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Achondrogenesis type I

Sir,

We read with great interest the article by Lauder *et al.* (*Archives*, 1976, **51**, 550). Their observation is correct in that their 2 cases are subvariants of achondrogenesis type I according to the McKusick classification.

We would like to point out that two distinct subtypes of that condition have already been described (Spranger *et al.*, 1974; Yang *et al.*, 1974, 1975). According to the criteria developed in our studies, the 2 cases reported by Lauder *et al.* should be classified into type I of lethal achondrogenesis (Yang *et al.*, 1974, 1975, 1976, 1977), which is characterized by deficient cranial ossification, multiple rib fractures, shorter appendicular bones,

sufficient cartilage matrix in the epiphyseal cartilage, higher incidence of familial occurrence, etc. Recently we have observed intracytoplasmic inclusion bodies in the resting chondrocytes (Yang *et al.*, 1976, 1977). We suggest that Dr. Lauder and colleagues re-evaluate their histological sections of cartilage, which seem in the low magnification photomicrographs to contain several inclusions.

Type II lethal achondrogenesis differs roentgenographically and morphologically from type I. Grebe achondrogenesis, formerly type II, is nonlethal and appears to be an unrelated disease.

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Drs. Lauder and Ellis reply as follows:

We are grateful to Drs. Yang and Bernstein for their comments on our paper on achondrogenesis type I. At the time of preparation of our manuscript we were not aware of their recent paper (Yang *et al.*, 1974) in which they report 2 cases, one of which exhibits similar clinical and pathological features to our own 2 cases. We agree with them that the available evidence seems to suggest two distinct forms of achondrogenesis type I. It remains our view that until the precise biochemical defects have been elucidated, no definitive classification is possible. This is also the view expressed by Wiedemann *et al.* (1974) whose collection of 6 cases is still the largest reported. The difficulties in attempting a classification based on 2 cases plus a review of previous studies are exemplified by the necessity for a 'readjustment of eponyms' which Yang *et al.* (1975) subsequently reported. As our paper points out, many of the studies are unsatisfactory in that no comments are made regarding the appearances in undecalcified material. The absence of adequate biochemical and histochemical data in these reports also makes distinction from conditions such as severe hypophosphatasia extremely difficult. It was also our aim to stress the importance of pulmonary hypoplasia as a cause of death in these infants.

We were most interested in the observation by Yang *et al.* (1976) of intracytoplasmic inclusion in resting chondrocytes. We have not had the opportunity to study their report or any illustrations of these bodies. We have not as yet been able to convince ourselves of the presence of inclusion bodies in our material.

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Radiological findings in urinary infections

Sir,

We read the article on radiological findings in newborn infants with urinary infections (*Archives*, 1976, **51**, 628) with great interest and were prompted to review our own experience with neonatal urinary tract infection. This hospital is responsible for the great majority of deliveries in Harrow and over a 3-year period between July 1973 and June 1976, 7962 babies were born here. During this time urinary tract infection, confirmed by suprapubic aspirate urine specimen, was found in 20 neonates, an incidence of 0.25% which approximates to that of Smellie *et al.* (1964) and Bergström *et al.* (1972).

Our policy is to perform intravenous pyelograms (IVP) within one month of diagnosis but to wait until the infant is 6 months old before doing a micturating cystogram. Of the 20 neonates studied, 17 have had both an IVP and a micturating cystogram, 3 have had an IVP only. Abnormality was found in 2 of the 20 IVPs (10%), which again agrees with the commonly reported incidence (Smellie *et al.*, 1964; Littlewood, 1972; Drew and Acton, 1976). However, only 1 of the 17 micturating cystograms showed ureteric reflux (6%). This contrasts sharply with the findings of several groups as shown in the Table. We think that this difference may be related to the variation in timing of the micturating cystogram.

We suggest that the technical difficulties in taking x-rays of the lower urinary tract in the neonatal period may cause difficulties in interpretation of cystograms. Furthermore, the x-ray findings may be due to

Table Micturating cystograms showing reflux in neonates

	No. done	No. showing reflux	% showing reflux	Time interval
Our series	17	1	6	6 m
Smellie <i>et al.</i> (1964)	23	5	22	Not stated
Drew and Acton (1976)	64	30	44	7 d
Stansfeld (1966)	15	3	20	Not stated
Abbott (1972)	14	8	57	1 m

the acute effects of the infection (Dunbar *et al.*, 1972; Pais and Retick, 1975). Therefore, while accepting the need for full radiological investigation of the urinary tract in all children with proven urinary tract infection, we think that in the presence of a normal IVP the micturating cystogram can usefully be postponed until the age of 6 months.

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