due to excessive apocrine activity. The tumour was lateralized by intravenous pyelography combined with tomography. Presacral air insufflation was rendered unnecessary by this manœuvre. It is noteworthy that the length of this history (18 months) was not incompatible with a localized carcinoma in this case.

Steroid Findings.—Despite the probable zonation of function within the normal adrenal cortex (Chute *et al.*, 1949; Landing, 1955), the tumour, a strikingly undifferentiated carcinoma, possessed the ability to synthesize all the three major groups of adrenal hormones. While the simultaneous hypersecretion of glucocorticoid and androgen by adrenal tumours is commonplace, and that of glucocorticoid and aldosterone has been described, the present case offers a remarkable example of pluripotency of an adrenal tumour. The increase in aldosterone excretion was notable not only for its magnitude but also for the absence of its expected effect on electrolyte excretion or serum potassium level. The opposing effect of androgens in promoting potassium retention may be relevant in this connexion. In common with some cases of Conn's syndrome of primary aldosteronism, sodium metabolism was not grossly disturbed; arterial hypertension was, however, present. Possible explanations for lack of electrolyte disturbances include the simultaneous hypersecretion of an unknown sodium-diuretic hormone; alternatively, some combination of known hormones may possess sodium-diuretic activity, or conceivably the renal tubular epithelium may lose its responsiveness to aldosterone.

Treatment.—When there is no evidence of secondary deposits, adrenalectomy is indicated in cases of adrenocortical carcinoma. When there is doubt about which adrenal is involved, the transperitoneal approach is favoured. Adrenalectomy may be followed by a period of hypoadrenalism, because the opposite adrenal is usually atrophic. To prevent this, cortisone is usually given pre-operatively and continued during the operation and in the immediate post-operative period. When cortisone is withdrawn, corticotrophin may be given in an effort to stimulate the remaining adrenal gland. In our patient there was a precipitous drop in the blood pressure 12 hours post-operatively. This was believed to be an indication for intravenous cortisol. It suggested that the remaining adrenal was atrophic and not also carcinomatous. The change in the appearance of the patient 12 weeks after the operation was very gratifying. A striking feature was the disappearance of the strong body odour. Though androgen overactivity as judged by the 17-ketosteroid excretion was now controlled, the hirsuties and clitoral enlargement did not disappear until a further four months had passed. These signs of virilism usually take a long time to regress and may in some instances be permanent, in which case clitoridectomy may be necessary.

Prognosis.—If the offending adrenal is removed before metastases have developed, the chances of complete cure appear to be good. Unfortunately, in many cases of Cushing's syndrome in childhood the progress is rapid and adrenalectomy useless. Our patient seems to be fortunate, and the probability of cure must be considered high, since steroid excretion has returned to a normal level.

Summary

The relationship between Cushing's syndrome in childhood and adrenocortical carcinoma is briefly considered. A "mixed picture" of Cushing's and adrenogenital syndromes is characteristic, and is illustrated by a reported case. In this patient an excessive production of glucocorticoids, androgen, and mineralocorticoids (aldosterone) was demonstrated. In some ways the usual clinical effects of each of these steroids were annulled by the opposing effects of the others.

The offending adrenocortical carcinoma was removed. Its histological appearance is described. The operation was followed by a remarkable improvement, with return to normal in the child's appearance and her hormone output.

We thank Professors J. F. Brock, G. C. Linder, F. Forman, J. H. Louw, and F. J. Ford for their continued help and interest. The patient was under the care of the latter two professors while in hospital. Dr. T. Sacks took the photomicrograph. The costs were covered by grants from the Council for Scientific and Industrial Research of South Africa and the Staff Research Fund of the University of Capetown.

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MULTIPLE EPIPHYSIAL DYSPLASIA

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Multiple epiphysial dysplasia is an uncommon condition, but is likely to be of interest to general practitioners on account of its marked familial features. Although Fairbank (1947) did not stress the genetic aspect in his original description, a number of affected families have since been recorded. Often a correct diagnosis in one member of a family will uncover several previously undiagnosed or misdiagnosed cases. Recognition of this condition is of more than academic interest, since many patients derive benefit from appropriate physiotherapy, while harm may result from treatment based on the wrong diagnosis. To make this condition more widely known, we report three further affected families, with descriptions of the clinical, radiological, and genetic features of these and previously published case reports. The family trees are shown in Figs. 1, 2, and 3. The clinical features are summarized in the Table.

Historical Note.—In 1935 Fairbank suggested the term "epiphysial dysplasia" for a patient with multiple irregular epiphysial ossification. To this title "generalisata" was added, but it was changed later to "multiplex" when the epiphysial changes were found to be multiple rather than generalized. Twelve years later he published a masterly account of the clinical and radiological features of the disease based on a study of 20 patients, many of whom had previously been reported as atypical examples of a variety of conditions, including achondroplasia, stippling of the epiphyses (dys-

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plasia epiphysialis punctata), and Morquio-Brailsford's osteochondrodystrophy. Since then a number of cases have been reported in this country—for example, three sisters by Waugh (1952); one case by Scott (1952); four cases, two of them siblings, by Watt (1952); 14 cases in three families by Maudsley (1955); three cases in three generations of one family by Shephard (1956); and, in America, six cases by Jackson *et al.* (1954)—two sporadic and four from one family—and one case by Levy *et al.* (1957).

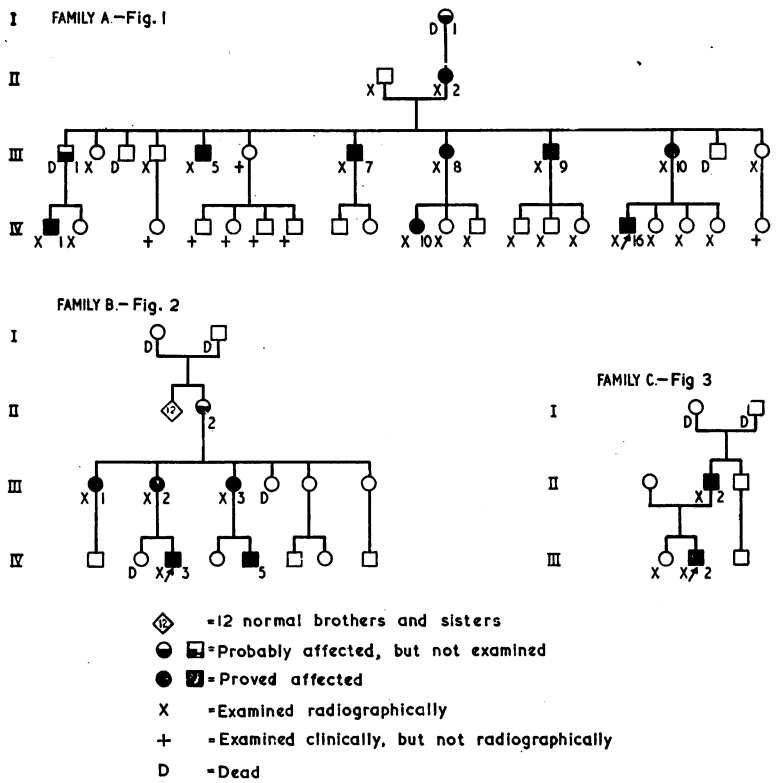

FIG. 1.—Tree of Family A. FIG. 2.—Tree of Family B. FIG. 3.—Tree of Family C.

Clinical Features

Definition.—Fairbank defined multiple epiphysial dysplasia as "a rare developmental error characterized by mottling or irregularity in density and outline of several of the developing epiphyses, dwarfism, and stubby digits."

Age at Onset.—Symptoms invariably begin in childhood, but most adults cannot recall their age at onset with any accuracy. The youngest recorded case is that of a 19-months-old infant by Gardiner-Hill (1937), while Shephard (1956) reported a probable example of the condition in an 80-year-old woman. In the present series the ages range from 2 to 71 years.

Symptoms

The most prominent symptoms are joint pain and stiffness, with a variable degree of disability. Since the lesions are multiple rather than generalized, symptoms often predominate in specific joints. Most joints have been incriminated at some time, the hips and knees being the commonest, probably because they are the most liable to weight-bearing strain. In this series the ankles, hands, and spine have also been affected, while the shoulders and elbows have caused disability in other reported cases. However, it must be emphasized that the distribution of symptomatically affected joints does not parallel the radiological distribution, which is often severest in the epiphyses of the wrists, hands, and ankles.

The degree of disability varies from slight discomfort to marked impairment of function. In family A all affected members were fully active and readily able to follow a variety of occupations, such as domestic, manual, and clerical work; none of them was able to indulge in athletic sport without after-effect, and the two youngest members often limped. In family C, in contrast, symptoms were almost continuous, and one patient could walk only with the help of a stick. With severe pain in the hip or knee,

Patients Affected with Multiple Epiphysial Dysplasia and Living in England

	Age	Sex	Symptoms: Age at Onset	Symptoms: Joints Symptomatically Affected	Symptoms: Pain	Signs: Height in cm.	Signs: Stubby Fingers	Signs: Wasting	Signs: Crepitus	Signs: Limitation of Movement	Signs: Deformity	Previous Diagnosis	Remarks
Family A:													
II 2 ..	71	F	60	Knees	+	157	+	0	+	+	0	Osteoarthritis	Some disability
III 5 ..	42	M	S.C.	Ankle, hands, knees	+	159	+	+	+	0	K.K.		,, ,,
III 7 ..	38	M	S.C.	Knees, right hip	+	165	+	0	+	0	0		,, ,,
III 8 ..	36	F	S.C.	Knees, ankles	+	157	0	+	+	0	K.K.		,, ,,
III 9 ..	34	M	S.C.	Ankles, hands, knees	+	168	+	+	+	0	K.K.	Arthritis	
III 10 ..	33	F	S.C.	Knees	+	154	0	+	+	0	K.K.		Some disability
IV 1 ..	26	M	S.C.	,,	+	160	+	+	+	0	,,	Spinal cord lesion	
IV 10 ..	10	F	9	,,	+	127	0	0	0	0	0		
IV 16 ..	9	M	3	,,	+	127	+	+	+	0	K.K.		
Family B:													
III 2 ..	48	F	25	Elbow, shoulder, knees, ankles, hips, back	+	145	+	+	+	+	+	Polyarthritis	1 in. (2·5 cm.) shortening of left leg. Also wears back brace
III 3 ..	44	F	10	Hips, knees, back	+	147	+	+	+	+	+	Bilateral Perthes's disease	Severe disability necessitating cup arthroplasty
IV 3 ..	12	M	6	Hips, back	+	142	+	+	+	+	+		Waddling gait
IV 5 ..	16	M	4	Knees, hips	+	163	+	+	+	+	0	Bilateral Perthes's disease	Waddling gait; attending school for physically handicapped
Family C:													
II 2 ..	48	M	3	Ankles, hips	+	156	0	0	0	+	0	Dislocation of hip; tuberculous osteitis	Some disability
III 2 ..	4	M	1½	Nil	0	90	0	0	0	0	0	Atypical achondroplasia	Occasional limp

S.C. = Since childhood. K.K. = Knobbly knees.

patients may have great difficulty in negotiating stairs. Flexion deformities have been described, particularly of hips, knees, and elbows, while, paradoxically, hyperextensibility of joints due to laxity of the ligaments has also been reported.

Although the changes in the epiphyses are permanent, the symptoms are characteristically transient and fluctuating. Pain and stiffness are the invariable aftermath of strenuous exercise, and for this reason children and young adults are the most likely to seek medical attention. Failure to recognize the true nature of the condition may lead to a false assessment of whatever form of therapy is used, since symptoms usually subside spontaneously after a few days and may not reappear for several weeks. In later life the gradual superimposition of osteoarthritis accounts for the common complaint of pain after rest. All the affected adults of family A noticed stiffness after sitting for a few hours, and, similarly, felt worse on rising in the mornings. This has also been observed in children, and may well be a specific symptom of the disease.

Signs

Dwarfism.—Patients are characteristically short. The reduction in height is of the short-limb type, but epiphysial changes in the spine may also contribute to the shortness. Marked dwarfism is rare, but the affected members of a family can usually be singled out in being several inches shorter than unaffected members. Here comparison with relatives is more important than reference to so-called normal standards.

Stubby Digits.—This feature is probably present to some degree in all cases, and is often striking. The hand is broad, while the fingers are short, squat, and stumpy (see Fig. 4);

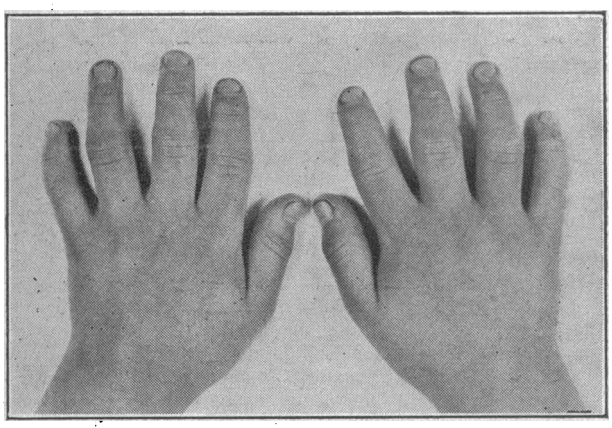

FIG. 4.—Broad hands; fingers short and stumpy

the nails may be deformed. The appearance is due to stunting of the phalanges and metacarpals, but gives rise to surprisingly little disability.

Enlargement of Epiphyses.—The affected epiphyses are often enlarged, and this may be accentuated by the presence of muscular wasting. Affected knees have a typical knobby appearance (see Fig. 5), which has earned one of our patients a prize in a seaside competition for knobbly knees. Epiphysial bossing may be irregular and lead to secondary deformities such as genu varum or valgum.

Limitation of Movement and Deformity.—In family A only one patient showed slight limitation of extension of the knees; family B and the father in family C, who were more severely affected, showed marked limitation of hip movement, particularly flexion and abduction. Involvement of the lower limbs leads to abnormalities of gait and posture, and a limping or waddling gait is common. Other described abnormalities include dislocation of the patellae, spinal abnormalities, platybasia, and subluxation of the elbows.

Intelligence and General Development.—The occurrence of subnormal intelligence (Fairbank, 1947), sexual infantilism (Buxton, 1930), and osteochondritis dissecans (Watt, 1952) in isolated cases is probably coincidental. No abnormalities of blood chemistry have yet been described. The association of cleft-palate in all three of Waugh's (1952) patients can hardly be coincidental; the family pattern does not fit dominant inheritance, and there was premature closure of the epiphyses, which has not been seen in other affected families, suggesting that some other condition operated in this particular family.

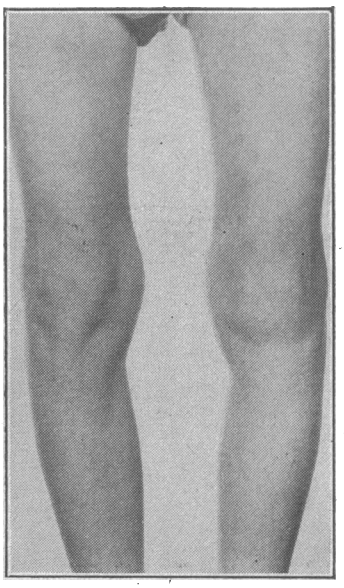

FIG. 5.—Knees have a knobbly appearance.

Complications

Osteoarthritis is an inevitable complication, beginning, as may be expected, in early adult life. Severe hip involvement may cause difficulty in natural childbirth and was the reason for termination of pregnancy in one member of the present series; six years later this patient underwent a cup arthroplasty for relief of marked superimposed arthritis of the hips.

Radiographic Features

Radiographic features have been well described previously, notably by Fairbank (1951). Multiple epiphysial dysplasia is a possible diagnosis in any case showing abnormal ossification of more than one epiphysis, particularly when like abnormalities can be demonstrated in other members of the family.

Typical radiographic signs are often found in the epiphyses at symptom-free joints. The changes are not necessarily generalized, and the normal appearance of many epiphyses may lead to the diagnosis not being considered. Some epiphyses may show slight irregularities of shape or ossification which assume significance only if the condition is suspected. Although the changes are usually symmetrical, there may be marked differences between the two sides, especially by the time adult life is reached. This was so in one patient (A, III 9; see Fig. 6) in whom there was gross deformity of the right femoral head; the left femoral head showed a slightly wavy outline, but the significance of this by itself might easily escape notice. Gross delay in the

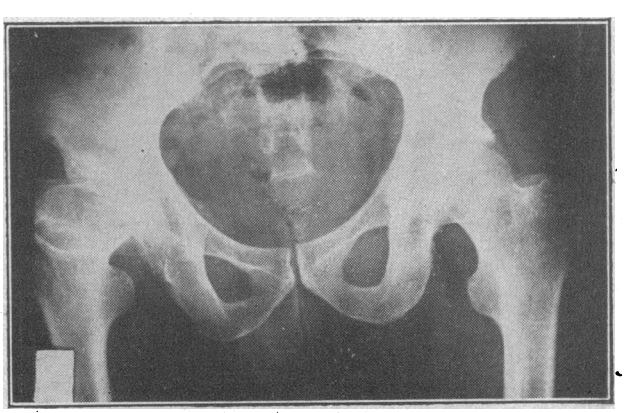

FIG. 6.—Showing deformity of right femoral head.

appearance and development of epiphyses can also occur as in the patient (C, III 2) in whom no ossification of the femoral heads could be seen at the age of 4 years (see Fig. 7): other epiphyses were thought to be normal, and the diagnosis was not made until the significance of slight thinning of the lateral part of the lower tibial epiphyses was realized. Advanced changes were then found in the hips of the child's father (see Fig. 8).

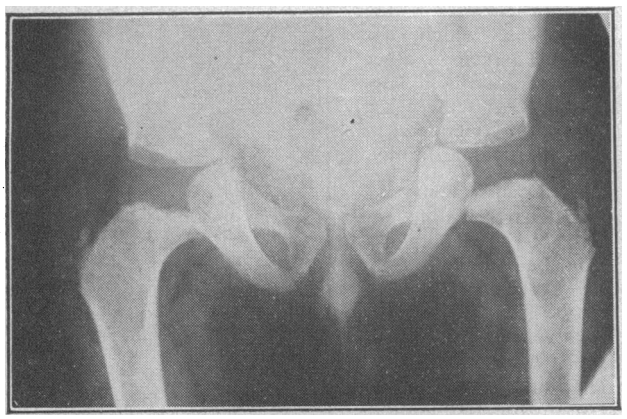

FIG. 7.—Absence of ossification of the femoral heads at the age of 4 years.

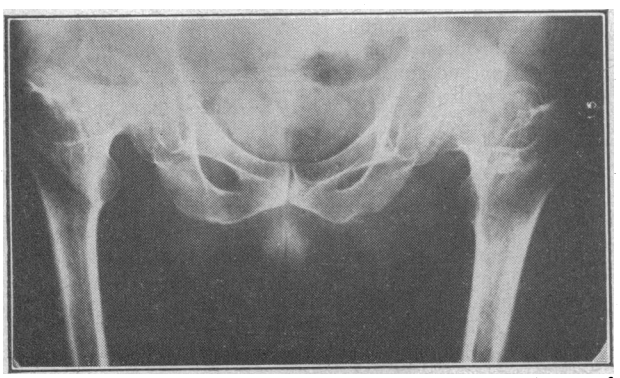

FIG. 8.—Showing advanced changes in the hips of the father of the case illustrated in Fig. 7.

Epiphyses may be seen to be arising from more than one centre of ossification. However, the appearance is quite different from that in dysplasia epiphysialis punctata, as in the condition under consideration the main bulk of the epiphyses arises from a single centre and the accessory centres are few in number and are scattered at the periphery of the cartilaginous epiphysis (see Fig. 9).

There is usually marked similarity in the radiographic features between affected members of the same family but considerable variation from one family to another. The distribution of the major changes appears to be a family characteristic. Even within families, however, some variation can occur. Moreover, members of an affected family may show such slight anomalies of epiphysial growth that it is sometimes difficult to decide, in early childhood, if they are affected or not.

Fairbank (1947) lists a number of conditions in which irregularity of the epiphyses is a feature, including cretinism, stippling of the epiphyses, osteochondrodystrophy, Perthes's disease, and dyschondroplasia. In our experience, the correct diagnosis has been neglected in favour of more common conditions. Thus in family A symptoms had been ascribed to osteoarthritis and rheumatism, two members of family B were thought to have bilateral Perthes's disease, and the father in family C had been told that he had bilateral dislocation of the hips. The radiographic appearances of multiple epiphysial dysplasia are quite distinct, however. Attention to the following points has been thought to be of most use in diagnosis :

Hands.—The carpus is small in depth and the carpal bones are irregular in shape. During childhood there is marked delay in ossification of the carpal centres, although their abnormal configuration makes accurate comparison with normal standards impossible. The central parts of the lower radial and ulnar epiphyses are thinner than normal, so that in adult life the lower ends of radius and ulna make a distinct V. The metacarpals and phalanges are not involved in all families, but are usually very short and stubby, with square angular heads.

Ankles.—There is marked thinning of the lateral part of the lower tibial epiphyses during childhood, leading in adult life to a characteristic downward inclination of the ankle-joint from the lateral to the medial side.

Knees. — The femoral and tibial condyles are square and angular. The lower end of the femur is flat and the intercondylar notch notably shallow. Irregularity of the ossification of the femoral condyles may lead to an appearance suggestive of osteochondritis dissecans.

Hips. — The femoral heads show great variation in that they may be absent, grossly deformed, irregularly ossified (as in Perthes's disease), or normal. Progressive flattening and enlargement of the femoral heads similar to that in Perthes's disease may also be seen and lead to permanent deformity and secondary osteoarthritis.

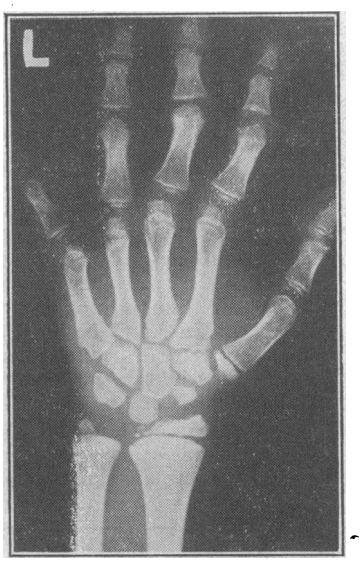

FIG. 9.—See text.

Spine.—This was not radiographed in most of our cases. One child (family B, IV 3) showed changes in the dorsal spine which, without the changes at other epiphyses, would have been indistinguishable from Scheuermann's disease. In the other affected members of this child's family the vertebral bodies appeared flat but normally ossified.

Genetic Aspects

The families were investigated by taking radiographs, so far as was possible, of the knees, ankles, and hands of all the offspring of affected individuals. It was found, however, that clinical examination was reliable for picking out those affected, and the only individuals possibly misclassified are some of the younger members of family A.

The first genetic problem is whether all cases are determined by dominant genes. Fairbank (1947) observed: "It affects both sexes, and as a rule is not inherited or familial." However, one boy appeared to have inherited from his mother, while two sisters are also included in this series. In addition he reported: "J. A. Cholmely has furnished me with details of two cases, who are brothers and apparently the only affected in a family of 10." A number of the families reported since then are similarly inconclusive from the point of genetic determination. But in 1954 Jackson *et al.* reported the disease in a mother, two sons, and a daughter, and in 1955 Maudsley reported its presence in three families. He noted that in the first of these the pattern of inheritance, with 10 of 17 members in three generations affected, strongly suggested that a dominant gene was concerned. His other two families were not fully investigated, but are compatible with determination by a dominant gene. In 1956 Shephard reported a family with three affected members in three generations, again suggesting dominant inheritance.

The three index cases in our families were unselected and were all that could be found among the children attending

the Hospital for Sick Children, Great Ormond Street. A search for further possible cases among children with the diagnostic labels " bilateral Perthes's disease " and " multiple osteochondritis " produced no cases other than one child already known to be a member of family B. In two of these families a dominant gene is clearly responsible, and the third, with father and son both affected, also suggests this type of genetic determination.

All the known examples of the condition to date, with the exception of those reported by Waugh (1952), can be ascribed to dominant inheritance. On this hypothesis, where only sibs were affected, an abnormal parent would have been found on investigation ; for example, Watt (1952) notes that the parents of his patients were dead but that the father " was said to be a small man with a large body and short limbs, and had occasional trouble with his hips." On this hypothesis, also, sporadic cases will occur due to fresh mutations, as in the family with one boy affected and both parents and four sibs normal on radiographic examination (Levy *et al.*, 1957). It would appear that, while the gene may not always cause symptoms, it always leads to the development of abnormal radiographic signs.

The second genetic problem is raised by the suggestion made by Maudsley (1955) that there is a similarity of the type of affection within each family. In two of our families the affected members all showed dwarfism and major lesions in the hips, and all the adults had considerable disability. In one of these families, B, all affected members in addition had stubby hands. In family A, on the other hand, the hands were less stubby, and in some instances nearly normal, some patients were normal in height, and patients mostly complained of pain only in the knees, while several had never sought medical advice. The resemblance of the radiological features within families has already been mentioned. This suggests that two genes at least are responsible—one for the relatively mild form seen in some cases, and another for the more severe form ; but to prove this point conclusively it would be necessary to take more radiographs than is justified for a point of purely theoretical importance.

Treatment

Although the pathological basis of the condition cannot be influenced in any way, it is of interest that those patients who received physiotherapy, particularly in early life, experienced considerable relief at the time and developed less disability later on. Frequent attacks of pain and stiffness are likely to cause disuse and local muscular weakness, which, in turn, favours instability of the affected joints and recurrence of further attacks of pain. Where benefit was derived from physiotherapy it could be ascribed to the measures directed to improving muscular support of the affected joints, such as non-weight-bearing exercises. One member of the second family had long remissions after courses of underwater exercises, and the children in the present series are attending regularly for non-weight-bearing exercises which they continue to practise at home, in the hope that these will prevent muscular wasting, reduce the number of acute episodes, and delay the development of osteoarthritis, which, however, will eventually dictate the form of treatment.

Illustrative Case Histories

A boy (family A, IV 16) aged 9 was referred to the Hospital for Sick Children, complaining of recurrent pain in the legs since infancy, in acute attacks at intervals of three to four months, when he would be almost completely disabled for as long as a week. Attacks were often precipitated by exercise and resolved spontaneously. His milestones were not delayed and he walked at the age of 18 months. On examination he looked thin and unwell, his height, 50 in. (127 cm.), was normal for his age, but his fingers were short, his knees knobby, and his right leg was of poor musculature (see Fig. 5). Radiographs showed the typical changes of multiple epiphysial dysplasia in him and other members of his family.

A boy (family B, IV 3) was referred to the Hospital for Sick Children at the age of 7 years with a two-year history of pain and stiffness in the left knee. On examination he had a rolling gait and slight shortening of the left leg, and it was thought that the pain in the knee was referred from the hip. Abnormality of the upper femoral epiphyses was noted on *x*-ray examination and on investigation of similar symptoms in several members of his family ; the diagnosis of multiple epiphysial dysplasia was subsequently confirmed. He still has severe pain and stiffness in both hips and usually walks with a limp. On examination at the age of 12 there was some limitation of movement at both hips, his height, 56 in. (142 cm.), was below average for his age, and his fingers and toes were short and stubby. He recently developed inversion of the left foot. Skeletal survey showed dysplasia of the epiphyses of the tibiae, femora, radii, humeri, and lower dorsal spine.

A male child (family C, III 2) was referred to the Hospital for Sick Children at the age of 19 months because his parents had noticed a waddling gait, that his left leg turned out, and that he seemed short for his age. His father was said to suffer from congenital dislocation of the hips, but has since been confirmed as suffering from multiple epiphysial dysplasia. On examination the child was short—28 in. (71 cm.)—and he had a rolling gait and a lumbar lordosis with a protuberant abdomen. The main radiological abnormality was delay in the appearance of the upper femoral epiphyses, which were still not present at the age of 4 years (Fig. 7). He was otherwise well and there had been no delay in the appearance of his milestones.

Summary

Multiple epiphysial dysplasia is a genetically determined condition in which there is irregular and often retarded ossification in several of the epiphyses. Disability may be severe, with recurrent attacks of joint pain and stiffness: subsequently, osteoarthritis may lead to severe limitation of movement. The commonest sites for symptoms are the hip and knee, and the most marked signs are dwarfism and stubbiness of the fingers. The symptoms and signs are remarkably similar in the various affected members of the same family.

Three families, consisting of 15 affected members, are reported, the family pattern being that of dominant inheritance. It is suggested that dominant genes are responsible for all examples of the disorder, although more than one mutation may be concerned to account for differing degrees of severity in different families.

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Mr. RODNEY MAINGOT, F.R.C.S., was awarded a Sydney Body gold medal at a ceremony at Southend-on-Sea Hospital on May 30.